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OF THE

PRINCIPLES AND PRACTICE

OF

MEDICINE

BY THE LATE

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THE

PRINCIPLES AND PRACTICE OF MEDICINE

VOL. II

DISEASES OF THE HEART AND BLOOD-VESSELS

FUNCTIONAL AFFECTIONS OF THE HEART

"Faintings, swoonings of despair."
Samson Agonistes.

"Arteriarum pulsus—index fere morborum, stabilis, aut citatus, aut tardus, observatione crebri aut languidi ictûs gubernacula vitæ temperat."—PLIN. *Nat. Hist.*, xi, 88.

History and general pathology of cardiac diseases—their arrangement—The pulse—The sphygmograph—Changes in the frequency of the pulse—Irritable heart—Intermittent and irregular pulse—Alternate and twin pulse—Palpitation—Cardialgia—Treatment of the above conditions—Tension of the pulse—its indications—Syncope—its distinction from epilepsy, sunstroke, and collapse—its treatment.

ANGINA PECTORIS—*Definition — Onset — Symptoms — Event — Pathology— Pseudo-angina and cardialgia — Anatomy — Ætiology — Prognosis — Treatment.*

IN passing from the affections of the lungs to those of the heart, we enter on a chapter of medicine which is even of more recent origin than that we have just completed. In fact, with the exception of the spinal cord, there is no organ the pathology of which was so entirely unknown until the present century.

Like the lungs, the heart is inaccessible to ordinary scrutiny; and like the lungs, our present knowledge of its diseases depends almost entirely upon the application of the sense of hearing, by the arts of percussion and auscultation. Harvey's great discovery had no direct effect upon cardiac pathology. Sufficient physiological knowledge of the circulation existed at the beginning of the present century, but it was almost useless for want of satisfactory methods of investigation. The study of the pulse is, indeed, of very ancient date, but its indications were scarcely better understood by European physicians seventy years ago than by those of ancient Greece or modern China.

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Beside the variations of the pulse*—which were referred to general conditions like fever, and not to disease of the heart—the chief symptoms belonging to the circulation which were recognised before the present century were palpitation, syncope, and heartburn or cardialgia, otherwise known as *passio cardiaca*, together with the rare and terrible malady *angina pectoris*, or breast-pang. The only anatomical lesions of the heart recognised were pericarditis with hydropericardium, and general enlargement; and these, as well as the cardiac symptoms just mentioned, were supposed to be the result of disturbed passions and mental emotions, not the cause of direct mechanical derangement.

Auenbrugger only mentions incidentally the dulness of note which percussion over the heart produces. His translator, Corvisart, in his essay on the 'Diseases of the Heart and Great Vessels,' published in 1810, made the first serious attempt to describe the morbid anatomy of these organs. Diseases of the pericardium, aneurysm of the aorta, dilatation and hypertrophy of the several cardiac chambers, and contraction of its orifices were the lesions that he most accurately described.

Laennec's discovery of auscultation began with auscultation of the heart (cf. *supra*, vol. i, p. 931), and he laid the foundation of cardiac semiology; but it is remarkable that while his account of the physical signs of diseases of the lungs left little to correct, and often little to add, his investigation of the heart was far less successful.†

Laennec referred almost all valvular lesions to narrowing and obstruction; the leaking produced by a damaged valve was scarcely apprehended until long after; hypertrophy and dilatation, moreover, were regarded as primary diseases, and their relation to valvular lesions was not understood.

Nevertheless, Laennec's discovery of the normal sounds of the heart, and of the fact that they become altered and obscured in disease, furnished the key to the problem. The work of associating the several abnormal murmurs with the various valvular lesions was continued by his successors in France; and in this country by Corrigan (1829), Hodgkin (1829), Hope (1832), C. J. B. Williams (1835), Latham (1845), Hughes (1845), Walshe (1851), Billing (1852), Stokes (1854), and Gairdner (1861); by Austin Flint (1860) and Da Costa (1864), in America; in Germany by many investigators, of whom Skoda, of Vienna (1850), and Traube, of Berlin (1867), were the most eminent.

In arranging this section, we will first deal with such disorders of the circulation, as are common to various kinds of organic disease of the heart, diseases of the lungs, the kidneys, and other distant organs, but which also occur as purely functional derangements.

Passing next to organic diseases, we shall take first those of its muscular substance, next those of its lining membrane, with the important subject of valvular lesions, then those of its investing membrane; and last will come a chapter on aneurysms of the aorta, with which, for clinical purposes, it will be convenient to associate other tumours within the chest.

* These were familiar to the ancients, as is shown by the quotation from Pliny at the head of the present chapter.

† This seems to have chiefly depended upon imperfect physiological knowledge. The action of the valves, though understood by Harvey, was scarcely appreciated until the experiments of Hope and Williams. So late as 1864, when the present writer was a student in Paris, one member of the Faculty of Medicine taught in his public lectures that the first sound of the heart coincided with the diastole and the second with the systole.

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Of all known diseases, those of the heart are most dependent upon direct and obvious mechanical causes. The most common and typical "heart disease" depends upon damage to the valves, which interferes with the circulation exactly as it would in an artificial machine, and which can be imitated in the schema used in physiological lectures. The next most frequent and important division, occurring usually as the result of the first, is dilatation of the cavities and hypertrophy of their walls. These are both the consequence of increased blood-pressure: the former its mechanical effect, the latter its physiological response. Degeneration of the muscular walls of the heart directly enfeebles its action as the chief motor power of the circulation. Inflammation of the pericardium, and particularly pericardial effusion, mechanically interferes with the movements of the heart. In all these cases, speaking generally—whether the primary disease affects the orifices of the valves, the size of the cavities, or the contractions of the left ventricle—the effect is the same: to impair the action of the heart as a force-pump, to diminish the blood-pressure in the aorta, and to produce a condition throughout the whole systemic circulation of arterial anæmia and venous congestion.

Disorders of the pulse.—The *frequency* and the *rhythm* of the radial pulse correspond with the time-relations of the cardiac contractions. The *size* or volume of the pulse depends partly on the natural size of the artery in the individual, but in each person upon the fulness of the vessel, *i. e.* on the force of the circulation at the time and the degree of contraction or relaxation of the arterial tunics. The *strength* or force of the pulse as appreciated by the eye or by the compressing finger is due to the energy of the ventricular contraction, but is apparently increased by relaxation of the arterial walls which allows them ready expansion in systole and collapse in diastole. The *length* or duration of the pulse depends on the length of the cardiac systole, but also on the tension or relaxation of the artery, its walls yielding slowly or easily to the wave of increased blood-pressure. The *tension* of the artery is the degree in which it yields to the compressing finger. If readily obliterated, it is said to be soft and of low tension; if firm and unyielding when squeezed or rolled under the finger, it is termed hard, wiry, or cord-like.

The sphygmograph.—Since the graphic method of registering the tracing made by a moving index on a recording surface was introduced into the physiological laboratory by Ludwig,* attempts have been made to register the arterial pulse, either by *tambours* connected by flexible tubing, or by a lever and style writing on a blackened card, which is moved rapidly past in a straight line by clockwork.

The form most generally used, and, on the whole, most useful, was devised by Marey. The ingenious modification of the instrument by Dr Dudgeon, though easy, cheap, and convenient, is more open to serious fallacies.

The sphygmographic tracing, when skilfully taken on a normal artery, shows a series of waves of equal height and force, and recurring at equal intervals. Each wave corresponds with the arterial systole. The upstroke

* The method was taken from an invention of the famous engineer James Watt. "If we repeat the experiment performed in Guy's Hospital many years ago by Mr Wilkinson King, that of attaching a bristle to the skin over an artery by means of a piece of wax, we at once perceive that the pulse is something more than an up and down movement—that the end of the bristle is making vibrations or curves. If this bristle, which Mr King called the "sphygmometer," could be made to take tracings, you would then see the natural movements of the pulse" (Wilks). Vierordt's sphygmograph suggested Marey's.

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shows the expansion of the vessel by the wave of pressure, and its degree of slope (supposing the rate of the moving card to be the same) the tension (or "fulness," as it is sometimes called) of the coats.

The apex of the wave is formed by the inertia of the lever carrying the writing point a little higher than the expanding artery does. It is known by its sharp point, and by a rounded shoulder following it on the down stroke. By connecting this latter with the upstroke we strike out the "percussion-wave" of inertia, and obtain more or less precisely the true "tidal" wave, as it is called, which would be traced by the expansion and relaxation of the arterial wall. The breadth (*i. e.* bluntness) of this tidal wave shows the tension of the artery and the length or duration of the systolic pulse; if narrow, it points to a low tension with a compressible and probably dicrotic pulse.

In the downstroke is normally seen a "notch," followed by a second wave. The former is called *the aortic notch*, on the supposition that it depends on closure of the left sigmoid valves checking the fall of blood-pressure in the arteries, early in diastole. The latter is called the *dicrotic* or second pulse-wave, and of course denotes increased pressure at this point in the diastolic collapse of the artery. Those who see the difficulty of the explanation just given, which refers it to closure of the aortic valves, explain it as due to a back wave of pressure from the small arteries and capillaries in front, which react in virtue of their elasticity when filled by the systolic wave. Whatever its precise origin, this dicrotic wave is small or absent when there is high arterial tension, and well marked when the tension is low. Sometimes it is so large that it rivals the tidal wave.

If situated high up on the downstroke, the notch and second pulse-wave show an early occurrence of the reflux, and high tension of the arterial system; if far down on the descending line, the inference is a relaxed artery with low tension. When the second wave is very low (*i. e.* very late) and very large, the pulse is called hyperdicrotic.

If we draw a line from the summit of the tidal wave to the aortic notch we have, as Dr Mahomed said, a measure of the amplitude of the pulse-wave, and so of the arterial tension. When the tidal wave is well marked and broad, the tracing will fall far outside this straight line, the tension is high, and the pulse feels hard; when there is no projection to be noticed, the tracing falling directly from the apex of the percussion-wave, the tension is low and the pulse compressible. Again, the aortic notch will come high up on the downstroke (*i. e.* early in diastole) in the former case, and low down (*i. e.* late in diastole) in the latter.

It is often difficult to get a good sphygmographic tracing, and it is only by trial that we can find the most suitable pressure to bring out the characteristics of the pulse in each case. Sometimes in chronic Bright's disease, in fever, in advanced mitral incompetence, in aneurysm, and in aortic regurgitation, the tracing is so peculiar that it carries the diagnosis with it. But, on the whole, the expectations raised by Marey's instrument have not been realised. We have as yet no means of measuring the arterial blood-pressure during life, and the sphygmograph is rather useful as a corrector or confirmer of a diagnosis based on other grounds, than a discoverer of unexpected lesions, like the stethoscope or laryngoscope.

Sometimes we see differences between the right and left pulse, of which we could not otherwise be sure; often the indications derived from the finger receive useful modification; but probably the chief benefit of the

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instrument is that it has made feeling the pulse a more intelligent procedure; it has made our nomenclature more precise, and our conclusions less individual and arbitrary.* It is possible to auscultate the chest without the aid of any instrument, but it needed the invention of the stethoscope to teach auscultation; and so also, while the practised physician is almost independent of the aid of the sphygmograph, this practice, which makes the true *tactus eruditus*, is only to be gained by assiduous comparison of the results of palpation with the tracings of the sphygmograph.†

Changes in the frequency of the cardiac beats.—Of the Functional Disorders of the Heart, we will take first that which affects the frequency of its contractions. We usually estimate this by counting the pulsations of the radial artery at the wrist; and provided that they are strong enough to be readily felt by the finger, and are separated by appreciable intervals, this answers perfectly well. But sometimes the radial pulse gives no correct information as to the frequency of the heart's action, and the stethoscope alone enables one to count its contractions satisfactorily. This is the case when they are very frequent and feeble, or when some of them fail to transmit an impulse so far as the smaller arteries.

With regard to the rate of the pulse, it is difficult to avoid an ambiguity, which may cause serious misunderstanding. Almost every one speaks of the pulse as being "slow" or "quick," when what is meant is that it is infrequent or frequent. In strictness, a *slow* pulse (*pulsus tardus*) is one in which each pulsation of the heart takes more than usual time for its completion; a *quick* or *rapid* pulse (*pulsus celer*) is one in which the ventricular contraction is short and soon over. In the present chapter, at any rate, we will employ the terms *frequent* and *infrequent* when the number of beats in the minute is referred to, and *short* or *long* when we refer to the duration of the ventricular contraction and pulse-wave.

As is well known, the frequency of the pulse is in health liable to wide variations in different circumstances. It is greater in the young than in the old, in women than in men, under exertion than during rest, in the upright posture than in sitting or lying, after a meal than when fasting. It is also liable to be increased by exertion, by emotion, or by excitement. There are many persons who cannot place themselves under a medical examination, especially for life insurance, without the heart's beats at once rising to over a hundred in the minute. This source of error may usually be avoided by counting the pulse a second time after an interval.

It has been said that the pulse may permanently stand at 100 in a healthy person; but it is doubtful whether a pulse habitually above 80 ought not to be regarded as evidence of some morbid condition or tendency (Latham's 'Collected Works,' New Syd. Soc., vol. ii, p. 526).

(1) An abnormally *infrequent* pulse is natural to some persons, in whom the heart never beats oftener than fifty or even than forty times in the minute; this does not appear to affect the prospect either of longevity or of recovering from illness. Among the few conditions which may render the pulse infrequent are aortic stenosis, some cases of fatty degeneration of the heart, and jaundice; a *pulsus rarus* is also observed during convalescence

* See Dr Galabin's valuable papers in the 'Guy's Reports,' 3rd series, vols. xix and xx, and the account taken therefrom in 'Foster's Physiology.'

† On the use of the sphygmograph, and on many points in the semiology of the pulse, see Dr Broadbent's 'Croonian Lectures' for 1887.

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from various acute diseases. It does not in itself indicate danger. The same infrequence of the cardiac beat is observed during collapse, and in some cases of cerebral tumours, compression, concussion, and apoplexy (vol. i, p. 579).

A very infrequent pulse has been often described, when the condition is really that of a *pulsus bigeminus*, in which the weak pulsation is not noticed or felt. Such a pulse with every other beat "dropped" occurs in atheroma and in mitral stenosis.

Dr Broadbent has seen the same pulse produced by digitalis; and Tripier, of Lyons, believes that a slow pulse depending on a dropped beat may also be frequently observed in epileptics.

(2) The diseases in which the frequency of the pulse is *increased* are numberless. They include all fevers and inflammations, and the great majority of primary affections of the heart.

In these conditions the blood-pressure is usually low, and the pulse is often not only compressible or dicrotic, but also irregular.

There are, moreover, some cases in which a very frequent action of the heart appears to constitute a disease by itself, and may be distinguished as primary or idiopathic *Tachycardia*. Three such instances are recorded in the 'Brit. Med. Journ.' for 1867 by Dr Cotton, Sir Thomas Watson, and Dr Edmunds. The patients were all males of middle age. In two of the cases there were several attacks at varying intervals, each lasting from a few hours to two or three weeks. The rate of the pulse was from 200 to 230, yet it was perfectly regular. The termination of the attacks seems always to have been absolutely sudden; in Sir Thomas Watson's patient, on one occasion, the beats of the heart, directly after having been counted at 216, fell to 72, exactly one third of the former number. Dr Walshe states that, in the cases of this kind, his patients, who were women, were by no means all of them hysterical or nervous, and some were distinguished by force of character. The causes which he recognised were: pedestrian excursions, the ascent of mountains, acute pain with effort to control its manifestation, and prolonged mental distress; but in not a few instances no cause could be discovered. In Dr Cotton's case the attacks were preceded by gastric disorder. The affection is not always devoid of danger. A sensation of faintness, dyspnœa, and even œdema of the lower limbs has been present in more than one instance; and Sir Thomas Watson's patient died during his fourth seizure, the heart, on *post-mortem* examination, being found large, as if it had been distended, while its muscular walls were very thin and soft.

Dr Bristowe had a remarkable case of rapid pulse, which is quoted by Dr Broadbent in his Croonian Lectures ('Brit. Med. Journ.,' 1887, vol. i, p. 659). A young man, under twenty, had a pulse of 200 or 240 beats in the minute; and the beats were ineffectual, for he suffered from dropsy and hæmoptysis. This condition of *tachycardia* had continued in occasional paroxysms since childhood. He improved with treatment, although the pulse was liable to run up under excitement to 300. He died suddenly while playing the piano. *Post mortem*, the heart was found dilated and the valves normal.

In 1870 Dr Wilks brought under the notice of the Clinical Society certain cases in which an extreme frequency of the pulse, associated with alarming dyspnœa and with palpitation, was due to nephritis, which itself was generally the result of scarlatina, although in some instances the indications of that

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disease had been almost unnoticed until the urine was found albuminous. In almost every instance recovery took place within a few days, notwithstanding that the symptoms appeared to be of the most alarming character.

In the *treatment* Dr Wilks recommends purging, cupping, and salines with antimony, rather than the administration of stimulants. In his primary cases of rapid pulse in women, Dr Walshe found digitalis of no service, but nervine tonics were decidedly useful.

Irritable heart.—The close connection between the various forms of functional or neurotic disturbance of the heart is well illustrated in a very interesting paper by Da Costa ('Am. Jour. of Med. Sc.,' 1871) on what he terms "irritable heart." This paper is further of great importance as containing what is almost complete proof of the gradual passage of such an affection into a condition of organic slowly progressive disease, by the heart undergoing hypertrophy. It is based upon no fewer than three hundred cases of soldiers in the army of the United States during the Civil War. The general history of these men is that, having been called from civil pursuits into active service without previous training, they became liable to attacks of palpitation, to more or less severe pain in the chest, of a sharp and stabbing, or of a dull aching character, and to dyspnœa on exertion, so that they became unable to keep up with their comrades, and were distressed by the weight of their accoutrements. On examination the pulse was found to be greatly increased in frequency; it was much influenced by position, so that there was sometimes a difference of thirty beats or more between its rate during standing and that during lying down; in some instances it was intermittent or irregular. The men themselves often looked strong and healthy, though their hands were apt to be bluish and mottled, and to be easily made pale by pressure. The cause of the affection was sometimes hard service in the field, particularly excessive marching. But in many cases it was directly brought on by an attack of diarrhœa, not sufficient to interfere with duty; or by fever, necessitating a few days' stay in hospital. The patients were generally young men, from sixteen to twenty-five.

The course of this affection was usually slow. But after some months of treatment it often ended in complete recovery, so that active exercise of all kinds could be borne without discomfort. In other cases the cure was imperfect, there being still a liability to cardiac symptoms on exertion. Hypertrophy of the heart was believed to have developed itself in twenty-eight cases out of two hundred.

Da Costa's observations with regard to the *treatment* of his cases are of great interest and importance. Rest was found to be very useful. Making the patient lie down for several hours daily often led to strikingly good results: and two men who were kept in bed—one by an attack of dysentery, the other by a broken leg—improved very rapidly.

A point on which he laid stress was the maintenance of great care during convalescence. Before allowing the men to return to their regiments he tested them by running and by other exercises, so as to see how the heart bore itself under strain. He gives reports of some patients who came under notice again after an interval of several years, and in whom no relapse had occurred.

Arrhythmia—intermittence and irregularity of pulse.—A common kind of perversion of the cardiac rhythm is for the heart from time to time to

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"intermit" or leave out one of its beats, or two or three successive beats, while its action in all other respects remains perfectly regular. This is not so alarming an occurrence as might be naturally supposed. Sometimes the intermissions take place at intervals of only a few pulsations, sometimes not oftener than once in two or three minutes; sometimes the intermission is regular, sometimes quite irregular in the frequency of its recurrence. The patient may be unconscious of any disturbance of the cardiac function, or he may (according to Dr Walshe) experience an instantaneous and transitory feeling of faintness. More often, what draws his attention to the fact that his heart now and then misses a beat, is that the beat which follows every intermission is unusually thumping. One may then say that besides the intermittence he suffers from palpitation. Even apart from subjective sensations, it may sometimes be noticed that there is an unusual force and fulness of the pulse in the radial artery after each pause in the heart's action.

Intermittence of the heart's action should never be made light of until the state of the organ itself and that of the vessels have been thoroughly investigated. If there be any undue arterial tension in particular, it should be carefully noted; in one well-marked instance intermittence of the pulse, observed from time to time during three or four years, was at the end of that period followed by indications of organic disease of the aorta and of the aortic valves. It is almost always a serious matter if the heart's beats begin to intermit after slight exertion, such as walking a little faster than usual, or hurrying for fifty or a hundred yards to be in time for a train.

Otherwise, there is no doubt that occasional or even habitual intermittenco of the pulse is in many persons compatible with a good state of health, and with a fair prospect of longevity.* Dr Richardson relates that he once made an autopsy in the case of an aged man, in whom for many years the pulse had always intermitted once in eight beats; he found the heart perfectly sound and the coronary arteries normal. Dr Walshe remarks that some people actually feel more comfortable when the rhythm of the heart is irregular than when it becomes (as it sometimes will) perfectly regular.

In a patient eighty-six years old, a subject of gout, the writer recently noticed the pulse repeatedly to intermit for three, four, or even five beats; yet there was no discomfort felt, and no appearance of anæmia or of venous fulness.

It is said that the tendency to intermittence passes into abeyance for the time during any illness attended with pyrexia. In one case Dr Richardson ('Trans. St And. Med. Grad. Ass.,' 1869) found the pulse intermittent in an infant on the day of birth, and this condition lasted for five years, after which it gradually disappeared; in another case he found it present in a boy five years old, who afterwards became entirely free from it. In adults, as a rule, it is due to some one of the complex conditions which are included under the term dyspepsia. Dr Balfour, indeed, is disposed to think that it is rarely dependent upon any form of indigestion except that which is associated with a gouty tendency. But in this few will agree with him. A circumstance observed with regard to this kind of pulse seems to be very suggestive as to its mode of production. As is well known, intermittence

* "Such trivial causes will occasion them (intermittent pulses) that they are not worth regarding in any illness, unless joined with other bad signs of more moment" (Heberden).

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of pulse, when the result of dyspepsia, is very apt to come on after the patient lies down in bed. Now, the author has noticed in his own person —and patients have assured him that they have noticed the same thing— that when the pulse is intermittent overnight, it often is so on the following morning also, although it becomes regular after one has risen from the recumbent posture. The probable explanation seems to be that what causes the heart's action to intermit is the presence in the stomach of solid pieces of food imperfectly masticated. It is well known that lumps of potato and other substances may remain in the gastric cavity for days together; and nothing seems to be more likely than that when one lies down such masses should fall from the greater curvature of the stomach towards the cardia, and should consequently irritate the terminal branches of the vagi spread over that part of the mucous membrane.

In some persons, intermittence of the pulse is produced by drinking green tea; and in others tobacco-smoking has a like effect. Dr Walshe cites a case in which a shot, lodged in a bronchial tube, set up asthmatic seizures and rendered the pulse extremely irregular, both symptoms disappearing when it was expectorated. In many persons intermittence of the heart's action appears to be traceable to the shock of some emotion, such as sudden terror or grief. Dr Richardson and Dr Balfour allude to cases in which it was set up by a railway accident or by a shipwreck; and the former relates the cases of two patients in whom intermittence of the pulse preceded an attack of mania, in one of them on several different occasions.

Irregularity.—In some cases the beats of the heart are perpetually varying among themselves, not only in their character, but also in the rate of their repetition. The ventricle occasionally makes a series of short ineffectual contractions in rapid succession, causing what the patient describes as a fluttering sensation within the chest. As Dr Balfour remarks, this may occur only at very long intervals, perhaps not oftener than once in several months.*

Even the combination of extreme irregularity with frequent intermittence of the heart's action is sometimes a much less serious matter than might have been expected, especially in persons no longer young, who are able to lead quiet lives, and are neither called upon for active exertion nor compelled to endure the pressure of emotion. Such persons often continue to live for years, and pass their days happily and usefully to others. This kind of cardiac disorder, however, may be a symptom of dangerous organic disease; even when no murmur can be detected with the stethoscope, there is always the possibility that some degenerative change in the walls of the left ventricle may be present.

As a rule, irregularity of the pulse should be regarded as of more importance than mere intermittence. There is, however, one kind of irregularity which signifies nothing, though it has been made the subject of grave consideration when observed in a candidate for life insurance: viz. a temporary acceleration of the pulse, for perhaps ten or twenty beats at a time, with

* A curious point, confirmed by the author's personal experience, and by the statement of at least one of his patients, is, that when a person is accustomed to having his attention drawn to simple intermission of the pulse by slight feelings of palpitation in the left side of the chest, similar feelings may be occasionally experienced without any intermission taking place. Is it possible that there was a momentary contraction of fibres of an intercostal muscle, in obedience to some association of the superficial and deeper structures analogous to that pointed out by Van der Kolk and by Hilton?—C. H. F.

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subsequent slackenings, occurring in nervous persons, while one has one's fingers on the wrist, as the result of transient waves of excitement or uneasiness under medical examination.

Irregularity of the pulse, as a constant symptom, is associated with mitral incompetence, and the later stages of all forms of cardiac disease.

It is also an important sign of failure of the ventricle in cases of enterica and other fevers, in pneumonia and in peritonitis.

It is met with in some cases of flatulent dyspepsia, particularly when due to tea-drinking, and is a well-known result of the excessive use of tobacco.

Allorrhythmia.—In some cases disturbance of the heart's rhythm shows itself, not in ordinary irregularity nor in intermission, but in the regular succession of beats of different force, or in the regular coupling together of beats in pairs. The former variety is named by Traube the *Pulsus alternans*, the latter the *Pulsus bigeminus*. By Sommerbrodt ('Deutsches Arch.,' xix) they are included together under the common name of "allorrhythmia." In English we might use the terms "grouped pulse" to denote a more or less constant succession of similar irregular contractions, and "doubled" or "twin" pulse for the more or less regular succession of a strong and a weak contraction.

An example of the *pulsus alternans* was recorded by the author in the 'Guy's Hospital Reports' for 1871, vol. xvi, p. 330. The patient was a woman of thirty, who came into the hospital with mitral stenosis (a condition not infrequently attended by *pulsus bigeminus*), denoted by a well-marked presystolic murmur. The usual rate of her heart's action was about 70. But sometimes it would rise to 92, and then only every other beat produced a pulse at the wrist, which accordingly was counted as 46; there was, however, reason to believe that the beats which failed to reach the radial arteries were attended with reflux into the systemic veins, inasmuch as a pulse could be felt at the root of the neck, apparently in the jugulars. It is worthy of notice that 70 is almost exactly the arithmetical mean between 46 and 92, for this fact perhaps justifies the conclusion that the efficiency of the alternate beats which were not felt in the arteries was just half that of the regular beats. At one time the allorrhythmic state of the pulse could in my patient be stopped at will, by making her walk so as to quicken the heart's action; the rhythm was then normal, but afterwards, when the heart began to slow again, it fell into the peculiar alternate rhythm. Digitalis, too, seemed at one time to be concerned in producing it, as has been suspected also in some cases of the *pulsus bigeminus* recorded by German observers. An allorrhythmic state of the heart's action, however, is not peculiar to cardiac affections; it has been observed in cases of cerebral hæmorrhage or softening (hitherto only when the right side of the brain was affected ?), and also in one case during convalescence from acute peritonitis. How it is produced is not yet clear. Traube supposed the *pulsus bigeminus* to be a sign of the near approach of death, but on this point he was entirely wrong. This is clear from the cases that have been recorded by different observers; the patient above mentioned lived ten or eleven years after the publication of her case. Sommerbrodt compares this state of the heart with the modification of breathing associated with the names of Cheyne and Stokes; and the two conditions are sometimes found together, as in a case now under the writer's observation. Just as the latter depends upon rhythmical changes in the degree to which the respiratory centre is stimu-

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lated by the circulating blood, so the former may perhaps result from a like influence of the blood supplied to the cardiac ganglia. It is to be noted that when the pulse is at times allorrhythmic, it is apt at other times to be arrhythmic, *i. e.* irregular and intermittent. Allorrhythmia has been frequently observed as a sequel of epileptic attacks, and Tripier, of Lyons, has collected a large number of these cases.

Palpitation of the heart.—In ordinary circumstances, and in a state of health, we are unconscious of the heart's action—if one becomes aware of its pulsations, palpitation is said to be present. This does not always denote permanent cardiac disorder; it may occur to any person after great exertion, or under strong emotion. But, apart from such causes, it may be due to various kinds of disturbance of the heart; and it sometimes appears to be the chief or even the only thing that a patient complains of, and thus constitutes a disease by itself. In some cases in which the cardiac pulsations seem to the patient to be extremely violent, the physician may find, on placing his hand over the left side of the chest, that they are in reality perfectly quiet and natural in character. Much more often the subjective sensation corresponds with the fact that they are greatly increased in force, so as to shake the chest, or the entire body, or even the couch on which the patient lies. This distressing palpitation may be accompanied by violent throbbing of the carotids, a sensation as though "the heart were jumping into the throat," or "the eyes bursting from the sockets," flashes of light before the eyes, dizziness, faintness, or an indescribable sense of discomfort in the region of the heart, rarely amounting to actual pain. The attempt to lie on the left side often greatly aggravates the symptoms. There is often extreme anguish with a fear of impending death. On examination the apex-beat is seen to be in its natural position, but it occupies too extensive an area. Dr Walshe describes the impulse as feeling like a *blow*, with even somewhat of a heaving character, if the heart is well nourished; like a *slap*, if the organ is feeble. The area of percussion dulness is usually normal in extent, but in prolonged paroxysms there may be some increase of it to the right of the sternum. The sounds are loud and clear, with a metallic ringing character; the first sound can sometimes be heard at a distance of some inches from the chest. Dr Walshe speaks of a basic systolic murmur as of frequent occurrence in patients who are in the slightest degree anæmic. He is also disposed to suspect that a transitory condition of mitral regurgitation may be present, causing a systolic murmur at the apex; but one ought to scrutinise very carefully the relations of any such murmur to the respiratory rhythm, for cardiac palpitation seems extremely likely to give rise to a peculiar form of murmur which will be described elsewhere as resulting from disturbance of the edge of the left lung by the heart's movement. The pulse is not necessarily increased in frequency; Dr Walshe says that in vigorous and plethoric subjects its rate may be normal or even below normal.

Palpitation of the heart is commonly present in various forms of organic cardiac disease, and the physical signs are then of course modified by those which belong to the particular affection that may be present. It may also accompany other forms of functional or neurotic disturbance of the organ; in Exophthalmic Goitre it is one of the principal symptoms. In these circumstances it may be more or less persistent. On the other hand, when it occurs by itself, it is usually paroxysmal. The duration of an attack may then be from a few minutes to several hours. Dr Walshe remarks that it

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may terminate with an abundant flow of watery urine, and that as it subsides the patient often falls asleep. Palpitation about puberty is often due to the heart not developing in proportion to the bodily growth.*

It is especially during early adult life that palpitation is apt to occur as a substantive disease. Nervous hysterical women are very liable to it; but the most severe cases of all are seen in excitable youths. It may be the result of exhaustion from over-study, from sexual excesses or masturbation, or from the abuse of alcoholic stimulants, of tobacco, or of strong tea. But in some cases none of these causes can be made out. It is then often due to overloading of the stomach with imperfectly masticated food, especially shortly before bedtime. It is probable that, like intermittence of the pulse, palpitation may have such an origin, even when there is no sense of fulness or discomfort at the epigastrium, nor any other of the common symptoms of dyspepsia. It is especially when the affection recurs night after night, when the patient is in bed, that this cause should be suspected. In other cases it comes on during exertion, as when the patient begins to walk, even slowly and on the level. Dr Balfour says that one characteristic of the nervous or functional nature of the palpitation in such circumstances is that it disappears if he exerts himself a little more, as by taking a short run. In other instances palpitation may be noticed to subside under any kind of pleasurable emotion.

Treatment of palpitation and irregular pulse.—In ordinary cases functional disorder of the heart is often due to overloading of the stomach, and regulation of the diet is essential. Moderation in the use of stimulants should be insisted on. Neither tobacco nor tea must be used, for both are undoubtedly frequent causes of cardiac palpitation; all forms of excitement, including sexual indulgence, must be kept under control. When there is anæmia, tonics, such as iron, zinc, or quinine, may be prescribed with advantage.

Among drugs intended to affect the heart directly, digitalis is by far the most useful. This was Da Costa's experience, and it is in entire accordance with that of English physicians. Occasionally, however, like every other drug, it fails either in diminishing the frequency of the pulse, or in correcting its irregularity. In cases of palpitation one might have doubted, on theoretical grounds, whether digitalis would do good; but clinical experience is in its favour. Other medicines which may be of service are the bromide of potassium or of ammonium, hyoscyamus, cannabis indica, ammonia, and almost any form of ether, but particularly the spirit. ætheris comp. In hysterical females, assafœtida, musk, and valerian are useful. Schrötter recommends the application of cloths wrung out in cold water, or even of an ice-bag; but Dr Walshe says that this is a dangerous practice, especially if the cardiac rhythm be at all affected.

For palpitation accompanied by great irregularity and intermittence of the pulse it is agreed by all writers that alcohol is one of the best remedies. A drachm to half an ounce of brandy should be given, according to circumstances, but with due sense of the risk of dram-drinking becoming a habit. Digitalis is often of great use in steadying the heart's action; and belladonna or atropine is believed to do good, particularly in cases in which the pulse intermitted.

Aconite seems to be of little or no service in cases of mere functional

* See Bowditch on "The Growth of Children," and Beneké's observations on the volume of the heart at different ages, quoted by Dr Pitt ('Brit. Med. Journ.,' Nov. 27th, 1886).

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palpitation, but Da Costa obtained striking results with it when the heart was beginning to undergo hypertrophy. It often exerted a marked influence upon the force of the cardiac beats without diminishing their frequency, whereas exactly the opposite effect was produced by digitalis. Consequently, in suitable cases the two drugs were given together with great advantage. Veratrum viride seemed to be intermediate in its action between them.

Tension of the pulse.—An important character of the radial pulse is the resistance which it offers to compression by the fingers. This was well recognised by the older physicians, who used the names *pulsus durus* and *pulsus mollis* to denote the degrees of tension, and called a small incompressible pulse "wiry," a small and compressible one "thready." These terms were still used after increased attention was given to the symptoms of disturbed circulation on the introduction of cardiac auscultation; but they were not better understood until the researches upon arterial blood-pressure by Ludwig and the subsequent invention of the sphygmograph by Marey gave more definite meaning to the terms previously in use.

Even now we have no trustworthy method of measuring the blood-pressure in an unopened artery. Neither the pressure necessary to obtain a sphygmographic tracing nor, beyond a certain point, the characters of the tracing, will tell us this.

The tension felt by the educated finger placed on the radial artery depends first on the force of the left ventricle behind, secondly on the resistance in the capillaries and contracted arterioles in front; and this feeling of pressure or tension will be modified by the softness and elasticity or the hardness, rigidity, and thickness of the walls of the radial artery.

The highest tension is commonly found in the most chronic form of Bright's disease when there is increased resistance in front and a hypertrophied ventricle behind. But high tension is also found in many cases of early and acute nephritis, such as occurs after scarlatina, and in a less degree in other acute inflammations, especially serous and parenchymatous; likewise in cases of gout and plumbism, and in many epileptic patients. It is also met with during pregnancy, in some cases of chlorosis, and in full-blooded, free-living patients, who find constant relief in blue pill, colchicum, and a purge.

A low tension with a relaxed artery is characteristic of pyrexia generally, particularly of the specific fevers, and of acute rheumatism.

The pulse is large or "full," with high tension, in some cases of pneumonia and meningitis, and in early cases of acute Bright's disease. A more common condition is the short, full pulse with low tension, which, when fully developed, becomes the collapsing or "water-hammer" pulse of Corrigan.

A small pulse with high tension, shown by the length of the systolic expansion and the slight degree of the diastolic fall, is the opposite of the last variety, and constitutes the "persistent" and hard pulse of chronic renal disease.

A small, weak, and compressible pulse, which is also irregular, is one of the characteristic symptoms of mitral regurgitation.

The same hardness which is felt by the finger in the contracted and thickened artery of Bright's disease may be produced by calcareous (or even ossific) degeneration of the middle coat of the radial artery, or by the more common atheromatous degeneration of large arteries. The visible pulsation and emptiness during diastole, which in young subjects with

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elastic arteries is indicative of aortic imperfection, denotes in older persons a want of elasticity of the arterial coats, due to degenerative changes.

The pulse of the radial artery may be always weak, and its lumen easily compressible, without there being any lesion of the heart or any deviation from health. Hence "a poor pulse" is often a deceptive guide in a patient seen for the first time, or in a candidate for life assurance. A short, "slapping" pulse—*pulsus celer*—is associated with low tension, and is often felt after depletion by hæmorrhage or purgation.

The pulse of high tension may often with advantage be *treated* by purging and moderate blood-letting; the soft pulse often calls for stimulants and steel. But as in other cases, such indications are only to be followed when the primary disease has been ascertained.

Absence of the pulse (which would be etymologically denoted by the word asphyxia) is not only observed in syncope and collapse when the heart's beats are too feeble to be felt at the wrist, but may be the result of peripheral causes affecting one or both radial arteries.

Weil has described the pulse disappearing in inspiration at one wrist only, and has noticed the same, less frequently, coincident with deep expiration. He thinks that this may depend upon adhesions of the subclavian artery with the pleura and the dome-shaped process of cervical fascia at the root of the neck.

Hamburger remarked a very singular fact with respect to the pulse. If a healthy person throws the shoulders well backward, and the arms backwards and downwards so as to cross the hands over the sacrum, on deep inspiration both radial pulses disappear for the time. Hyrtl explained this as due to compression of the subclavian artery by the first rib, and Hamburger believed that when this is not the case, it is due to perichondritis fixing the first cartilage, a frequent concomitant of phthisis with pleurisy of the apex.

If the two radial pulses differ markedly in size or in force of beats, there is either some abnormal distribution of the arteries, or obstruction from a tumour pressing from without, or atheroma or some other lesion within. In many cases the tumour or internal lesion is an aneurysm, and this may either act by pressing on the artery or may diminish the flow of blood, and thus the size of the artery, by diverting it from its normal channel.

*Syncope.**—The symptoms of fainting or failure of the heart vary greatly in degree. In the most severe cases the patient experiences distressing sensations of giddiness, nausea, and sinking at the epigastrium. He turns cold and pale, and breaks out in a clammy sweat. His sight becomes dim, and everything appears black. He hears rushing noises in the ears. More or less rapidly he becomes completely insensible. His pulse is frequent and very weak; and it soon ceases to be felt at the wrist, though it may still for a time be counted in the carotids. The heart's impulse grows more and more feeble, until it may be no longer perceptible. Still, however, one may be able to hear with the stethoscope that the organ continues to beat, though the sounds are very feeble, and the second one is audible only at the base. The respiration, in the meantime, becomes infrequent, irregular, and shallow, and the pupils are dilated.

In slighter cases, after having suffered for some minutes from giddiness

* *Syn.*—Lipothymia—deliquium animi. Συνκοπή is the regular term for a faint or swoon in Galen, Aretæus, and Plutarch; λιποθυμία is used in the same sense by Hippocrates.

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and nausea, the patient just for an instant loses himself more or less completely, and then gradually recovers. Sometimes he remains unconscious for a considerable time. Dr Walshe disbelieves in the possibility of recovery after the sounds of the heart have ceased for so long as five minutes. But it is not uncommon for "fainting fits," with an imperceptible pulse and very feeble cardiac action, to last for an hour or longer, and yet to end favourably. The subsidence of an attack is ushered in by "gasping, or rather sighing, respirations at long intervals, and by gradual return of pulse, consciousness, and colour. Sometimes vomiting or dischage of flatus, convulsions, or profuse perspiration takes place at the time of returning consciousness." Dr Walshe believes that these symptoms are generally painful and distressing, whereas in many cases the passage to unconsciousness is pleasurable.

There are other instances in which the stoppage of the pulse and the interruption of the mental faculties are absolutely sudden, and in which the resumption of the heart's action and the recovery of consciousness are no less instantaneous, while there are no subjective sensations whatever. But although seizures of this kind are spoken of as "fainting fits" by unprofessional persons, they probably are always cases of the minor form of epilepsy.

One great distinction between the attacks which are *epileptic* and those which may properly be referred to fainting, is that the former generally, if not always, occur without any definite exciting cause. On the other hand, true syncope is, as a rule, clearly traceable to some disturbing cause, though it may be apparently of a trifling character; the heated air of a crowded room, the sight of blood (even from a cut finger), the strong odour of flowers, the introduction of a catheter, may each cause fainting in certain persons. It is most apt to occur in young adults, and in women rather than in men, but it may frequently befall healthy and vigorous youths. A person feeling faint can usually sit or recline, she droops rather than drops; but an epileptic attack is more sudden, and hence the face and head are more often injured by a fall.

Among the causes of the graver forms of syncope, such as are apt to prove directly fatal, are hæmorrhage and various organic diseases of the heart and aorta, pulmonary embolism, and occasionally the too rapid withdrawal of ascitic or pleuritic fluid by tapping.

In syncope due to hæmorrhage there is a waxy pallor of the face and lips, of the hands and finger-nails, and of the whole surface. Consciousness is more gradually lost, though much depends upon the rapidity of the bleeding. Delirium is often present, and occasionally epileptiform convulsions occur.

Sometimes the patient lapses from time to time into a state of insensibility, regaining consciousness in the intervals of these "fainting fits." They are especially apt to arise if the patient attempts to sit up, and at the same time the pulse at the wrist becomes more feeble, or even imperceptible. After death from hæmorrhage the heart is found contracted and empty.

The physician sees this form of death in cases of hæmoptysis purpura, intestinal hæmorrhage, and ruptured aneurysm.

Syncope must be clinically distinguished from the condition known as *collapse*, although both are accompanied by failure of the heart, and their pathological difference is not clear.

In collapse, beside extreme feebleness of the cardiac action, the pulse at the wrist becoming imperceptible, and the cardiac sounds being scarcely audible, the surface of the body, and especially of the hands and feet, is

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cold and death-like; the features are sunken, the eyes retracted in their sockets, and the orbits surrounded by deep brown rings. Yet the patient is often entirely free from sensations of giddiness, nausea, or faintness. He may still have considerable muscular power, may get out of bed, and even walk for some distance.

Either fainting or collapse may result from affections of the abdominal organs, and is then probably due to reflex inhibition from the solar plexus. But either of them may also be due to other causes; collapse, for example, occurs in certain cases of diphtheria, and in those of pulmonary embolism.

The "cardiac" variety of *sunstroke* must also be mentioned. In this the sufferer gives no sign of illness until he falls, gasps, and perhaps at once expires before anything can be done to help him. Dr Maclean says that this is the form most often seen in soldiers exerting themselves in the heat of the sun when dressed and accoutred (cf. vol. i, p. 788).

Death by syncope may, from organic disease of the heart, be absolutely sudden; the patient falls to the ground or sinks back in his chair unconscious, and is dead before help can be given. The respiration may cease at the same instant as the beats of the heart, or a few deep gasps occur, and then a kind of shiver passes through the frame, pallor spreads over the surface, and all is over.

It is often impossible, in such cases, to determine whether paralysis or spasm of the heart is the cause of death. If an autopsy is made its chambers may be found either relaxed or contracted, either empty or more or less full of blood. But it is difficult to say how far its state may be modified by contraction of the ventricular walls after death. If they are flabby, while the muscles generally are in a state of rigor mortis, it seems fair to conclude at the time when life became extinct the heart stopped in a condition of diastole. On the other hand, stoppage in systole is not necessarily due to spasm; it may be the result of pressure upon the outside of the heart, as when the pericardial sac becomes filled with blood by rupture of the aorta. An appearance which is not seen when death begins at the heart is an overloading of the right cavities with blood while the left are empty, as in cases of asphyxia.

The causes of sudden stoppage of the heart are various. There may be disease of the heart itself, or of its valves, or of the base of the aorta; and in many cases of this kind it is possible that lesions of the nervous ganglia situated near the base of the organ might be discovered if carefully sought for. In animals powerful stimulation of one of the *nervi vagi* arrests the cardiac beats; a similar effect follows irritation of the cardio-inhibitory centre in the bulb; frequently it is the result of direct or reflex inhibition through the vagi; and, lastly, it can be produced as a reflex effect by a violent impression upon peripheral nerves, as by suddenly crushing the foot or striking the exposed intestine with the handle of a scalpel in the frog (the *Klopfversuch*), or even by gentler stimulation when peritonitis has first been set up.

In human pathology all these varieties of inhibition seem to occur more or less frequently.* That of which in medical practice we know least is

* If the analogy with the inhibitory phenomena observed in the physiological laboratory can be looked upon as tolerably complete, it is an interesting question whether in any circumstances the supervention of collapse or of fainting can be prevented by the injection of atropine, the effect of which in animals is to completely annul the normal inhibitory action of the vagus.—C. H. F.

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perhaps direct inhibition by stimulation of the vagus. Czermak, however, was able at will to stop the beating of his own heart by pressing the trunk of the pneumogastric nerve against a small osseous tumour in the neck. In cases of aneurysm of the aorta, it is not uncommon for sudden death to occur without the autopsy revealing a rupture of the sac or any other definite change; in such cases the fibres of the left vagus nerve are often spread over and inseparably blended with the walls of the aneurysm; and this may perhaps cause the arrest of the cardiac pulsations. Inhibition of the heart by stimulation of the centre in the bulb probably occurs when fainting is caused by emotion. Whether intracranial diseases ever destroy life in this way is doubtful; if so, one would expect the heart's beats to cease before the respiratory movements.

Death by reflex inhibition appears to be of frequent occurrence. There can be little doubt that the "fatal shock" which accompanies severe injuries of the limbs or of any part of the body is of this nature. But what is most striking is that abdominal diseases are specially apt to be attended with sudden death. In the collapse of acute peritonitis death is often sudden. Just as in the *Klopfversuch* cases of sudden and fatal syncope from slight blows on the epigastrium are not uncommon. The classical instance is that recorded by Sir Astley Cooper of a sailor, who, while playing with a shipmate, and lifted on his shoulders, received a tap on the abdomen which caused immediate death.

In the *treatment* of a faint, the first thing is to place the patient in the open air or near a window and to make him lie down with the head as low as the shoulders; the clothes must be loosened about the throat and chest, and the crowd of sympathising friends must be sent away. A bottle of ammonia may be held to the nostrils; or, if this be not at hand, a bunch of feathers may be burnt and the fumes inhaled. Cold water may be poured upon the face, and if the stomach be overloaded, an emetic of mustard should be administered; for this, as Anstie says, has a powerfully rousing influence upon the heart. Sal volatile, brandy, ether, or a tumbler of cold water may be given by the mouth; or, in severe cases, when the patient continues unconscious, fifteen or twenty minims of either ether or brandy may be injected hypodermically. Our house physicians frequently adopt this practice, and in many cases with marked success in stimulating the action of the heart, although the result is too often temporary. Another method, advanced by Dr J. C. Reid ('Brit. Med. Journ.,' 1880, vol. ii, p. 1014), is that of pouring hot, but not scalding, water over the præcordial region. He cites a case in point that occurred in an old man of seventy, who was thus restored from apparent death, and lived for many years afterwards.

It is not clear whether galvanism can be used with advantage in such circumstances. Ziemssen recently found ('Deutsch. Arch.,' xxx, 1881) in a patient whose chest wall was deficient so that the heart was covered only by the skin, that the heart's beats could be accelerated by powerful currents. Erb recommends that in galvanizing the heart large electrodes should be used, one being applied over the surface of the organ, the other over the dorsal vertebræ; a current of high intensity should then be passed, its direction being reversed seventy or eighty times in the minute. Previous observers, as cited by Walshe, had found that with strong currents there was a risk of inhibiting the cardiac contractions instead of stimulating them. Although it were admitted that, if the irritability of the organ

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were lowered, a powerful current, which, under ordinary circumstances would have arrested its action, might perhaps have the effect of reviving it, it would be advisable first to make trial of a feeble current, and afterwards to increase its strength. But in the cases now under consideration there is seldom time for such a method.

ANGINA PECTORIS.*—Under this name is known a very severe form of cardiac pain, occurring in sudden short paroxysms, accompanied by a sense of impending death, and often immediately fatal.

It was first described by Heberden† as follows :—"There is a disorder of the breast marked with strange and peculiar symptoms, considerable for the kind of danger belonging to it and not extremely rare, which deserves to be mentioned more at length. The seat of it and sense of strangling and anxiety with which it is attended may make it not improperly be called Angina Pectoris. Those who are afflicted with it are seized while they are walking (more especially if it be uphill and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life if it were to increase or to continue; but the moment they stand still all this uneasiness vanishes. In all other respects the patients are at the beginning of the disorder perfectly well, and in particular have no shortness of breath, from which it is totally different" ('Commentaries on the History and Cure of Disease,' 1782, chap. lxx, p. 364).

In its well-marked and typical form, angina pectoris is a rare malady. Cases are not infrequent, which more or less closely resemble it in the character of the pain, although in these there is not the same danger to the patient's life. Whether or not they should be classed under the same heading is a question that can be satisfactorily discussed only after the pathology of the affection has been considered. The following description refers to the classical form of the disease.

Onset and exciting causes.—Angina pectoris is almost always absolutely sudden. The first attack usually occurs while the patient is walking, especially on rising ground, or with a strong wind against him, or shortly after a meal. But sometimes (as in the case of Arnold, of Rugby, recorded by the late Dr Latham) a person who has never suffered from angina before is awakened from his sleep by the first attack. Subsequent seizures are apt to be brought on by comparatively slight causes, until the patient gradually finds that one form of exertion after another is unsafe for him. Emotional excitement is a powerful exciting cause, as in the well-known case of John Hunter, who died within the walls of St George's Hospital in the midst of a dispute, of which he had foreboded the fatal result. In many instances the seizures, which at first took place only during the daytime, afterwards begin to recur also in the night. Dr Latham alludes to

* *Synonyms.*—Pectoris dolor—Breast-pang—Syncope anginosa (Parry)—Asthma dolorificum (E. Darwin)—Cardiodynia—Neuralgia cordis (Laennec)—Hyperæsthesia plexus cardiaci (Romberg)—*Fr.*, Angine de poitrine—*Germ.*, Brustbräune.

† His first description was in the 'Medical Transactions' of the College of Physicians for 1768. Some French writers have set up a claim of priority for one of their countrymen, Rougnon, on account of a letter written by him to Lorry a few months earlier, in which is related the death of a cavalry officer, M. Charles, by what was probably the same disease. But so far as the account of a single case can be held to have anticipated Heberden's observations, the merit really belongs to Morgagni, who recorded a similar instance in a Venetian woman early in the eighteenth century, quoted by Dr Gairdner ('Reynolds' System,' iv, p. 537).

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one patient who was attacked as soon as he lay down. The act of stooping, as to pull on the boots, or even in washing, is a frequent cause of the attacks of angina. In some cases they are brought on by such slight efforts as coughing, defæcation, or the hasty swallowing of cold water.

Symptoms.—The chief seat of the pain in angina pectoris is usually behind the lower part of the sternum, rather to the left than the right side; sometimes behind the middle or the upper part of that bone. Patients attempt to describe it, as gnawing, tearing, stabbing, or lancinating in character. But in many cases the agony is indescribable—so intense as to make the sufferer feel that unless it abates, it must bring his life to an end. The pain often spreads round, generally through the left side of the chest, to the spine. It is sometimes accompanied by a sense of constriction, as though the sternum were forcibly drawn backwards: Dr Gairdner cites the case of a medical man in whom there was a subjective sensation as though the front of the chest were "bulged out in a convex prominence, terminating suddenly at the lower end of the sternum in a sharp and deep depression." Very often the pain radiates upwards into the neck or towards the occiput, down the left arm to the elbow or the fingers, occasionally down the right arm, and less frequently still it shoots into the lower limbs or into the testes. In these distant parts it may be accompanied by feelings of tingling or of numbness. In a case observed by Dr Walshe, it took a course the reverse of what is usual, beginning at the left wrist and extending upwards to the heart. In the præcordial region there is often tenderness to pressure; but sometimes friction gives relief.

A patient attacked by a fit of angina is instantly arrested in whatever he is doing; if walking he stops motionless; if standing, he dares not sit down. It is, however, a curious fact that some persons, after having been pulled up by the pain three or four times at the beginning of a walk, will afterwards go on with ease for several miles. The feeling of constriction in the chest may cause the patient to speak of experiencing a "want of breath" or a "sense of suffocation;" but all observers are agreed that there is no dyspnœa in the proper sense of the term, and no lividity of the face. The breathing may be somewhat increased in frequency, but this is because the patient instinctively keeps the thoracic movements as shallow as possible, for fear of increasing the pain. By an effort of the will he can, if he chooses, freely expand the chest, and there are exceptional cases in which drawing a deep breath gives momentary relief.

With regard to the state of the heart and pulse during a paroxysm of angina pectoris, accounts have differed widely; and probably all cases are not alike in this respect. It is said that the impulse and the sounds of the heart are sometimes unaltered in character throughout the seizure, and the pulse regular and neither frequent nor weak. Walshe has observed that at least towards the close of a paroxysm, when the patient is about to recover, there may be no acceleration nor any irregularity of the pulse. But Parry long ago described the pulse as being more or less feeble, according to the violence of the attack; and Dr Gairdner expresses the same opinion. In many cases it is expressly noted that the pulse has been small and irregular in rhythm, but not always increased in frequency, and sometimes morbidly infrequent. The failure in the circulation is also shown by deadly pallor of the face, by coldness of the limbs, by the presence of clammy per-

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spiration. In cases about to prove fatal, the pulse becomes imperceptible a little while before death.

The mental faculties usually remain unimpaired throughout the seizure; but after its subsidence the patient is said sometimes to have no remembrance of anything except the intense agony which he has undergone. Thus the state of the brain in angina pectoris would appear to resemble that of collapse, rather than of syncope. Walshe says that the sight sometimes fails. There may be slight convulsions, or even tonic spasms of a severe kind, with opisthotonos.

There is in some cases violent and continued eructation, or vomiting; or the whole abdomen may become distended with flatulence. A copious flow of watery urine sometimes occurs as the attack is passing off. Trousseau relates a case in which the paroxysms, though frequently repeated, and though each lasted only about a minute, were always accompanied by an irresistible desire to micturate.

The duration of an attack of angina pectoris is usually a few seconds or minutes; but attacks may recur again and again for an hour or longer. A patient of the author's remained for many hours stooping over the end of a couch, refusing to move for fear of the return of the pain. But sometimes when a seizure occurs during walking, it ceases as soon as the patient stands still. Trousseau remarks that very different attitudes are assumed in different cases. One patient will lie motionless on his back, another will incline backwards on his chair, a third will bend forwards as far as possible, and a fourth may place himself on all fours, resting on his knees and his elbows.

Fatal event.—When angina proves fatal, the heart is found at the autopsy to be relaxed and flabby, even though there is marked cadaveric rigidity of the muscles generally. Walshe says that there is an almost complete absence of blood from the cardiac cavities, which fact certainly looks as though a ventricular systole had been the last act of life.

Sometimes death appears to be absolutely instantaneous; Walshe relates an instance in which the patient had been reading quietly in bed, and in which the thumb and the forefinger were found in the pamphlet on which he had been engaged, the bedclothes being also quite undisturbed. The same quietness of attitude was observed in the case of Dr Chalmers which Gairdner describes as an undoubted case of pectoral angina. In some of these cases, even where there have been former attacks attended with severe pain, it seems probable that the fatal seizure must have been so brief as to be painless; and one may perhaps suspect that sudden death, in persons who have never been known to suffer from angina, is often of the same nature.

Sometimes death is more gradual, being preceded by increasing failure of the pulse, laboured breathing, and unconsciousness. We have seen that, as a rule, the paroxysms of angina pectoris return again and again; there is generally an interval of some years between the first attack of the disease and its fatal termination. Whether a single paroxysm ever occurs without being followed by any others, seems to be doubtful; though the patient himself may do much to prevent their recurrence by avoiding exertion and emotion. Walshe speaks of having himself seen a patient who appeared to have been first attacked twenty-four years previously; and there is some reason to believe that John Hunter had begun to have seizures of angina twenty years before his death. A still longer and authentic case is one related by Dr Murrell, in which the patient had suf-

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fered for thirty years, the diagnosis of angina pectoris having been formally given by Sir Risdon Bennett twenty-six years before. In some instances the disease returns frequently, so that the number of paroxysms must be large. On the other hand, Walshe relates an instance in which there were only three, one being a year, and the other half an hour, before the third which proved instantaneously fatal. Latham met with two cases in one of which death occurred fourteen days, in the other ten days, after the first attack. The most rapid (not instantaneous) case on record is probably that of Dr Thomas Arnold, who, having never suffered from angina before, went to bed on the 11th of June, 1842, in apparent health, awoke in a paroxysm of pain at five in the morning, and died about a quarter past seven.

Pathology.—The pathology of angina pectoris is still obscure. Following Laennec (who called it *névralgie du cœur*), Romberg, Friedreich, and Trousseau maintained that it should be regarded as a neurosis, or a "visceral neuralgia."* Anstie declares that those who suffer from the disease are always of nervous temperament, and that other neuroses are frequent in their families; and, like Trousseau, he insists on the existence of a relationship between angina pectoris and asthma. On this view angina pectoris might be classed with the paroxysmal neuroses.

But for a neuralgia to prove habitually fatal is without precedent. Moreover, angina pectoris differs from all neuroses in being generally, if not always, associated with the existence of organic lesions in the heart or in the great vessels, although it would seem that no one lesion is constantly present; this, at any rate, is true of the cases that destroy life. Thirdly, it is unlike a neuralgia to attack, as angina does by a large preponderance, more males than females—as many as ten men to one woman.

Within the last few years observations have been made of organic lesions affecting the cardiac nerves and ganglia in cases of angina pectoris. The earliest record of such changes is fifty years ago, when Heine ('Müller's Archiv,' 1841) published a case treated by Skoda, in which Rokitansky made the autopsy, and found the right phrenic nerve, the *N. cardiacus magnus*, and the descending branches of the left vagus, each involved in pigmented nodules, doubtless altered lymph-glands. In this case, however, the symptoms during life consisted, not in paroxysms of angina, but in attacks of intermittency of the heart's action, continued during a period of from four to six beats, and attended with a feeling of inexpressible anxiety. In 1864 Lancereaux ('Gaz. Méd.,' 1864) had an opportunity of examining the body of a man who had long suffered from angina pectoris, and who had at last died suddenly; he found a raised patch in the aorta between the orifices of the coronary arteries, with injection and thickening of the corresponding part of the external coat of the vessel; in this injection the adjacent cardiac plexus took part, and some of its fibres were surrounded by a nucleated material. Similar changes in the nerves (of what ever importance they may be) are recorded and figured by Peter ('Traité des Maladies du Cœur,' 1883) in two cases which came under his care, and he cites a fifth instance ('Bull. de la Soc. Clin.,' 1878).

Since the year 1867, however, clinical experience has taught in a very

* The same opinion is formally upheld by Eulenburg in 'Ziemssen's Handbuch,' but perhaps a more correct statement would be that he rejects it, since he would exclude from the disease, in its "purely nervous" form, the very cases involving danger to life, on which the foregoing description is based, and which from the days of Heberden to the present time have been regarded as the true and typical examples of it.

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decided manner that the immediate exciting cause of the paroxysms of angina pectoris is a sudden *rise in the tension* of the systemic arteries. A similar view had been previously suggested by Traube. But what first proved its correctness was a series of observations made by Dr Lauder Brunton on a patient in the Royal Infirmary of Edinburgh. The man was affected with aortic regurgitant disease, and was liable to frequent attacks of angina-like pain. Dr Brunton found that during these attacks the sphygmographic tracing of the pulse became rapidly altered, the wave being broader and lower, the dicrotism disappearing, and both the ascent and the descent being more gradual—changes indicative of a marked increase of arterial tension. These observations led him to propose the inhalation of nitrite of amyl as being likely to relieve the pain by relaxing the peripheral arteries, and the trial was attended with brilliant success. The pallor and the coldness of the face and of the limbs that accompany severe seizures can hardly be cited as evidence in themselves of arterial spasm, for failure of the heart's action might produce the same effects. Trousseau, however, noticed that the pallor is sometimes followed by a reddish or livid-bluish hue, and the same thing was noticed in one case by Anstie ('Trans. Clin. Soc.,' vol. iii), and attributed by him to paralytic dilatation succeeding spasm of the arterioles.

Under the name of "Angina Pectoris vasomotoria" Nothnagel recorded ('Deutsches Archiv,' iii) a series of cases, which seem to have an important bearing upon this question. Their peculiarity lies in the fact that the earliest and most conspicuous symptoms of the paroxysms from which the patients suffered were coldness and pallor with numbness and stiffness of the limbs; the palpitation, the feeling of oppression at the chest, the giddiness, the sense of impending death, being all apparently secondary and attributable to the increased efforts which the heart was called upon to make to overcome the peripheral resistance. The attacks were also definitely traceable to external cold, and were relieved by hot foot-baths and frictions; in fact, the state of the peripheral circulation seems to have been very similar to that which in other patients leads to paroxysmal hæmoglobinuria. Pain appears to have been a much less marked symptom than in ordinary angina; however, a dull pain is mentioned, seated chiefly in the cardiac region, but in one case extending over the whole of the left side of the chest, and sometimes down the left arm.

On the whole, Nothnagel's cases prove that a sudden increase of tension in the peripheral arteries due to a cause acting upon the body from without is capable, in some persons, of giving rise to phenomena approaching those of a paroxysm of angina pectoris.

The next question is how we are to suppose the arterial spasm to be brought about in the more usual form of the disease. If the anatomical researches of Lancereaux and Peter are to be credited with the significance attributed to them, nothing seems more simple than to suppose that from the cardiac nerves and ganglia there is transmitted to the vaso-motor centre an impression which causes it to throw the muscular walls of the smaller vessels throughout the body into contraction. But it is necessary to exercise much caution before we adopt such a view. The lesions discovered by the French pathologists were after all in every instance secondary. If the nerves lying adjacent to areas of chronic thickening and induration at the base of the heart (or, indeed, elsewhere throughout the body) were to be systematically dissected, is it not probable that they might often be

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found involved in the adhesions, when no symptoms had existed that could possibly be traced to them? It is surely significant that, like Rokitansky, Peter found the phrenic nerves affected in exactly the same way as those of the heart.

One point in which the paroxysm of angina pectoris seems to differ from what might be expected on the view that it depends upon increased tension in the peripheral arteries, is its not being invariably, or even generally, attended with a reduction in the frequency of the pulse; among Nothnagel's vasomotorial cases there is only one in which a fall from 80 to 64 or 60 beats in the minute is noted.*

It must be remembered that in the severe and dangerous form of angina pectoris, which is regarded as typical of the disease, organic changes in the heart and large vessels are usually, if not always, present. As a rule, the most conspicuous lesion is either a soft, flabby, or fatty state of the cardiac muscle, or else a chronic inflammatory change in the coats of the aorta at or near its origin, leading to atheroma, to calcification, and perhaps to aneurysm; Dr Gairdner has specially insisted upon the frequency with which symptoms of angina accompany even small aneurysms, if they arise very near the heart and project into the pericardium.

There is, however, a lesion specially connected with angina pectoris, which is often associated both with fatty heart and with arteritis deformans of the aorta, namely, obstruction of the coronary arteries. This was first suggested by Jenner, in a letter to Parry, and afterwards in one which he addressed to Heberden in 1778, but which he did not send, lest it should be seen by his friend John Hunter, whom he rightly believed to be at that time a sufferer from angina. Stenosis of the coronary arteries was found in Hunter's case after his death in a fit of the disease. Sometimes the coronary arteries have their orifices more or less completely obliterated by disease of the aorta itself, their coats in the rest of their course being healthy; sometimes they are converted into thick calcified tubes in nearly their whole length.

But it would be an untenable position to maintain that obstruction of the coronary arteries is constantly present in cases of fatal angina, either with or without other more conspicuous lesions of the heart or of the aorta; and it is no less certain that these vessels are often found much narrowed when there were no symptoms of angina during life.

The long duration of the disease in some cases seems to be inconsistent with the idea that any of the organic lesions above described can have existed throughout its whole course. Possibly the paroxysms of angina owe to the lesions in question their severity and their tendency to prove fatal, but do not stand to them in the direct relation of effect to cause. For, if the disease be regarded as a struggle on the part of the heart to overcome an excessive resistance in the arteries, enfeeblement of the cardiac muscle, whether as the result of fatty change or of a mere deficiency of blood-supply, cannot but add greatly to the embarrassment of the heart and the danger of the patient.

Pseudo-angina.—From this point of view it seems probable that many instances of the affection which Walshe describes as "pseudo-angina," and which is admitted to be of far more frequent occurrence, are fundamentally

* Landois's physiological explanation of angina, on which Nothnagel's theory is founded, will be found in Eulenburg's article in 'Ziemssen's Handbuch' (Bd. xii, 2, S. 45—48), and also in Ross's 'Treatise on Nervous Diseases' (vol. i, p. 731).

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of the same nature; but it would still doubtless be necessary to exclude cases dependent upon hysteria or upon flatulent distension of the stomach. Within a single year the author saw two young clerks in the same London bank, each of whom described attacks that appeared indistinguishable from those of angina pectoris, although their age rendered it very unlikely that the affection would prove dangerous to life. But that the age of the patient does not always form a safe criterion is well shown by a case of Dr Balfour's —that of a man, aged only twenty-four, who died after four months' illness with paroxysmal pain in the epigastrium, and in whom (as had been correctly diagnosed during life) the base of the aorta presented a ring of atheromatous thickening, by which the mouths of the two coronary arteries were greatly narrowed. Such a case, however, is altogether exceptional; for, as Dr Walshe says, angina pectoris (exclusive of his pseudo-angina) is rare before the fiftieth. and excessively so before the fortieth year.

Cardialgia.—Pain is a symptom, though far from a constant one, of many affections of the heart, both functional and organic, beside angina pectoris; on the whole, perhaps it is more marked in the former than in the latter. Its most usual seat is a spot a little outside the left nipple, beneath the fourth intercostal space or the fifth rib; but it may be situated nearer the sternum, or to the right of it. Cardiac pain is generally more or less paroxysmal in character, and is often described as sharp or lancinating, sometimes as burning, tearing, or cutting. There may also be a constant dull heavy pain, and in some cases this exists by itself. It is often increased by exertion or fatigue, and it generally undergoes aggravation when palpitation occurs. It often radiates widely over the side of the chest, up into the neck, into the axilla, and down the left arm, less often down the right arm. When it thus affects the arm, it is apt to be associated with a sensation of numbness or tingling; it sometimes ceases abruptly at the inner side of the elbow, sometimes extends down the forearm to the inner side of the hand. In certain cases its principal seat is near the angle of the left scapula. Walshe says that tenderness of the surface is absent; pressure upon the principal spot rather relieves than increases the pain. Da Costa, however, in describing his cases of irritable heart (p. 7), says that the cardiac region was hyperæsthetic, especially after attacks of palpitation.

A modification of cardiac pain described by Dr Walshe is one which comes on when the patient bends forward, as in pulling on his boots, and is relieved by stretching out the chest-wall and pressing on the surface. He has observed it more often in elderly than in young persons. He thinks that it may depend upon "twisting of the præcordial costal cartilages, which have lost the flexibility of youth," but it seems more likely attributable to upward pressure upon the heart through the diaphragm. Possibly this kind of pain ought to be regarded as one of the slighter forms of angina pectoris; at any rate, it is not seldom present in that disease.

Morbid anatomy.—Symptoms of angina pectoris ending in death have been found associated most often with atheroma, thickening, obstruction or obliteration of the coronary arteries.

In Hunter's case (1793), "the coronary arteries had their branches which ramify through the substance of the heart in the state of bony tubes, which were with difficulty divided by the knife, and their transverse sections did not collapse" (Home). There was atheroma and dilatation of the ascending aorta and calcification of both aortic and mitral rings, but the valves were probably competent and the cavities were not dilated. Sir Everard

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Home notes with much emphasis two "milk-spots" on the surface of the heart, but he omitted to weigh it ('Life of Hunter,' by Adams, p. 203).

In Robert Hall's case (1831) Dr Pritchard reported "the muscular structure (of the heart) to be soft, like macerated cellular membrane; the left ventricle was judged to be one third larger than usual. The aorta contained in several places patches of bony matter." The valves are not mentioned, nor the coronary arteries (Memoir by Gregory, note D.).

In Arnold's case (1842) Dr Bucknill and Mr Hodgson found the aorta and all the cardiac orifices healthy. The muscular structure of the heart in every part was remarkably thin, soft, and loose in its texture. The walls of the right ventricle were in some parts not much thicker than the aorta. There was but one coronary artery, which with some difficulty admitted a small director ('Latham's Lectures,' vol. ii, p. 377).

In the case above quoted from Dr Balfour (1877) the heart of the young man weighed thirteen ounces, the cavities were slightly dilated and hypertrophied, the mitral and tricuspid orifices enlarged, the aorta atheromatous, and the coronary arteries "both so extremely contracted as barely to admit the point of an ordinary surgical probe." The muscular structure was healthy to the naked eye, and under the microscope "presented no abnormality, except the presence of a considerable number of reddish-brown pigment granules in some of the fibres" ('Clinical Lectures,' p. 301). In a second case of angina, in a woman of eighty, Dr Balfour found disease of one coronary artery, with dilated ventricle and thin, pale, fatty muscle.

Apart from occlusion of the coronary arteries, angina (or symptoms clinically resembling it) most often appears in cases of atheroma of the aorta, whether leading to aneurysm or to valvular lesions with dilatation of the heart, and very rarely in cases of mitral disease.

Ætiology.—With regard to the remote causes of angina pectoris, as distinguished from the exciting causes of the paroxysms, there is little to add to what has already been incidentally mentioned. There has been some discussion whether the disease is related to gout; probably if any connection exists, it is indirect; arteritis deformans of the base of the aorta is of frequent occurrence in gouty subjects.

It is a remarkable circumstance, which was first pointed out by Sir Gilbert Blane, that angina is much more frequent in the upper classes than among the poor. Gairdner throws some doubt upon the facts given, but there is no question of the general belief. Walshe says that his experience scarcely supplies him with more than a solitary well-defined example of the affection in hospital practice.

It must, however, be remembered that while many physicians have large hospital practice and see few rich patients, with others the reverse is the case; that the fatal diseases of public men excite interest and are fully recorded; and that the attacks of angina are so sudden and pass off so quickly that a patient subject to them would scarcely apply for admission to a hospital. Moreover, the first seizure is sometimes fatal. Hence almost the only cases properly recorded among the poor would be either out-patients happening to be attacked on their weekly visit, or in-patients admitted to hospital with organic disease of the heart or aorta.

However it may be accounted for, the fact remains that, as Sydenham remarked of gout, and as we found it to be true of megrim, an unusual number of the cases of fatal angina pectoris—always a rare disease—which have been recorded since Heberden's paper appeared, have occurred in men

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who were remarkable for their ability. Dr Gairdner has set forth the argument in favour of the philosopher Seneca having been subject to angina. This was the opinion of Sir John Forbes; but the absence of severe pain, and the fact that Seneca died a voluntary and easy death by venæsection at about the age of seventy, surely preclude the inference; rather do the "history of the case" and the patient's own account of his attacks point to chronic bronchitis or possibly phthisis, ending in emphysema with spasmodic asthma; and this was the judgment of Parry and of Stokes. John Hunter undoubtedly died of angina pectoris, and so did Dr Arnold. Dr Chalmers's death during sleep was unlike ordinary angina; but it was doubtless the cause of the last illness and death of a still more eloquent preacher, Robert Hall. A patient who described the distress of an attack as "a sense of dissolution, not the fear of it," was "one of the most gifted men" his physician, Dr Reynolds, "ever knew, and one most competent to analyse sensations."

Excluding, on the one hand, angina-like attacks of pain in cases of aneurysm or disease of the aortic valves, and on the other the exaggerated descriptions of neurotic and hysterical patients, the writer has never seen classical angina pectoris in hospital practice. One typical case occurred in a private patient, a man between fifty and sixty, short and stout, endowed with great energy and ability in business. He had no discoverable disease of the heart or aorta, the lungs, or the kidneys; and after being less than a year subject to attacks of increasing severity, all characterised by cardiac pain shooting into the arms, by a sense of imminent death, and by complete immunity in the intervals, he died in one more severe than any that preceded it (1887). A second case, seen with Mr Edward Acton, in 1890, was in a stout man of seventy-three, with very feeble cardiac action, occasionally irregular or intermittent pulse, and probably extensive atheroma, but with no cardiac murmur. A third, seen with Dr Few, in 1884, occurred in a healthy farmer between forty-five and fifty, with marked symptoms and no signs of organic disease.

"Males are most liable to this disease, especially such as have passed their fiftieth year" (Heberden). Walshe quotes Sir John Forbes's collection of 88 cases: of these 80 occurred in men and only 8 in women, while of 84 patients 72 were more than fifty years old.

In some cases (as Arnold's) a tendency to angina pectoris (or rather, perhaps, to atheroma) has appeared to be transmitted by inheritance. His father died of angina, and his second son of sudden syncope from disease of the aortic valves. Gairdner remarks upon its greater frequency in persons who are corpulent and of sedentary habits. It is, however, to be observed that the liability to attacks of angina interferes greatly with the activity of those who had before been accustomed to take exercise.

Beau described eight cases of angina due to tobacco-smoking; similar ones have been recorded by other French physicians, and one by Eulenburg. Mostly they are not fatal, and belong to Walshe's pseudo-angina; but in one recent case the patient died ('Brit. Med. Journ.,' August, 1887).

A point worthy of notice is that persons suffering under frequent attacks of angina pectoris often have a haggard, frightened aspect; if this needs any explanation beyond the terrible nature of the malady, we may add that their sleep is often broken by bad dreams; some patients are actually afraid to lie down in bed for fear of the occurrence of a seizure.

Prognosis.—It is important to remember that the cardiac affections most frequently associated with angina pectoris are such as it is at present impos-

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sible to diagnose with certainty. Consequently, one must never be led to give a favourable prognosis by the fact that on examination of the chest one has failed to detect evidence of organic disease. Dr Walshe, however, states that in every one of twenty-four cases which had been examined by him during life he was able to make out physical signs of some morbid change either in the heart or in the aorta, or in both. And Dr Balfour says that he has never met with an instance of angina in which signs of dilatation of the heart were not present. Latham's experience, again, was very similar; among thirteen cases there were only three in which neither increased dulness on percussion nor any murmur on auscultation over the heart or the aorta was present; and even in those three cases the cardiac impulse was extremely feeble, while the sounds, "though natural in kind," were "raised to their highest intonation and diffused over the entire front of the chest." In the next chapter, however, we shall have to consider how far signs of dilatation and of other changes in the muscular walls of the heart can be relied on.

Beside being unable to assert, in any case of angina pectoris occurring at a time of life when organic changes in the cardiac muscle are apt to take place, that the disease is free from danger to life, one is also never justified in giving an opinion as to its probable *duration*. A patient's first seizure may have been mild, yet after a longer or shorter interval it may be followed by another of such severity as to prove fatal. Or whereas the first may have been very severe, the subsequent ones may be far less so.

Treatment.—In the treatment of angina pectoris a great deal more can be done than formerly. The older physicians could recommend nothing better than large doses of laudanum and brandy; but now, knowing how slowly absorption from the stomach takes place, we prefer to give *morphia* subcutaneously. Dr Balfour seems to have shown that it is perfectly safe to use *chloroform* freely, so as completely to narcotise the patient. When the pain is very severe this is the only method by which it can be relieved; in protracted paroxysms he follows it at once by subcutaneous injection of morphia, so that the chloroform sleep may pass into the morphia sleep, from which the patient awakes after some hours free from suffering but exhausted, and generally with some œdema of the lungs. It is worth remembering that patients under the influence of morphia take chloroform easily.

As a rule, however, one should rather have recourse to the *nitrite of amyl* or to *nitro-glycerine*. The advantage of the former agent is the rapidity of its action. The best way is to employ the glass capsules, each of which contains from three to five minims of it. One of them is broken within the folds of a handkerchief, and the vapour is inhaled as freely as possible. In from fifteen to twenty seconds the face flushes, a sense of fulness in the head is experienced, the pulse at the wrist loses its tension, and the pain ceases. Dr Balfour has found in two cases that nitrite of amyl kept in a stoppered bottle gradually loses much of its efficacy in cutting short the paroxysms of angina, notwithstanding that it still flushes the face. Clinical experience does not appear to confirm Anstie's apprehension that this remedy might possibly induce cerebral hæmorrhage if the arteries of the brain should be diseased.

Since the year 1877 *nitro-glycerine* has been largely used in the treatment of angina pectoris; it was first tried by Dr Murrell. Although its physiological action is less rapid than that of nitrite of amyl it is yet commonly quick enough to cut short an attack if taken at the very com-

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mencement. But for this purpose it must be employed either in solution or in the form of Martindale's chocolate tablets. Pills, unless broken up by mastication, are too slow in their effects. The proportion most commonly used is an alcoholic solution of the strength of 1 per cent. It may be given in a drachm of water, when it is almost tasteless; or, if there be flatulence, in peppermint water with a little chloric ether. The dose necessary to give relief varies very widely in different patients; sometimes several doses have to be taken in succession at short intervals before the pain can be entirely got rid of. In beginning the treatment, it is generally best to prescribe at first half a minim or one minim of the solution; in one case Dr Murrell pushed the dose until 110 minims (more than a minim of pure nitro-glycerine) were taken at a time; but most patients are liable to experience alarming effects with fifteen or twenty minims. It gives rise to headache, a rushing noise in the ears, a sensation of fulness in the neck, and sometimes to nausea, languor, and drowsiness or even complete insensibility. These symptoms generally become more marked as the dose is increased; but with time a certain amount of tolerance of the remedy is often established.

The great use of nitro-glycerine, however, is in preventing the recurrence of seizures. In many cases if taken in one-minim doses from three or four times to every three or four hours in the day, the patient is freed from his attacks, and may be able after a time to leave off taking it, and remain apparently well, able to walk long distances and even uphill without discomfort.* The author has met with two cases in which persons thus freed from their symptoms ultimately died suddenly. Anyone who has suffered from the disease in a well-marked form should always afterwards lead the most quiet life that he possibly can, avoiding all kinds of exertion and excitement. One of the two patients just mentioned had returned to his business on the Stock Exchange. Rest must, indeed, be regarded as an essential part of the treatment of angina pectoris. Probably all the general measures found useful in the management of cases of thoracic aneurysm and severe cardiac disease find their application here also.

It is right to add that Dr Balfour says of nitro-glycerine that he has "used it without any benefit in the treatment of angina."

Before its introduction, arsenic was the medicine which proved most efficacious in warding off the seizures. In a good many cases it yielded very satisfactory results.

* As an instance of the successful administration of nitro-glycerine in what Dr Walshe would doubtless have regarded as "pseudo-angina," I may relate the following case, which occurred to me in 1882. A bank clerk, about twenty-one years of age, had for some weeks been suffering from what he described as a "sudden tightness of the chest," which would stop him in walking, so that he could not walk more than thirty or forty yards. He experienced a pain at the lower end and a little to the right of the sternum as well as behind near the spine at about the same level. He also noticed that he could not lie on the right side in bed without discomfort. His own impression was that the seat of the affection was in the lungs rather than in the heart. I found that he had a pulse of 120, but this was in part due to nervousness, for it soon afterwards fell to 104. No sign of any organic cardiac affection could be detected. I prescribed for him one minim of the solution of nitro-glycerine three times a day. In about a week he lost his complaint entirely, so that (as I afterwards learnt) he took only a single bottle of the medicine containing twelve doses.—C. H. F.

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AFFECTIONS OF THE MUSCULAR WALLS OF THE HEART

"ἐν δέ τέ οἱ κραδίη μεγάλα στέρνοισι πετάσσει."—*Iliad*, xiii, 282.

DILATATION AND HYPERTROPHY.—*History—Physiology of the process—its estimation—its anatomy—Diagnosis and signs—Ætiology—Symptoms—Treatment.*

FIBROID DISEASE.—*Chronic myocarditis—Anatomy—Origin and pathology—Cardiac aneurysm—Symptoms—Course and event.*

Acute myocarditis—Its origin, anatomy, and results.

FATTY DEGENERATION.—*Fatty overgrowth and infiltration—Intrafibrous fatty metamorphosis: its histology, causes, and symptoms.*

Granular or pigmentary, and calcareous degenerations—Rupture of the heart.

BEFORE the invention of the stethoscope the little that was known of organic disease of the heart referred almost entirely to its bulk. It was known to be wasted in certain cases, and more frequently to be enlarged, the cavities more capacious, and the walls much thicker. Valvular lesions were either not observed or not understood. More attention was paid to the size, form, and colour of the polypes or masses of clot which are found in the cardiac cavities after death. Even Laennec, in the fourth part of his great work which refers to the heart and vessels, puts hypertrophy and dilatation of the heart first. These subjects occupy six chapters, pp. 258—284; then follow five on atrophy, softening, induration, and fatty degeneration of the heart, pp. 285—301. "Valvular Disease, its Anatomy and its Physical Signs" occupies a single chapter of only 13 pages, while pericarditis takes 35 and aortic aneurysm 41 pages. At that time enlargement of the heart was regarded as a primary disease. But the progress of knowledge has limited idiopathic maladies in this as in other departments of medicine, and we now know that in nine cases out of ten hypertrophy or dilatation is secondary to changes in the kidneys, the lungs, or the valves of the heart itself.

Hypertrophy, generally attended with dilatation, of the heart is an almost inevitable result of all the more grave affections of the cardiac valves; another frequent cause of the same changes in the right chambers is emphysema and pulmonary obstruction generally; and a third, affecting chiefly the left ventricle, is chronic Bright's disease. In each instance it is clear that the organ enlarges because the work which it has constantly to do is increased.

DILATATION AND HYPERTROPHY.—The obvious enlargement of the heart which is often seen on examining a patient's body after death had been described by the pathologists of the eighteenth century, and was more accurately discriminated by Corvisart, who named dilatation without hypertrophy *anévrysme passif*, and the two combined *anévrysme actif*.

Laennec adopted his facts and his nomenclature. Hypertrophy with

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dilatation of the left ventricle, the most striking and frequent condition, was diagnosed by a strong pulse and apex-beat, diminished resonance on percussion over the cardiac area, and a red colour of the face; but Laennec admits that these symptoms may be all absent, and that *le pouls surtout est très trompeur*. He described the first sound (*la contraction du ventricle gauche*) as dull and prolonged, and what we now call the second sound (*la contraction de l'oreillette*) as very short, faint, and sometimes inaudible.

Dilatation of the auricles was also described, and Corvisart recorded a case of "partial dilatation of the heart," or as we now call it cardiac aneurysm. Induration and softening of the muscular substance of the heart, atrophy, and fatty degeneration were also recognised.

Theory of hypertrophy.—How increased activity produces hypertrophy is hard to say; the necessary condition for overgrowth is no doubt hyperæmia, and this probably begins as the result of increased waste of tissue from exertion. The passive tissues hypertrophy either in direct consequence of hyperæmia, as the clubbed fingers of chronic cardiac disease and of long-continued venous congestion generally; or from some unknown stimulus, as in the overgrowth of the bones seen in osteitis deformans, or of the spleen and lympharia in leuchæmia and Hodgkin's disease. Of the active organs, those of the nervous system, whether belonging to the central, the conducting, or the peripheral and receptive apparatus, appear to be incapable of true hypertrophy, however much exercised: the so-called cerebral hypertrophy (described vol. i, p. 691) is probably always a form of interstitial sclerosis. Glands hypertrophy from the stimulus of overuse, as the liver in beer-drinkers, the kidneys in diabetes, or one kidney when the other has been destroyed by some local disease. The voluntary muscles hypertrophy remarkably—by increase of the fibres both in number and in size—in response to exercise; but the effect of training in removing interstitial fat is often sufficient to mask the true muscular development and make the firm and powerful muscles of an athlete less bulky than those of an indolent free-liver.

Moreover, in this, as in other physiological developments, the degree of change is limited by the law of each individual organism. However well nourished and well worked, the muscles will not hypertrophy beyond a certain point for each man, just as the possible degree of speed or flesh-forming is limited for each horse or ox, and the possible attainments of the senses or the intellect are limited for each human being.

Involuntary muscular fibre undergoes hypertrophy from overuse at least as readily as that which is set in action by the will: the ciliary muscle grows larger as the lens becomes less elastic, the bladder hypertrophies behind a stricture, the intestine above a chronic obstruction. In like manner the ventricle or the auricle hypertrophies as the result of narrowing of its orifice of egress, by which the discharge of its contents is obstructed. In the left auricle and left ventricle this is frequently seen; on the right side of the heart, stenosis of the pulmonary or tricuspid orifice is rare; but when present, the same effects are produced, except that for some unknown reason hypertrophy of the right ventricle produces more hardening and closeness of texture, with less increase of bulk, than that of the left.

The obstruction need not be from narrowing of the ostia through which each cavity discharges its contents; it may depend on mechanical hindrance to the flow of blood further on. Mere dilatation of the arch of the aorta can have no effect of this kind, but this dilatation is always associated with loss of elasticity; and, directly or indirectly, rigid arteries lead to hyper-

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trophy of the left ventricle, probably by increased tension during systole. Increased friction from roughness of the tunica arteriosa intima, contraction of the arterioles, obstruction in the capillaries from external pressure, retardation of the flow in the veins from deficient muscular exercise or deficient respiratory movements—all these conditions increase intraventricular blood-pressure and thus stimulate the muscular walls to hypertrophy. In the same way obstruction in the pulmonary capillaries produces hypertrophy of the right ventricle.

Increased stimulus, however, does not always call forth adequate hypertrophy. Impaired nutrition of the muscular fibres, from want of food, from imperfect digestion and absorption, or from loss of blood, may prevent them responding as they should; just as labour and gymnastic exercises, if undertaken by the ill-fed, the anæmic, or the invalid, or if disproportioned to the powers of childhood or old age, will not cause hypertrophy of voluntary muscles, but rather atrophy and increased weakness.

Moreover, increased fluid-pressure will always tend to dilate the walls of the containing cavity, even when its elastic as well as its contractile force is increased by hypertrophy of its muscular coats. Accordingly, some degree of dilatation is rarely absent, even when compensatory hypertrophy is well developed. This is seen in the dilated stomach which follows pyloric stenosis, the dilated œsophagus which forms a pouch above a stricture, the dilated intestine above a chronic obstruction, and the dilated renal pelvis, ureter, and bladder, which result from stricture of the urethra.

In the heart, dilatation may occur with but little hypertrophy, particularly in the auricles, from mitral or tricuspid regurgitation; and hypertrophy may occur with little or no dilatation, as in uncomplicated cases of chronic Bright's disease; but as a rule the two conditions are met with together, both at the bedside and in the deadhouse. Nevertheless we can, in the majority of cases, recognise as the leading process (better perhaps by studying the physiology of the circulation during life than the cardiac anatomy after death) either the compensatory and favourable one of hypertrophy, or the ingravescent and unfavourable one of dilatation.

We shall see hereafter that the most difficult cases of cardiac hypertrophy to explain are those of chronic Bright's disease, but there is little doubt that the immediate cause of the change in the ventricle is increased blood-pressure from obstruction in the systemic circulation.

There is one form of obstruction which is less obvious because it is scarcely pathological. It is that caused by over-exertion in what often appears to be healthful labour or athletic sports. Each contraction of a muscle, if extreme, and particularly if long sustained, causes pressure on the arterial trunks which pass through or beside it.* This leads to increased blood-pressure in the aorta and left ventricle, and becomes a stimulus to cardiac hypertrophy and a cause of dilatation of the heart and aorta, as well as of irritative arteritis of the latter.

The healthy heart naturally possesses a considerable amount of reserve force beyond that which is required to carry on the circulation in ordinary circumstances. Consequently, although the strain upon it is augmented by muscular efforts of all kinds, it is generally able to meet the call without suffering damage. Even if its walls should to some extent yield to the increased pressure which they have to bear, they generally recover themselves

* The smaller arterioles which supply the muscle itself are probably protected by their physiological dilatation at the moment of stimulus of the motor nerve.

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afterwards, when the body returns to a state of rest, and the blood-current resumes its usual tranquil course. But such is not always the case. It sometimes happens that violent or frequently repeated bodily exertion leads to permanent enlargement of the heart. We must suppose that one factor concerned in bringing about this result is a deficiency of power in the muscular fibres. Either they are originally weak, or their strength may have been lowered by previous disease or by an insufficient supply of food.

Again, there are probably cases in which a like enfeeblement of its walls renders the organ unable to maintain even the natural tranquil circulation. This feebleness is not due to fatty degeneration, or other obvious organic changes in the fibres, but to a state of the heart like that which in the voluntary muscles is recognised as "weakness," leading to fatigue on moderate or even slight exertion. It seems likely that the immediate effect of the failure of the heart to perform its work efficiently is a yielding or *dilatation* of its walls, and that *hypertrophy*, or the increased growth of their substance, sets in afterwards. But, as a rule, it is not possible to trace this clinically.

Definitions.—As regards the application of terms, which has sometimes been somewhat confusing, it seems reasonable that "simple hypertrophy" should mean an increase in the amount of the heart's muscle, its cavities remaining unaltered in capacity; "simple dilatation," an increase in their capacity, the amount of muscle remaining stationary. The former is frequently observed, especially as a result of Bright's disease. And although the latter is perhaps never actually seen in the deadhouse, it must yet be supposed to occur at the beginning of many cases of what afterwards appears as dilatation with some hypertrophy. Practically in almost all cases consecutive to valvular lesions, and in many consecutive to chronic renal degeneration, the condition is one of dilatation with hypertrophy, or hypertrophy with dilatation.

A dilated heart is said to be hypertrophied if its weight as a whole is above the normal standard, even though every part of its walls may be below the natural thickness; and it was probably in order to avoid this apparent inconsistency that Dr Walshe has defined as "simple dilatation" a condition in which "the walls are of such thickness as would be normal had the capacity been unchanged." But such a conception really rests upon an arbitrary basis, because, for a dilated heart, the normal thickness of walls should be proportionately augmented, in order to maintain its functional power; below this it is only a question of varying degrees of failure of "compensation." In fact although simple hypertrophy is in its physiological effects the very opposite of simple dilatation, yet in all the mixed forms of these affections it is to the latter rather than to the former that the case approximates clinically, however much increased the bulk may be.

Measurements.—In the *post-mortem* room, then, in order to ascertain whether or not a heart is hypertrophied, one has only to weigh it after it has been emptied of its contents, and separated from the great vessels and pericardium. According to Dr Peacock ('Reynolds' System,' vol. iv), the ordinary range of the weight of the organ is, in men who have died with acute non-cardiac diseases, from nine to eleven ounces, with chronic non-cardiac diseases from eight to ten ounces; in women affected with acute disease from eight to ten ounces, with chronic disease from seven to nine ounces. But in large and powerful men, who have been killed by accident or have died after a

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short illness, the weight may sometimes be as much as twelve ounces, or even more, without the slightest evidence of disease.

It is much less easy after death to determine the presence of dilatation, at least in its slighter degrees; when well marked there can be no doubt about it. Peacock gives the following figures as representing the normal dimensions of the two principal chambers:

	Males.			Females.		
	Lines.	Inches.	Millimetres.	Lines.	Inches.	Millimetres.
Circumference of heart	103·7	9·209	233·32	104	9·236	234
Girth of right ventricle	55·4	4·919	123·85	58·4	5·184	131·4
„ left „	48·3	4·289	108·67	45·6	4·049	102·6
Length of cavity of right ventricle .	43·3	3·821	96·42	44·3	3·925	99·67
„ „ left „ . .	37·6	3·333	84·6	37·1	3·197	83·47

But it is very difficult to make sure how far the apparent size of the organ is modified by the state of contraction or of relaxation of the muscular fibres. The difficulty is greatest in cases of what was once described as "concentric hypertrophy," a condition in which it is supposed that the wall of the ventricle grows in thickness inwards, so that its cavity actually becomes smaller than natural. But although some recent German writers are still disposed to admit that concentric hypertrophy occurs in some rare cases, it is probably never a true pathological condition. In the *post-mortem* room hearts may at first look as though they might be specimens of such a kind, but they always yield to a little stretching with the fingers, and regain their normal size. Even when a hypertrophied heart seems to have moderately large chambers it still remains a question whether, in a fully relaxed state of their walls, they might not be larger; in other words, the pathological proof of even excentric hypertrophy without dilatation is less simple than might be supposed.*

It is only when the capacity of the cardiac chambers is not above the normal that one is justified in taking the thickness of their walls as proof of the presence or absence of hypertrophy. Peacock gave the following as the normal measurements (1854). The figures in the third columns are those given by Bizot (1838).

Thickness of the	Males.			Females.		
	Lines. P.	Millimetres. P.	Mm. B.	Lines. P.	Millimetres. P.	Mm. B.
Walls of the right ventricle: base . .	1·85	4·16	4	1·85	4·16	3
„ „ „ midpoint	1·98	4·35	3	2	4·5	2
„ „ „ apex . .	1·42	3·19	2	1·3	2·92	2
„ left „ base . .	5·15	11·58	10	4·9	11·02	9
„ „ „ midpoint	6	13·15	11	5·6	12·6	10
„ „ „ apex . .	2·4	5·4	8	2·5	5·62	7
Septum between the ventricles . . .	5·73	12·89	11	4·7	10·57	9

* It is said that the description of concentric hypertrophy given by Corvisart and followed by other French pathologists, including Laennec, was influenced by the number of hearts examined, during the reign of terror, of persons executed by the guillotine; for after rapid death from hæmorrhage in healthy subjects the left ventricle contracts closely and looks smaller and much thicker than in ordinary cases.

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Even when dilatation is present, the determination of the thickness of the several chambers is still of great importance, as enabling one to form some estimate of the degree to which the heart was competent to carry on its functions. In different cases there are very wide differences. One left ventricle may measure five or six inches in length, and its walls be only two or three lines thick; in another not more capacious they may be more than an inch thick.

As a rule, the weight of a heart, morbidly dilated and hypertrophied independently of valvular lesions and of Bright's disease, does not exceed fifteen or twenty ounces. But in one case observed at Guy's Hospital it reached thirty-three ounces. Peacock speaks of hearts hypertrophied without any material valvular lesion or obvious source of obstruction in the aorta, and reaching the weight of from twenty-six to forty ounces. But it is not quite clear whether the presence of chronic renal disease was also excluded in these cases.

In one of them, to which he refers as having been exhibited by Dr Bristowe at the Pathological Society in 1853, it is possible that the state of the kidneys was really the cause of the cardiac affection, although it is right to add that this view was rejected after due consideration by Dr Bristowe himself.

In the cases which are now mainly under consideration, we have no means of determining the rapidity with which the heart undergoes hypertrophy; probably the process is very slow and gradual. But when it arises secondarily to other lesions, of which the starting-point can be fixed, it has sometimes appeared to be much more rapid than could have been anticipated. Dr Stone ('Lancet,' 1879) has related two examples of injury to the aortic valves by blows upon the chest, in each of which, if the heart was healthy at the time of the accident, it must have gained weight at the rate of nearly an ounce a week during the four or five months that elapsed before the patient's death. And Dr Goodhart ('Path. Trans.,' vol. xxx) has published a case, the history of which would suggest that the organ within three or four weeks grew to a weight of nineteen ounces, as the result of an attack of pericarditis. Dr Stone remarks that the increase in size of the pregnant uterus is less rapid, namely, two thirds of an ounce a week; but this assumes that the growth of the organ is uniform during the whole period of gestation, which is not likely to be the case.

There has been much discussion as to whether cardiac hypertrophy depends upon an overgrowth of existing fibres or upon a formation of new ones. Schroetter (in 'von Ziemssen's Handbuch') states that the fibres measure 0·03 mm. in a hypertrophied heart, as compared with a normal thickness of 0·007 mm. Friedreich also is said to have arrived at 0·025 mm. as the mean of ten measurements of the fibres of a hypertrophied left ventricle. But Rindfleisch says that he failed to discover any such difference, and his conclusion is that the fibres, being unlike those of all other muscles in forming a network, undergo a further splitting up, which leaves them apparently of the same size as before.

As already remarked, different cases seem to differ as to the order in which dilatation and hypertrophy are developed. In most instances it is probable that dilatation occurs first as the result of over-distension or of weakness of the cardiac muscle, and that hypertrophy follows later. In others hypertrophy appears to be the primary condition. In either event, that which finally brings about a failure of the heart's functional activity is

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commonly progressive dilatation, which causes the hypertrophy to be relatively inadequate. It is usually said that such an ultimate breakdown of the organ depends upon the occurrence of fatty changes in its muscular tissue. But Cohnheim ('Vorlesungen,' i, p. 72) throws doubt upon this view. There has, he says, been much exaggeration in the statements that have been made as to the frequency of fatty degeneration in hypertrophied hearts; and even when it is present, he thinks it just as likely to be the effect of the disturbed compensation as its cause. His own view is that there is a mere "fatigue" or "exhaustion" of the fibres, unattended with any anatomical change that can be recognised with the microscope. Dr Allbutt ('St Geo. Hosp. Rep.,' vol. v) has drawn attention to a very interesting fact, which perhaps has a bearing on this question. It is that in the file-cutters of Sheffield, who are constantly using the arm in rapid flexions, the biceps muscle undergoes great enlargement, but that after a few years it again wastes, and now falls far below its normal size. If the functional activity of hypertrophied muscles has thus a more or less definite period of duration, it is obviously important to relieve them, as far as may be, of all strains and extra calls upon their strength, so that if they are to break down the time may be postponed to the furthest possible limit.

Diagnosis.—The clinical recognition of hypertrophy and of dilatation of the heart rests partly upon percussion, partly upon inspection and palpation, partly upon auscultation.

By *percussion* one determines to what extent the heart comes into contact with the anterior wall of the chest, uncovered by the lungs. Consequently, the results of percussion are modified not only by the state of the heart, but also by that of the lungs. If the lungs are emphysematous, the area of dulness due to the heart may be diminished, notwithstanding that the organ is enlarged; if the lower and anterior part of the chest on the left side is flattened, or if the corresponding part of the lung is collapsed, the dull area may be increased, though the heart is no larger than natural. There is even a difference in the extent of cardiac dulness, according as the breath is drawn deeply in or forced out, so that for accurate percussion it is necessary that the breath should be held. A good way of arriving at a satisfactory result is to make a series of marks with an aniline pencil upon the patient's chest indicating the different points at which dulness begins to pass into resonance round the circumference of the organ. One thus obtains a more or less triangular figure, representing the region within which an absolutely dull note is obtained. Above, it forms an angle, which in normal circumstances is situated at the upper border of the fourth left costal cartilage close to the sternum. From this point two lines are traced downwards, diverging in their course. One of them corresponds with the left border of the sternum, for although the right ventricle uncovered by lung lies behind the lower part of this line as far as the median line, the tone yielded by the bony tissue when percussed masks the dulness that should theoretically be present, and prevents any accurate definition of the space occupied by the heart on this side.* The other line extends downwards and outwards, passing to the inner side of the nipple until it reaches a point at which the apex of the heart can be felt beating. Along this line

* Guttmann says that the bone may be rendered less vibratile if the hand be laid over the upper part of it, or if an assistant press firmly with the hands placed upon the rib cartilages on each side of it; in these circumstances a dull sound may be obtained.

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the transition from dulness to resonance takes place gradually, so that above it one can trace another line running more or less parallel and about half an inch distant, which indicates the upper limit of a region of partial dulness, just as the lower one indicates the upper limit of absolute dulness. The upper line begins above at about the level of the third rib. To complete the triangular figure, a base line has to be drawn from the lower end of the sternum to the point at which the heart's apex beats. It can scarcely be traced by percussion, because the cardiac dulness passes insensibly into that caused by the left lobe of the liver.*

It must be added that, when the stomach is distended with gas, the resulting tympanitic sound is not infrequently transmitted by lateral conduction beyond the region which the stomach actually occupies. In fact, such a sound may often be elicited by tolerably firm percussion over the very spot beneath which the apex-beat can be felt. But in percussing over the heart, it is essential that the stroke should be light and free, and made from the wrist. The statements above made with regard to the natural extent of the cardiac dulness are applicable only when the patient is standing upright or lying on his back. When he lies over to the left, the left side of the triangular area shifts further to the left, even though the heart may be of the natural size.

When the heart is enlarged, the upper angle of the area of cardiac dulness commonly remains at the fourth rib. If it reaches as high as the second, there is probably some morbid condition beyond hypertrophy and dilatation—perhaps aneurysm, or pericardial effusion, or mediastinal tumour. On the other hand, the position of the two sides of the triangle varies widely from the normal. The right one may reach to the right border of the sternum, or half an inch or even an inch further to the right; this is an indication of increase in size of the right ventricle. The left line may run outwards as far as the nipple or even still further to the left, and it is carried much lower than usual, the apex-beat being situated at a lower level in the sixth interspace or even in the seventh; this is a sign that the left ventricle is enlarged.

If the right ventricle alone is dilated, the apex-beat is displaced outwards without being lowered, and pulsation may also be felt in the epigastrium. Walshe states that distension of the right auricle contributes largely towards the increase of dulness to the right of the sternum at the upper part of the cardiac region. He says, too, that distension of the left auricle may cause abnormal deficiency of resonance in the third and second left intercostal spaces. And he even speaks of having recognised an impulse of presystolic rhythm in these several positions as the result of enlargement of one or the other of the auricular chambers. In consequence of the displacement of the two lines marking the sides of the dull cardiac area, this acquires, when the heart is enlarged, a more or less definitely quadrilateral instead of the normal triangular form.

For practical purposes nearly all that can be learned from percussion of the heart, so far as concerns the diagnosis of enlargement of the left ventricle, may be gained by tracing one diagonal line from the inner end of the fourth left costal cartilage downwards and inwards to the apex-beat, and another line crossing it at right angles from the base of the ensiform process upwards and outwards to the point at which the absolute cardiac dulness

* Dr Gee says that sometimes a distinct heightening of pitch and increase of resistance can be made out in passing from cardiac to the hepatic dulness.

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ceases. Normally the first diagonal line should not exceed two and a half inches in length; when the heart is much enlarged it may extend to four or five inches. The second diagonal line should measure about one and a half inches; it may be increased by disease to more than two inches.

It must not be supposed that percussion affords an infallible indication of enlargement of the heart, even when the lungs are neither emphysematous nor retracted. On the contrary, great hypertrophy of the left ventricle may be present (as in cases of Bright's disease) without there being any increase in the area of dulness. The heart seems to bury itself in the hollow of the left lung, so that it is not more widely in contact with the chest wall than in normal circumstances. This is well shown in cases of cerebral hæmorrhage, where one often finds at the autopsy an enormously hypertrophied heart, although the day before it was impossible to detect any clinical evidence of it.

If percussion fails to reveal the presence of enlargement of the heart, it is seldom that we learn it from *inspection* or from *palpation*. Displacement of the apex-beat we have already spoken of, and it is appreciable by the eye as well as the hand. The deliberate heavy impulse of hypertrophy and the short slapping impulse of dilatation are both often very characteristic; but great caution is required in diagnosing hypertrophy from palpation alone, at least while the action of the organ is disturbed by excitement.

When, however, it has been determined that the heart is enlarged, the question arises how far the enlargement is due to dilatation or to hypertrophy; and the solution of this question, so far as it is soluble, is found in careful examination by the eye and by the hand and finger. In either case the heart's impulse is seen and felt over a more extended area than that of the natural "apex-beat." When there is great hypertrophy the impulse is often diffused, laboured, and heaving, so that even a stethoscope, with the observer's head resting upon it, is lifted as though by an irresistible power. In some cases, while the fifth and sixth ribs are pushed forwards, other parts of the chest-wall are sucked in. In cases of nearly pure dilatation the impulse becomes (to use the words of Dr Walshe) "either a short feeble slap, followed by a sudden fall back of the organ, or a more prolonged faint tremulous motion." In the latter case it often has an undulatory character to the eye. Another peculiarity of the heart's action which indicates dilatation rather than hypertrophy is irregularity in the force of successive beats, or in their rhythm.

It is doubtful whether enlargement of the heart ever causes increased prominence or bulging of the præcordial region and widening of the intercostal spaces, as compared with the corresponding parts on the opposite side of the chest. Most authors admit that this is sometimes the case, especially in young subjects; but Schroetter, following Skoda, maintains that it occurs only when there is also pericarditis, by which the textures forming the chest wall are softened.

Little direct information as to the existence of hypertrophy or of dilatation is yielded by *auscultation*. But it is to be particularly noted that in cases of simple hypertrophy, with little or no dilatation, the first sound is not louder than usual, but fainter.* It is often dull and muffled.

* So Laennec, and so Walshe, who describes it as "prolonged and weakened, sometimes almost to actual extinction, the sensation reaching the observer's ear being rather one of impulse than of sound." But every beginner is surprised that a large muscle does not produce a loud sound.

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In cases of simple dilatation the first sound is often loud, though if the muscle is soft and flabby it may be weak; but it is short, and like a second sound. When dilatation and hypertrophy occur together, the first sound may be loud, higher pitched than natural, and widely audible over the surface of the chest. Any alterations in the second sound depend upon the state of the arterial tension, and afford only indirect indications of the condition of the heart itself.

The question whether a systolic murmur is ever due to dilatation of the ventricles, apart from organic lesion of the mitral or tricuspid curtain, is a difficult one; but one or both of the cuspid valves may be rendered incompetent as a result of the widening of orifices which necessarily takes place as the heart increases in size.*

Seitz ('Deutsch. Arch.,' xi, xii) has drawn special attention to the fact that the movements of an enlarged heart within the pericardial sac are sometimes attended with sounds having a grazing or scraping quality, so as to be very like those produced by pericarditis. In one case observed by him these continued up to the time of death, and no doubt was entertained as to the existence of pericardial inflammation; yet at the autopsy the serous membrane was found perfectly healthy, without any thickening or opacity of its surface. Dr Walshe, too, speaks of "knocking and rubbing additions to the first sound at the apex" as not very uncommon. One such instance occurred several years ago at Guy's Hospital, which led to a diagnosis of pericarditis that proved to be absent on *post-mortem* examination.

Ætiology.—We have now to consider what are the special causes of dilatation and hypertrophy of the heart.

In the first place, as stated at the beginning of this chapter, two of the most important causes are chronic Bright's disease and lesions of the cardiac valves. In many cases it is impossible at the bedside to determine whether the state of the kidneys accounts for the cardiac symptoms, or whether there is a primary valvular lesion—probably mitral—to which they may be due. Even in the deadhouse the very same points often remain doubtful, or the facts are so far ambiguous that different pathologists interpret them differently.†

* Formerly, in cases of heart disease with dropsy, the diagnosis of "mitral regurgitation" was deemed sufficiently accurate, and physicians frankly admitted that they were often unable to tell whether the left auriculo-ventricular orifice would after death be found narrowed or widely dilated. But now everyone recognises that in those instances in which it is dilated there is no lesion of the valve itself, but that the primary morbid process is dilatation of the left ventricle. Still it may be held—and until recently this was the common opinion—that regurgitation through the widened opening occurs secondarily, and constitutes the essential feature of the disease, being directly concerned in bringing about the induration of the lungs, the nutmeg change in the liver, the dropsy, and all the other obvious symptoms. Of late, however, the tendency has been to disregard to a great extent the presence or absence of mitral incompetency, and to look upon the impairment of the propulsive power of the ventricle as the really important thing in the cases in question. Dr Fagge long taught this; and he even entertained doubts whether secondary mitral regurgitation really occurs so constantly as is supposed by those who regard a systolic apex-murmur as a sufficient proof.

† As regards the kidneys, these organs may be of good size and not unhealthy in look, and yet may be shown by the microscope to have undergone changes so extensive as fully to account for any enlargement of the heart that may be present. The renal origin of such cases is by no means always, or even generally, indicated during life by albuminuria, a low density of urine, or the presence of casts, even when the autopsy leaves no doubt about the matter. And there are a great many other cases in which, after the most complete histological examination of the kidneys, different pathologists would express divergent opinions as to whether the cardiac affection should be looked upon as secondary to the renal, or the renal to the cardiac.—C. H. F.

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Another possible cause of enlargement of the heart has been found in extensive pleural adhesions. But it is allowable to doubt whether the cases supposed to be of this kind which have been recorded by Bäumler and by Brüdi ('Deutsches Arch.,' xix) really warrant the conclusions drawn. It must be remembered that the right side of the heart is not only subject to dilatation and hypertrophy from obstruction in the pulmonary circulation analogous to that in the systemic, but also from mere abolition of large capillary tracts, as in carnification of the lung by pleural effusion.

Primary dilatation and hypertrophy.—The direct causes of primary enlargement of the heart, independent of preceding valvular lesions or disease of the kidneys or lungs, are as follows.

(1) One cause of what may properly be termed relative hypertrophy of the heart is derangement of the natural growth of the organ about puberty. The heart, like the brain, does not develop at a uniform annual rate. It grows rapidly up to the sixth or seventh year, and again between fifteen and twenty. *La mégalocardie de croissance* has been described by Sée and other French physicians. It is most common in children under puberty who are put to work above their strength. There is often a quick pulse and palpitation, sometimes dyspnœa, and frequently headache. Beside the signs of hypertrophy there is not infrequently a systolic apical bruit. Fresh air, rest, and digitalis if there is tachycardia, are the remedies recommended.

(2) A clear and unquestioned cause of hypertrophy in adults is *over-exertion* of the heart. Da Costa, in his observations on 'Irritable Heart' among soldiers in the American war (p. 5), states that in twenty-eight out of a hundred cases there was evidence of hypertrophy. In one of them death occurred from strangulated hernia eleven months after the commencement of cardiac symptoms; the left ventricle, though not apparently larger than natural, had its walls seven eighths of an inch thick at the thickest part. Fräntzel ('Virchow's Archiv,' 1873) recorded a like affection in nineteen soldiers engaged in the Franco-German war, especially among those who took part in the arduous march to Orleans, or in the attack upon Belfort. He also refers to some statements according to which recruits in the German army frequently become affected with hypertrophy of the heart as the result of prolonged and heavy marches, especially in summer. Fräntzel is disposed to doubt the correctness of these observations, but our own Army Medical Department has been for many years familiar with the prevalence of the disease among British soldiers even in time of peace. In 1870 Dr Myers published an essay in which he showed that cardiac affections in general were considerably more common in soldiers than in sailors, and in the Foot Guards (who are chiefly stationed in London) than in the men of the Metropolitan Police. In a large proportion of cases he found neither valvular disease nor disease of the aorta, but an extreme excitability of the heart, leading after a time to its enlargement. The general opinion formerly was that the cause lay in the crossbelts, heavy accoutrements, and tight clothing which the men used to wear, and by the urgent advice of Dr Maclean and Dr Parkes the old form of knapsack was abolished, and a "valise equipment" was adopted in its stead. Dr Myers laid especial stress upon the effect of the tightness of the tunic collar in constricting the neck. It would be interesting to know whether the prevalence of cardiac dilatation and hypertrophy among soldiers has diminished since, but according to Prof. Veale ('Army Med. Dep.,' Report xxii) the necessary statistical data

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are wanting. He states, however, that this affection and the "palpitation" which is its most conspicuous symptom are still very frequent, and after careful inquiry into all the circumstances of a hundred cases he assigns it in different instances to seventeen more or less distinct causes. But it seems far more likely that some one cause is really responsible, and probably the true solution of the difficulty has been found by Surgeon F. A. Davy, who refers it mainly to the "setting-up drill," during which recruits are compelled to "swell the chest" so as artificially to expand it ('Army Med. Dep.,' Report xviii). To this they are subjected for four hours a day during a period of about six months, having to march, and even to "double," with the chest in an abnormal condition. Dr Davy shows that, in consequence of free expiration being prevented, the functions of the lungs and of the heart must be very seriously interfered with; and he appears to have ascertained by direct observation that soldiers under the drill, even when they are standing, have the frequency of the respirations increased to about 40, and the pulse to 110 in the minute, that the heart's rhythm is often disturbed, and that the impulse of the organ is altered in position, is more forcible, and is felt over a wider area than natural. It is surprising that Dr Veale does not allude to this view of the matter; but Dr Myers has often seen recruits completely exhausted after their morning's drill, which (one would suppose) ought not to have any such effect upon healthy young men. The notion is, of course, that the soldier's figure is improved by the capacity of the chest being enlarged, but, as Dr Davy remarks, this is of no advantage when obtained at the expense of its mobility.

Among civilians the ill effects of over-exertion of the heart, though they had been cursorily alluded to by many previous writers, seem to have been first fully recognised by Dr Peacock in reporting, in 1864, upon the health of the miners of Cornwall, who, besides heavy hammer-work in the day, have to climb ladders of immense height in order to get out of the pit every evening. He found that many of them suffered from cardiac dilatation. The cases described by Dr Allbutt in the 'St George's Hospital Reports,' vol. v, p. 23, hardly fall into the present category, inasmuch as he assigned a conspicuous place in the sequence of events to chronic changes in the aorta and its valves, leading at length to regurgitation. More strictly in point is a series of articles by Seitz in the 'Deutsches Archiv' for 1873 and 1874. He showed that at Zürich cases are tolerably frequent in which during life there is great uncertainty as to the exact diagnosis of the cardiac affection from which the patients undoubtedly suffer, and which after death are best explained on the view that the disease is a primary enlargement of the organ. The patients were almost all men, and engaged in heavy labour of one kind or another. But the most striking paper of all is perhaps one by Münzinger, in the 'Deutsches Arch.' for 1877, on what he terms "the Tübingen heart." It appears that at Tübingen, heart disease, without any valvular lesion, is very commonly seen both in men and in women who work as labourers in the neighbouring vineyards, situated often upon the slopes of hills, up which heavy burdens of manure have to be carried. A point on which great stress is laid is that these poor people are very ill-fed, living on potatoes and puddings, and scarcely ever tasting meat. Dr Allbutt, too, alludes to insufficiency of food as an important factor in the ætiology of cardiac affections due to overwork and strain; he cites two cases of Dr Paget's, which appeared due to the habit

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of taking long and active exercise while fasting; and he expresses the opinion that one reason why young men of the upper and middle classes do not more often suffer ill effects from athletic sports is that they habitually live well.

One must take into account not only the absolute amount of exertion which a person may have undergone, but also the probable condition of the cardiac muscle at the time. Muscular work of all kinds of course calls upon the organ for increased efforts to maintain the needful circulation. In strong, vigorous subjects there is a reserve force which is equal to all but the most excessive demands upon it. But in weakly, ill-nourished persons the heart may fail under comparatively slight efforts. And there probably may be great differences in the vital endowments of the cardiac muscle itself in different individuals, altogether apart from the state of nutrition of the body generally.

These remarks are applicable likewise to hypertrophy and dilatation occurring as the result of valvular lesions or of Bright's disease.

It is not to be supposed, however, that any change in the muscular fibres recognisable by the microscope is an essential feature of cases of enlarged heart from overwork. Some few fibres may be found fatty or granular, but such an appearance is altogether exceptional.

(3) Among patients belonging to the middle classes, Traube ('Berl. klin. Woch.,' 1872, p. 223) was inclined to refer some cases of cardiac hypertrophy to "excessive *smoking* and to congestion of the portal system, resulting from sedentary habits and excess of food." The probably injurious effect of tobacco has been taken into account by all the writers who have discussed the ætiology of cardiac affections in the British army, but there seems to be no clear proof of its action in producing direct hypertrophy. As for indulgence in eating, it probably falls under the category of those conditions which produce hypertrophy by increasing the arterial tension.

(4) Since the recognition of the fact that *anæmia* induces a fatty change in the muscular fibres of the heart, it has naturally occurred to pathologists that those forms of the affection from which recovery takes place possibly form the starting-points of subsequent cardiac disease. The question was discussed by Dr Goodhart ('Lancet,' i, 1880); but although he has shown that in women who are actually suffering from chlorosis the heart's impulse is diffused and displaced outwards, and that when anæmia proves fatal this organ is found to be dilated, we are not aware of clinical proofs of the development of permanent enlargement from this cause.

(5) Much the same may be said of the supposed production of enlargement of the heart by the exanthemata and other *febrile diseases.* Pyrexia is known to damage the muscular tissues, and Dr Goodhart has recorded ('Guy's Hosp. Rep.,' xxiv) four or five instances in which sudden or nearly sudden death has occurred during scarlatinal dropsy, and in which the heart has been found dilated or fatty. But in the cases in question it is difficult to say how much was due to increased arterial tension resulting from nephritis, and how much to the antecedent pyrexial state. And even if it be true that during enteric and other fevers the heart may become for the time dilated, it has yet to be shown that the organ is liable to remain in a morbid condition after convalescence.*

* Professor Veale, in his paper above referred to, on the causes of palpitation and cardiac disease in soldiers, says that the most common of them all is fever, chiefly malarial. But when he declares, in support of this view, that the physician must have had "either

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(6) Another occasional cause of primary enlargement of the heart appears to be acute *rheumatism*. At least, now and then cases have occurred in Guy's Hospital which seemed to admit of no other interpretation. The most striking of these was that of a girl aged eleven, who died of cardiac dropsy six months after a rheumatic attack. The most conspicuous lesion was dilatation of the left ventricle, which had reached such an extent that although its walls measured only from one eighth to a quarter of an inch in thickness, the organ weighed ten ounces. There had probably been regurgitation through the mitral orifice, for the papillary muscles were much wasted, but the valve itself was healthy or only slightly thickened. How such an effect is produced by acute rheumatism, apart from pericarditis, is uncertain. That dilatation of heart may be caused by extension of pericardial inflammation to the subjacent muscle is certain.

Symptoms.—The symptoms of primary enlargement of the heart vary widely in different cases. In the earliest stage of the affection they consist partly in *palpitation* and subjective sensations of pain or discomfort in the cardiac region, partly in an increased *frequency of pulse*, which is often irregular or intermittent in rhythm. These phenomena have all been fully discussed in the first chapter of the present volume. But another symptom, of which the absence is conspicuous in the merely functional diseases of the heart, is *dyspnœa*. This at first comes on during exertion only; the patient finds that he cannot walk so quickly as before with comfort, and that going uphill or ascending two or three flights of stairs makes him feel short of breath. From this condition there are all gradations up to a point at which even the slightest bodily movement becomes almost impossible. Inasmuch as in health muscular exertion makes the beats of the heart more frequent and calls for greater vigour of systole, there is no difficulty in understanding how it disturbs the action of the organ when diseased. And in cases of enlargement of the heart it has been specially noticed (as, for example, by Dr Veale among the soldiers whose cases he studied) that after even slight exertion or excitement the rate of the pulse becomes disproportionately accelerated. But what is not so obvious is why this disturbance should give rise in the patient to a sensation of dyspnœa, even when the left ventricle is the seat of hypertrophy. The explanation seems to be that in spite of its augmented frequency the heart fails to send the blood through to the aorta with the needful rapidity; there is therefore an accumulation in the pulmonary veins and capillaries, and the right ventricle has to make increased efforts to propel its contents onwards. The obstructed pulmonary circulation prevents free access of the blood to the air, and hence the feeling of dyspnœa.

An early effect of cardiac dyspnœa is that the patient is unable to sleep with the head low. Instead of one pillow he has to use two or three. In extreme cases he cannot lie down at all, and is obliged to sit up in bed or to lean forwards. This condition is termed *orthopnœa* (vol. i, p. 945). The upright position facilitates the descent of the diaphragm, which in the recumbent posture is hampered in its movements by the pressure of the abdominal viscera, and especially of the liver.

The assumption in a previous paragraph, that in cases of primary

small experience, or very limited powers of observation, who cannot call to mind many instances of permanent weakening of the heart after fever," he appeals to a court of which the verdict would scarcely be in his favour.—C. H. F.

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enlargement of the heart the left ventricle is the chamber earliest affected, is not in accord with the view of some recent writers, who are inclined to think that the right ventricle often undergoes dilatation while the left still remains in a normal state.

We are familiar with the fact that dilatation of the left ventricle leads to a secondary dilatation of the right, owing to the pulmonary obstruction produced; but it does not seem possible that the order of events can be reversed. All pathologists, however, are agreed that if the frequent cases of enlargement of the right side of the heart from pulmonary emphysema or severe bronchial affection be excluded, dilatation of the right side is scarcely ever seen unaccompanied by a like affection of the left. The following is a very rare exception. A man, aged forty-one, was admitted into Guy's Hospital in 1880 on account of dropsy of the abdomen and legs. On examination there was a loud systolic murmur at the ensiform cartilage, musical in quality at that spot, and propagated towards the right nipple as much as towards the left. On account of the rarity of primary disease of the right chambers this diagnosis was given doubtfully, but the autopsy left no question of the fact. The heart, which weighed 16½ oz., was extremely broad and rounded in shape; the right ventricle was large and massive, with large fleshy columns, but the left was small and flaccid; the right auricle formed the greater part of the base of the organ; the tricuspid orifice admitted more than five fingers, and its edge was thick and opaque.*

Writers upon diseases of the heart have drawn a contrast, which does not appear to be well founded, between the effects of *dilatation* and those of *hypertrophy* of the *left* side of the heart. Walshe, for instance, speaks of the pulse, in cases of pure hypertrophy, as full, tense, and resisting; he describes persons so affected as having a florid countenance and bright full eyes, and as liable to sensations of rushing of blood to the head, and to dull aching or throbbing cephalalgia. But, however true these statements may be of cases in which the cardiac hypertrophy goes with augmented arterial tension, as in Bright's disease, they do not appear to apply to those in which the hypertrophy of the left side of the heart is a primary effect of the causes described in the present chapter. The observations of Da Costa, Myers, and Veale, all point to the conclusion that whether hypertrophy or dilatation be the result of overstrain of the heart, the effect on the circulation, if any, is to impair its activity. The organ, in fact, becomes enlarged because it is unequal to carry on its function; and the hypertrophy never passes beyond what is required for this purpose.

Prognosis and treatment.—The slighter degrees of enlargement of the heart subside when the cause is removed. But when hypertrophy has developed itself, it is only as the result of long and patient treatment that a cure can be looked for. Physiological rest to the organ is essential. Among drugs the most useful appears to be *aconite.* Da Costa testifies most decidedly to its value when the cardiac impulse is unduly forcible; he

* It had been a striking feature of this case that there was no orthopnœa; the patient, though very dropsical, lay quite low in his bed. At the time the easy state of the breathing was regarded as a further argument against the view that a cardiac affection of whatever kind was the cause of the ascites and of the anasarca; for it has hitherto been the universal opinion that dyspnœa must necessarily be produced by any lesion of the right side of the heart interfering with the blood-supply to the pulmonary capillaries. But it admits of doubt whether this opinion is well founded, and the point is one well worthy of consideration in future cases.—C. H. F.

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generally gave one or two minims of a tincture three times as strong as the British tincture, three times a day; or gr. $\frac{1}{60}$—$\frac{1}{80}$ of aconitia. Walshe speaks very highly of the same remedy; the dose which he recommends is gr. $\frac{1}{8}$ of the alcoholic extract of aconite. The usefulness of this medicine in cases of primary hypertrophy is not inconsistent with what has been stated above as to the symptoms of that affection, for when under the influence of rest and of good nourishing food the cardiac muscle recovers its tone, and the dilated chambers resume their natural size, there is no longer any necessity for so forcible an action of the organ. In some cases *veratrum viride* may be employed with advantage; Da Costa administered drop doses of the fluid extract, or five to ten minims of the tincture, three times daily. *Bromide of potassium* is another remedy which is mentioned favourably by Walshe; the iodide does not seem to be of any service.

If, however, rest be not taken in cases of overstrain of the heart, and if the early symptoms be neglected, the result is that sooner or later the hypertrophy fails in its purpose, and a condition of dilatation succeeds. In such circumstances *digitalis* is often of the greatest possible service.

As the ultimate issue, cardiac hypertrophy and dilatation tend to pass into a condition of "asystole," leading to a series of changes in the lungs and in the liver, and to dropsy of the dependent parts of the body, precisely like the effects of valvular disease, which we shall discuss in the next chapter.

FIBROID DISEASE OF THE HEART.—Unlike fatty degeneration and other diffused lesions, *fibroid disease* is in by far the majority of cases limited to a definite area of the wall of the organ.

Dr Quain described in his Lumleian Lectures for 1872 ('Lancet,' 1872, vol. i) an affection which he terms "connective-tissue hypertrophy," in which he says that the muscular fibres throughout the organ are surrounded by connective tissue in all stages of its development—round cells, spindle-cells, and bundles of fibrillæ. Dr Charlewood Turner, in a valuable paper in the 'Trans. Internat. Med. Congress,' 1881, p. 427, describes, with drawings and figures, the histology of this condition as one of chronic interstitial myocarditis. He observed not infrequently coincident deposition of calcareous salts, but fatty degeneration was rare as a complication. The thickness of the heart's wall is in such cases increased, but the most striking peculiarity is its firm, tough, leathery feel. Dr Quain says that slight degrees of this change have been overlooked, and he refers to a greatly enlarged heart, weighing forty and a half ounces, which had been for thirty years preserved as a specimen of cardiac hypertrophy in the museum of St George's Hospital, but in which upon examination the increased size was found to depend only in part upon muscular overgrowth, the connective tissue being also greatly in excess.

Diffused hypertrophic sclerosis of the heart is, however, very rare as a primary lesion, although it frequently accompanies the consecutive muscular hypertrophy of Bright's disease and of valvular lesions.* But we have had a large number of instances of *circumscribed* fibroid degeneration. No fewer than eleven such cases came under observation in the *post-mortem* room of Guy's Hospital in one period of less than twelve months (1873–4); altogether twenty-seven cases were recorded by the author while he was pathologist. In its slighter degrees it consists in the presence of streaks and patches of a milky white colour in the substance of the muscular tissue. The wall of

* It was present in two of Dr Turner's cases.

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the heart is thereby rendered more hard and resisting to the knife, but it may also acquire a succulent and spongy appearance, and when incised its cut surface looks uneven. Microscopically there is seen a perfectly developed connective tissue, forming wavy bands, running in the same direction as the muscular fibres, some of which, or the remains of them, are still embedded in it. Or there may be a dense fibrous plate, looking like a piece of tendon, and consisting of a glassy substance with regularly arranged fissures or spaces, indicating the planes of fibrillation. It creaks when cut, and sometimes it contains calcareous salts in such quantity as to make it crackle under pressure, like an egg-shell.

The seat of this lesion is sometimes the apex of the heart, sometimes some part of the anterior or posterior wall of the left ventricle, sometimes the septum. It seldom or never begins in the right ventricle, but it may invade it by extension from the left. The fibrous substance is occasionally surrounded on all sides by muscular tissue, so that it touches neither the endocardium nor the pericardium; and it is then very likely to be overlooked, unless all parts of the organ are completely sliced up. In other cases it reaches one or both surfaces to a greater or less extent. The endocardium then shows a marked local thickening and opacity, while the visceral pericardium is found covered with lymph, or adherent to the parietal layer. Some observers have thought that fibroid disease of the heart is generally secondary to pericarditis or to endocarditis, which spreads to the muscular wall of the organ. But this view is clearly inapplicable to many cases; and so far as the endocardium is concerned there is an insuperable difficulty in the fact that inflammation of this structure, apart from the valves, is itself unknown. As regards the relation between pericarditis and fibroid disease, it is to be noted that adhesion is often present only just where the fibroid material reaches the outer surface of the heart, and that even when the whole serous sac is obliterated, the two layers are often found to be very firmly connected together at that spot, but elsewhere so loosely that it is easy to separate them. Moreover, if extension from a general pericarditis occurred, one would expect to find connective tissue dipping into the muscle at a number of different points, which is not the case. And in one instance pericarditis developed itself so as to be recognised by physical signs about two months before death, at a time when the fibroid affection must have been already of long standing.

When the part of the left ventricle that is affected by fibroid disease includes the base of either of the fleshy columns of the mitral valve, the process often spreads into that column, which becomes shrunken. This condition is distinct from the far commoner one in which the summits of the mitral columns undergo conversion into fibrous tissue by extension from the tendinous cords attached to them.

In all cases of fibroid disease of the heart there is considerable destruction of muscular tissue at the seat of the lesion; and we have often found the whole substance of the wall involved from one surface to the other, with not a trace of the normal structure between. The thickness of the wall is, as a rule, much diminished. In one case only was the result to produce an obvious increase of bulk; in that instance the septum was the part affected.

Pathology.—With regard to the pathology of this morbid change there is still some uncertainty; perhaps it is not the same in all cases.

1. In some, it results from a primary process of chronic inflammation,

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a *myocarditis*, arising spontaneously, or from rheumatism. At first, it is said, there is an infiltration of leucocytes; and these subsequently develop into connective tissue. The atrophy and disappearance of the muscular fibres at the seat of the disease is regarded as a secondary effect of the compression which they undergo. The author was never able to discover small-celled infiltration, even at the margins of the fibroid patches; but perhaps the reason may have been that the morbid process was no longer advancing when the patient died. In four of twenty-seven cases taken from the *post-mortem* records of Guy's Hospital there was history of a former attack of acute rheumatism. Another probable cause of chronic myocarditis with sclerosis is alcoholic intemperance.

2. In some instances *syphilis* gives rise to an affection which in its later stage is probably not distinguishable by its characters from fibroid disease due to other causes. At an earlier period it would doubtless be characterised by the presence of gummata, as in eight cases cited by Lancereaux. The development of gummata in the heart is by no means limited to the left ventricle; in two of the cases in question the right ventricle was alone affected, and in one the right auricle. Among our twenty-seven cases of fibroid disease of the heart there were four in which, from the presence of specific lesions elsewhere, the existence of syphilis could be safely asserted. In only one of them were gummata detected; these consisted of a mass as large as a bean within the wall of the right ventricle, and of a number of small, hard yellow points embedded in a reddish gelatinous substance at the growing edge of the fibroid material in the left ventricle. In one instance the morbid process was limited to the septum, this being changed through nearly its whole thickness into a tough fibrous material, with puckering and depression of the adjacent part of the endocardium. The disappearance of gummata in the more advanced stage of the disease corresponds exactly to what is observed in the liver, the testis, and other organs.

3. The formation of *thrombi in the cardiac cavities* may give rise to an inflammatory change, extending through the wall of the part of the heart to which they adhere. A well-marked instance of this occurred in the auricular appendix. It is surely not improbable that the ultimate result might be the formation of a patch of fibroid disease; and such an origin might explain the frequency of fibroid disease at the apex of the left ventricle, for this point is very apt to become the seat of thrombi during the course of enteric and other fevers.

4. In a series of eleven cases of fibroid disease of the heart brought by the author before the Pathological Society in 1874 there was one in which the cardiac muscle presented in addition a peculiar form of degeneration, consisting in its conversion into a dry-looking, greenish-brown substance, of the texture of wash-leather. At one part this formed a thin flat layer, embedded in the substance of the heart, and appearing as a narrow sinuous line in a vertical section. Under the microscope it was found to be merely muscular tissue, which retained its striation, and showed remarkably well the branching and reuniting of the fibres. Its characters contrasted strongly with those of the muscular fibres which lay within the area of the fibroid change; they exhibited but slight striation or had undergone fatty degeneration. In the case of another patient, aged sixty-two, with regard to whom the probable existence of fibroid disease of the heart was repeatedly discussed at the bedside, we found on *post-mortem* examination that the posterior wall of the left

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ventricle was much thinned, and was to a large extent converted into a lustreless yellowish-green substance, almost exactly like that just described; no fibroid material, however, was present. From these two cases it appeared clear that the peculiar change in the muscular tissue must be the primary affection, and that the fibroid development must be secondary; and Dr Ormerod, twenty years ago, suggested ('Brit. Med. Journ.,' 1863) that the latter was due to a "process of conservative substitution, designed to fortify the walls of an attenuated and weak heart." The true relation of the one morbid process to the other has now been demonstrated to be as follows.

Weigert seems to have been the first to state ('Virchow's Archiv,' vol. lxxix, 1880) that fibroid disease of the heart is often the result of a change analogous to the formation of *infarctus* in other viscera. This was afterwards confirmed by the observations of Hüter (*ibid.*, vol. lxxxix, 1882). He found precisely the same dry greenish or yellowish-brown patches as those above described, and traced them to obstruction of branches of the coronary arteries, sometimes resulting from thrombosis, sometimes from embolism, as in one case of endocarditis affecting the mitral valve in a young subject. And he recorded no fewer than eighteen cases of fibroid disease of the heart in each of which the affection was associated with sclerotic changes in the coronary arteries, corresponding more or less closely in distribution with that of the fibroid patches. At the same time he does not think that the formation of actual infarctus is a necessary step in the process by which coronary arterio-sclerosis leads to fibroid disease. It is sufficient that there should be a molecular change in certain of the muscular fibres, from interruption of their blood-supply. The fibroid patches themselves he regarded as the result of an inflammatory process set up by the disintegrating tissue elements. The author drew attention in the 'Pathological Transactions' for 1874 to the relation between fibroid disease of the heart and *arteritis deformans* of the systemic arteries generally, and commented on the fact that the former lesion was present in a very remarkable case, accompanied with absence of pulse in the limbs, which had been recorded by Dr Wilks, and to which reference will again be made under diseases of the blood-vessels. In the second of the two cases in which were found the greenish-looking patches now known to be infarctus, the coronary arteries were extremely diseased, and some of their branches were completely obliterated; but one could not make out that the two morbid changes were definitely related to one another.

Dr Wickham Legg, in his Bradshawe Lecture on "Cardiac Aneurysms" (1883), threw doubt on the correctness of Hüter's view, and expressed the opinion that the lesions found in the coronary arteries in his cases were only such as might have been anticipated from the advanced age of the patients. The importance of the peculiar change in the muscular tissue antecedent to the fibroid development seems not to have struck him. Our experience, however, at Guy's Hospital quite confirms what he says as to the frequent absence of all obvious disease of the cardiac muscles, notwithstanding that the coronary arteries are much obstructed. He cites a case of Dr Greenfield's, in which their orifices were indicated only by small vertical thickenings on the inner surface of the aorta; microscopically all that could be found was a slight but widely distributed fatty degeneration of many of the muscular fibres of the heart. In the year 1880 three instances occurred, in each of which arteritis deformans of the aorta had led to great narrowing of the mouths of both arteries and to almost complete obliteration of at least one of them.

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The cardiac muscles in these cases were either healthy or at most a little soft and pale.*

It might have been expected that the loss of contractile power of so much of the cardiac wall, inevitably resulting from the transformation of the muscular substance of the ventricle in its whole thickness into fibrous tissue, would seriously impair the efficiency of the organ. But Cohnheim has found experimentally that in the rabbit a large part of either ventricle, or even the entire lower third of the heart, may be held fast in a clamp so as to be completely deprived of its function without the arterial pressure becoming lower in consequence. And clinical experience shows that fibroid disease sometimes gives rise to no symptoms whatever. In three of the above-quoted twenty-seven cases, in which it was discovered at the autopsy, the patient had died from some other cause. On the other hand, the frequency with which dilatation and hypertrophy are associated with this morbid process proves that in many instances the systole of the ventricle is more or less interfered with; in ten of our twenty-seven cases the heart was considerably enlarged, weighing from twenty-one to thirty-five ounces.

A peculiar alteration in the shape of the ventricular cavity is produced by the presence of fibroid disease in its wall; it becomes deepened from before backwards, so that the mitral valve lies much further from the anterior surface of the heart than usual. Occasionally the valve is also separated from the posterior surface by a considerable interval.

Cardiac aneurysm.—During the systole any portion of the wall of the heart that has undergone the fibroid change must be exposed to great pressure; one can easily imagine it forming a protrusion like those that are observed in the frog's heart when the action of digitalis is beginning to manifest itself.† Indeed, when the whole thickness of the cardiac muscle is destroyed a permanent yielding of the affected part almost always results. Sometimes it forms a shallow pouch, sometimes a sac of greater or less size, communicating with the ventricle by a comparatively narrow opening. This condition is now generally known as "cardiac aneurysm." This term was used by Corvisart and earlier pathologists to denote general dilatation of the cavities of the heart (p. 24). With this, as described in a previous section, we are not here concerned, nor with the destruction by ulceration of a part of the wall of the heart in connection with a like affection of the valves which is still sometimes spoken of as "acute aneurysm." Leaving these affections out of consideration, we may affirm that cardiac aneurysm, in the modern sense of the term, is scarcely ever a primary lesion; it depends almost always upon a previous fibroid change in the muscle. Dr Legg, in his Bradshawe Lecture ('Med. Times and Gaz.,' August, 1883, ii), has cited three cases which appear to show that it may be produced by fatty degeneration; but there is no doubt that these cases are quite exceptional. The reason doubtless is that in fatty hearts the morbid process is too widely diffused, and the ventricular systole too feeble, for great pressure to be thrown upon any one part of the chamber.

Aneurysm of the heart, like fibroid disease, is met with at all periods of adult life up to a very advanced age. Among Hüter's eighteen cases of fibroid disease, only four of which occurred in patients under sixty years of

* It seems impossible to reconcile Hyrtl's and Cohnheim's statement that the coronary arteries and their branches possess no anastomoses, with the results of the injections made by Dr Legg and by Samuel West, who found that they could readily fill one artery from the other, the two communicating over the surface and at the apex of the heart.

† See a paper by the author and Dr Stevenson, in the 'Proc. Roy. Soc.' for 1866.

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age, there were but four in which aneurysms were present; the ages were fifty-six, sixty-two, seventy-three, and eighty. Both affections are far more common in men than in women.

Cardiac aneurysms do not usually attain a great size, but may occasionally become as large as the fist. They then of course project from the surface of the heart; but the smaller ones, especially if there are several of them, are sometimes excavated within its substance. In a remarkable instance ('Path. Trans.,' 1874) the wall of the left ventricle was tunnelled out in all directions into cavities, of which the largest was as big as a walnut. Fibrin is often deposited in large quantities in the interior of the sac of an aneurysm of the heart; in the specimen just referred to many of the cavities were filled with an adherent greenish gelatinous substance, containing curd-like degenerating flakes. Dr Wilks has placed on record (*ibid.*, vol. viii) a case in which there was found attached to the apex of the heart a cured aneurysm of the size of a pigeon's egg, of which the walls were calcareous and the interior completely consolidated.*

Symptoms.—Clinically fibroid disease and aneurysm of the heart can very seldom, if ever, be diagnosed. The presence of the former affection should perhaps be suspected when cardiac symptoms, without evidence of valvular lesions, are present in a patient suffering from senile gangrene or showing other signs of general arterial disease. A syphilitic history, too, may lead one to infer that the heart has been the seat of gummata, out of which a fibrous tissue ultimately has developed; and it is important not to overlook the possible existence of syphilis as a cause of cardiac disease, on account of the good results that may be anticipated from a prolonged course of mercury and of iodide of potassium. One striking instance of this has come under the author's notice, and Dr Balfour may be cited in testimony that cases of "excited action of the heart with hypertrophy" have yielded to antisyphilitic treatment.

The physical signs of fibroid disease of the heart are undistinguishable from those of enlargement due to overgrowth of the muscle. In either case a systolic apex-murmur may or may not be audible; at one time it may be present and not at another.

In a few instances the pulse has been unusually slow, varying from 28 to 48 in the minute.

When there is a large aneurysm projecting from the heart's apex, a careful mapping out of the area of cardiac dulness might possibly suggest the real nature of the case; but the disease would still have to be diagnosed from an aortic aneurysm pushing downwards into the region ordinarily occupied by the heart. In one instance Skoda is said to have observed bulging of an intercostal space overlying the seat of a cardiac aneurysm.

The *result* of fibroid disease of the heart is sometimes obstruction to the pulmonary and systemic circulation, and consequent dropsy; this was the case in nine among the above-cited twenty-seven cases. In one instance the period that elapsed from the beginning of the patient's illness up to the time of his death was remarkably short, only seven weeks. But other cases have run a protracted course, and have at first been benefited by treatment with digitalis and diuretics; for that which impedes the flow of blood is, after all, not the fibroid affection itself, but the failure of compensation on the part of the wall of the ventricle in general.

* I well remember the autopsy, for it was the first at which I was present when I entered as a student of Guy's Hospital in October, 1856.—C. H. F.

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In a great many cases, however, the heart has gone on discharging its functions quite naturally, so far as can be known, until the patient has suddenly fallen down dead. For example, Dr Whipham ('Path. Trans.,' xxi) has recorded a case of fibroid disease of the heart in a man, aged twenty-nine, who fell dead from his horse while riding in Hyde Park, having started in good spirits and apparently perfectly well, and having never before exhibited any symptoms of cardiac disease. The heart was of normal size and weight, and the valves perfect. The fibrous growth was very extensive, in discrete patches connected with the endocardium. There was no sign of syphilis. The abrupt stoppage of the organ is difficult to explain. But it is perhaps worthy of notice that there is an exact parallel for it in the results of the experimental ligature of one coronary artery, or even of a large branch of one coronary artery, in the dog, as practised by Cohnheim ('Virch. Arch.,' vol. lxxxv). After this operation the heart for a little while goes on beating with perfect regularity, and maintains the arterial pressure at its normal level. But at the end of about ninety seconds its pulsations become somewhat less frequent, and their rhythm is slightly disturbed; and about half a minute later both ventricles suddenly stop at the same instant, after which no stimulus whatever succeeds in restoring their contractions. Evidently, therefore, it is not sufficient, in searching for the cause of sudden death, to examine the orifices and main trunks of the coronary arteries. An arrest of the blood-current through any one of the principal branches must be supposed capable of accounting for it; and equally so whether there be embolism, or whether a more chronic change in the vessel has gradually led to the same result.

Acute Myocarditis.—Acute or subacute inflammation of the muscular tissue of the heart may take place under special conditions, but it is only positively recognised when it occurs in connection with endocarditis or pericarditis. In the latter case it is not at all unusual to see the layer of muscle immediately beneath the serous membrane involved in the inflammatory process; it is pale and soft, whilst the microscope shows it to have undergone a change in texture of a granular or fatty kind. In some exceptional cases the whole thickness of the walls is seen to be affected; then dilatation is apt to occur, and may lead to speedy death or become a permanent morbid condition. At times long subsequent to the inflammatory attack, its effect may be seen in a fibroid patch, showing on section numerous white fibrous streaks interspersed with the muscular fasciculi.

A more acute inflammation of the muscle, or rather one of a different kind, is met with in *pyæmia*, in which numerous small abscesses may be found scattered through the tissue of the organ. A single circumscribed abscess is sometimes met with in connection with endocarditis and disease of the valves. The diagnosis of myocarditis can only be conjectural.

Fatty Disease of the Heart.—(1) *Adipose overgrowth and infiltration.*—The natural layer of adipose tissue beneath the pericardium is often found increased in elderly persons, and within limits this may be regarded as a physiological condition which gives rise to no symptoms during life. It chiefly affects the right side of the heart, especially at the base of the ventricle.

But sometimes the fat grows in upon the muscular fibres so as to thin the cardiac wall, and this becomes a cause of atrophy of one or both ventri-

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cles. Occasionally the *adipose growth* may penetrate right through the wall until it meets the endocardium, and this is particularly seen near the apex of the right or the left ventricle.

Again, there may be *fatty infiltration*, the adipose tissue increasing between the fibres. A large amount of interstitial fat in the muscles of the limbs, as in cattle fatted for the market, and in men who drink largely of beer, is probably indicative of over-feeding and under-work. When it affects the diaphragm it may become more serious. But it is most dangerous when the cardiac tissue is so infiltrated, as frequently occurs in conjunction with adipose overgrowth and sometimes alone. In such cases it is probable that, though no certain signs or symptoms of its presence arise, it may be the immediate cause of death. Obesity is notoriously a bad condition for recovery from surgical operations and other injuries, and a "weak heart" is often the cause of the want of repair and fatal issue.

(2) *Fatty degeneration.*—This is a different pathological condition, recognised as such by Laennec. The heart may be free from adipose tissue, but its tissue is pale, soft, and flabby. On the inner surface, particularly of the musculi papillares of the left ventricle, pale yellow zigzag markings are seen, described in the 'Med.-Chir. Trans.,' vol. xxxiii, by Dr Quain as "tabby degeneration." Under the microscope the fibres are found to have lost their striæ, and black granules appear instead, at first in transverse lines, as if the change had affected disc after disc. Next the dark granules become larger and acquire a bright glistening centre, and all trace of structure disappears.

According to Dr Hermann Weber,* the amount of ethereal extractives from such hearts is not greater than normal. Dr Stevenson, however, as quoted by Wilks and Moxon, found the fatty matter nearly doubled; and Krylow has apparently settled that there is a decided relative increase, though much less than one would have anticipated.

The most frequent *causes* of this fatty degeneration of the heart (which is quite independent of obesity either of the heart or other parts) are anæmia, and certain poisons. It is most constant and well marked in those remarkable cases of idiopathic anæmia first described by Addison, and since known under the names of "anémie grave" and "perniciöse Anämie." It is also often found in cases of leuchæmia and Hodgkin's disease, and occasionally in phthisis, cancer, and other wasting and anæmic disorders. It is a constant appearance in fatal cases of poisoning by phosphorus, when the liver is also the seat of remarkable fatty degeneration; and the same thing has been observed in poisoning by arsenic, by mercury, and by lead. It has sometimes but not constantly been observed when the coronary arteries have been much diseased. It does not appear to be a frequent or extensive concomitant of cardiac hypertrophy and dilatation, whether due to valvular or other lesions, nor of fibrous sclerosis. Lastly, acute fatty degeneration often occurs as a superficial change immediately beneath an inflamed pericardium.

The *symptoms* of true fatty degeneration are very obscure and doubtful.

* "Zur Lehre von der fettigen Entartung des Herzens," 'Virchow's Archiv,' xii, 326 (1857). Dr Weber has informed the editor that subsequent extension of his inquiries confirmed his previous conclusion. The wasting of the normal adipose tissue and the small extent of the degenerative process, often limited to the left ventricle, may explain these results. Böttcher, however, and Valentiner support the same conclusion as Stevenson.

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It is usually surmised from our knowledge of pathology rather than diagnosed by physical signs.

The cardiac impulse is described as being weak but irritable, or sometimes "slapping," *i. e.* distinct but short. The first sound is often accentuated and has lost its booming character, so as to resemble the second. The radial pulse may be quite unaffected. General symptoms of lividity, dyspnœa, irregular pulse, &c., probably only appear when there is concomitant dilatation.

When a person past fifty, pale and thin, with a white, soft, "satiny" skin and early arcus senilis, suffers from dyspnœa, and his heart gives a short, sharp first sound, the presence of fatty degeneration of the heart is probable; and it becomes almost certain if grave anæmia is also present.

The *result* of fatty degeneration is undoubtedly in certain cases sudden and fatal syncope. Often, however, it is found after death has occurred in other ways. It does not appear in itself to lead to dilatation, to which it is rather secondary; but it certainly may end in rupture of the heart.

Experience in the deadhouse does not bear out the assertion that fatty degeneration is a frequent complication of valvular disease. In fact, this lesion usually occurs unexpectedly to the pathologist, though it is constantly diagnosed at the bedside. On the whole, ingrowth of adipose tissue is probably a more freqnent and perhaps a more dangerous lesion than interstitial fatty degeneration.

A granular or brown *degeneration* of the cardiac muscles has been frequently observed, but its relations to fatty degeneration and its pathological significance are still obscure. Spongy or cavernous degeneration has been observed in the fœtal myocardium by Virchow.

The rarest form of cardiac degeneration is that which has been described by Köster as *calcareous infiltration.* A good account of it, with two original specimens figured, is given by Dr Coats in his 'Manual of Pathology.' It sometimes complicates cases of fibroid sclerosis and cardiac aneurysm.

RUPTURE OF THE HEART.—This rare and interesting pathological condition owes its practical interest to the importance of discriminating it from rupture as the result of injury. In the latter case the lesion is almost always in the right ventricle or one of the auricular appendages. In idiopathic rupture it is almost always in the left ventricle; of fifty-five cases, forty-three affected the left ventricle and seven the right: the auricles are still more rarely found ruptured. In most cases the muscle is already weakened by fibroid, fatty, or granular degeneration; but several instances are on record in which no such changes have been detected.

The age of the patient is generally over sixty. George the Second died of rupture of the heart at the age of seventy-six: it is remarkable that he was the first English sovereign who exceeded the age of seventy.

The rent does not always go through the entire thickness of the wall, and may possibly in such cases be recovered from; but usually hæmorrhage into the pericardium ensues from the torn vessels, and causes death as certainly, though not so rapidly, as when a larger rent opens directly into the ventricular cavity.

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ENDOCARDITIS AND VALVULAR DISEASE OF THE HEART

> "Oft have I seen a timely-parted ghost
> Of ashy semblance, meagre, pale, and bloodless,
> Being all descended to the labouring heart,
> Who in the conflict that it holds with death
> Attracts the same for aidance 'gainst the enemy,
> Which with the heart there cools and ne'er returneth
> To blush and beautify the cheek again."—2 *K. Henry VI*, iii, 2.

ENDOCARDITIS—*The subacute, simple, or benign form—Its relation to rheumatism and its ætiology generally—Its anatomy—Its signs.*
Chronic, Fibroid, or Sclerotic Endocarditis—Its origin and anatomy.
The Ulcerative or Infective Form—Its origin, diagnosis, anatomy, and results—Its relation to pyæmia—Prognosis.
VALVULAR LESIONS—*Their origin, anatomy, and ætiology—Effects upon the heart itself and upon other organs—cardiac dropsy—dyspnœa and orthopnœa—hæmoptysis and other symptoms—Diagnosis: the murmurs attending on valvular lesions—their physical cause—their rhythm—their locality and conduction—Aortic stenosis—its signs and symptoms—Aortic regurgitation—Mitral stenosis and regurgitation—Lesions of the pulmonary valve and of the tricuspid—Functional murmurs—Relative frequency and general prognosis of valvular lesions—Treatment of organic diseases of the heart.*
Congenital lesions of the cardiac valves—Disease of the heart in children.

CARDITIS, or inflammation of the substance of the heart, was formerly described, but rather in accordance with the belief that every tissue was liable to every disease than from observation. There is no true carditis as there is no true inflammation of the brain. The affections of the muscular tissue of the heart have been described in the last chapter. The inflammations of the heart do not directly concern the muscle, but the covering and lining membranes. The former, pericarditis, will form the chief subject of the next chapter. The latter, endocarditis, with its effects, is still more important, and will now be considered in its three distinct forms.

SIMPLE ACUTE ENDOCARDITIS.*—The first is a somewhat acute endothelial inflammation closely resembling pericarditis, pleurisy, and peritonitis. The structure of the lining membrane of the heart is almost identical with that of the great serous cavities, the chief differences being the absence of close relation with the lymphatic system; this and the fact of the former membrane being continually bathed with the circulating blood are the most important peculiarities of the endocardium to bear in mind. It is remarkable that although the endocardium is continuous with the intima of the arteries and veins, no acute idiopathic inflammation of the latter is met with.

* *Synonyms.*—Subacute endocarditis—Endocarditis simplex v. benigna—E. verrucosa et vegetans—Rheumatic endocarditis.

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The process is acute or subacute in course, mild in its effects on the condition of the body in general, serous and fibrinous (or, as the German pathologists call it, "croupous") in the character of its exudation, adhesive and contractile in its effects. Adhesions, however, can only occur where the parts affected come into contact, and hence are limited to the neighbourhood of the valves; "puckering" and the formation of delicate bead-like nodules of fibrin are the characteristic effects on the valves, which have led to the epithets "warty" (*endocarditis verrucosa*) and "vegetative" being applied to this form of inflammation. Away from the valves, endocarditis produces opacity and thickening, like the milk-spots of the pericardium.

Endocarditis is usually limited to the left chambers of the heart; it is extremely rare to find the right auricle, ventricle, or valves affected, except along with those of the left side, and in the great majority of instances they do not even thus share in the inflammation. The exception that the right side is *more* frequently affected by endocarditis in the fœtus seems clearly to show that the more active function of the systemic half of the heart is the cause of its being more obnoxious to disease in extra-uterine life.

Ætiology.—By far the most frequent cause of acute endocarditis is Rheumatism, *i. e.* the acute febrile disease, associated with multiple synovitis, which is often called rheumatic fever. The so-called "chronic rheumatism," "muscular rheumatism," "rheumatic gout," "gonorrhœal rheumatism," and the other disorders which are still often vaguely styled rheumatic, show that they have no claim to the title by not producing endocarditis.

The only other diseases which probably produce endocarditis as an occasional complication are Chorea, Pyæmia (see three cases published by the author in the 'Path. Trans.' for 1866), and Scarlatina. Some authors mention smallpox, measles, and diphtheria, and others acute or chronic Bright's disease, but the evidence on which these statements rest is far from conclusive. Even scarlatina is perhaps only operative by its liability to be followed by multiple synovitis, which is itself in most cases genuine rheumatism; a choreic murmur has been most often preceded by rheumatism; and pyæmic endocarditis is very rare and of no clinical importance. Hence we may practically consider acute non-infectious endocarditis as "rheumatic" in the strict sense of the word.

It usually begins early in the course of the fever, and occasionally even before the synovitis has appeared, or altogether without synovitis, as seems proved by the testimony of such authorities as Graves, Stokes, Trousseau, and Latham.

Acute endocarditis is more frequent in the rheumatism of children than in the case of adults, and far more common between puberty and thirty years of age than later.

Anatomy.—Endocarditis is recognised by the increased vascularity of the serous membrane, and by an exudation which causes some roughness of its surface and thus leads to deposition of fibrin on the valves. It is this lodgment of fresh fibrin on the inflamed surface which constitutes the peculiarity of endocarditis, and makes it differ from inflammation of other parts. The living blood is always ready to deposit fibrin on a roughened surface, and especially when the current is in any way checked. Moreover, as Dr Moxon has conclusively shown, these fibrinous concretions again set up inflammation in neighbouring parts with which they come in contact.

The first indication of endocarditis is seen in redness and vascularity of the valve, afterwards in the production of a row of granulations along the

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lines where the cusps meet. These are bead-like elevations formed along the delicate curved line on each side of the corpus Arantii where the sigmoid valves touch, and along the corresponding edges of the mitral or tricuspid valve. If examined microscopically these nodules are found to consist of ordinary inflammatory elements, leucocytes, and fibrin. The whole of the valve may also become infiltrated with these inflammatory products, and thus being swollen and softened is ready to undergo still further changes. In recent endocarditis these granulations along the valves are the first and only indications of the inflammation. At a later stage they become larger and confluent; and hanging down into the stream of blood, attract coagulating fibrin, which collects upon them in large quantities. Thus vegetations on the valves have a double origin, being in part derived from the valve itself, and having therefore a true inflammatory source, and in part derived from a deposition of fibrin from the blood; the two products run into one another, and are not clearly separable.

Symptoms of endocarditis.—The presence of endocarditis is indicated by the occurrence of a murmur or bruit heard over the region of the heart. If, for example, in acute rheumatism a bruit arise near the apex of the heart we suppose that inflammation has been set up in the neighbourhood of the mitral valve; if it occur at the base we believe it may be associated with inflammation of the aortic valves. In the latter case the diagnosis is less certain, because bruits of a temporary, probably hæmic, character are often met with in acute rheumatism. The disappearance, however, of the bruit by no means warrants the positive denial of endocarditis, since there is good reason to believe that an inflammation of the valves may be recovered from; witness the case of the disappearance, on recovery from chorea, of the bruit which in many cases appears to be due to an organic change in the valve. The late Dr Sibson made the observation (and it has been corroborated by others) that, preceding the occurrence of a bruit in rheumatic fever, the first sound may often be prolonged.

The general symptoms of endocarditis are very slight. In itself it probably produces little pyrexia, and only moderate acceleration of the pulse, nor is pain by any means a constant symptom.

The *prognosis* is favourable as far as the endocarditis is concerned. The evils arise first from embolism, and secondly from the deformities of the valves which are likely to be produced.

CHRONIC FIBROUS ENDOCARDITIS.*—Acute or subacute inflammation of the valves often leads only to puckering and incompetence of the cusps, with consequent regurgitation of the blood-stream; but often there is so much thickening and contraction that obstruction is the result, with or without concomitant leaking from incomplete closure. Chronic endocarditis produces similar effects; but while in many cases it is merely the last stage of acute endocarditis, it is in many others a slow and insidious process from the first. Without previous rheumatism, the valves slowly thicken and the ostia contract. This process, chronic from the beginning, is found associated with gout, alcoholism, and cirrhosis of the kidneys. It is not infrequent in youth, but becomes more common in adult and later periods of life. It is often complicated by deposition of phosphates of lime and

* *Synonyms.*—Sclerotic endocarditis—Valvular atheroma—Chronic fibroid contracting endocarditis—Endurcissement cartilagineux (*i. e.* fibrous) et osseux (*i. e.* calcareous) des valvules du cœur (Laennec).

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magnesia, with carbonates of the same bases, in the indurated fibroid tissue. It is thus nearly identical anatomically with atheroma of the arteries, and is associated with the latter affection clinically as well.

The relation to rheumatism is through the acute form of endocarditis; probably more than half of the cases are thus rheumatic in origin. See the writer's paper in the 'Guy's Reports' for 1871, and Sir Dyce Duckworth's in the 'St. Bartholomew's Reports' for 1877.

From an analysis of the records in Guy's Hospital, Dr Pitt has found that sclerotic endocarditis, as it affects the mitral valves, and leads to stenosis of the left auriculo-ventricular ostium, is frequently associated in men with the more chronic forms of Bright's disease (renal cirrhosis), and in women with uterine disorders which lead to ascending nephritis or to consecutive renal cirrhosis.

The late Dr Peacock and some other writers have believed that a large number of these cases are congenital, but probably this is very exceptional.

The origin of this sclerotic form of valvular inflammation is therefore to be sought (1) in the acute or subacute rheumatic form, (2) in gout and chronic Bright's disease, or rather in the sclerotic "diathesis," if we may use such a phrase, which forms part of arterio-capillary fibrosis, (3) in strain on the aorta and its valves from over-exertion, while (4) a certain number of cases appear to be idiopathic from the first, and some of these perhaps are congenital.

The symptoms, the prognosis, and the treatment of chronic endocarditis are identical with those of the structural valvular lesions which it produces.

Acute Ulcerative Endocarditis.*—Of late years it has been possible to draw a marked distinction between the simple benign form of cardiac inflammation above described and a more acute, more dangerous, and septic form. It may be compared with the virulent forms of peritonitis which occur in the puerperal state. Beside the usual fibrinous products of endocarditis (which are present in exuberant amount) there is a breach of surface of the endocardium, often leading to rupture of the chordæ tendineæ or perforation of a valvular cusp. Moreover, micrococci or septic bacteria are found in the masses of fibrin, and the morbid process is accompanied by pyæmia and other symptoms of septicæmia. Lastly, the detached fragments of fibrin, which in the case of ordinary endocarditis produce only mechanical results by blocking the arteries into which they are carried, become here the means of transport of infective microphytes and ptomaines, which excite similar suppurative, "pyæmic" inflammation wherever they lodge as emboli. Hence this form of endocarditis has been termed "malignant" by Dr Osler in his Croonian lectures (1885), and the whole process was described by Dr Wilks as one of internal or arterial pyæmia.

The *origin* of this form of endocarditis, like that of the preceding, is most frequently in rheumatism. It most frequently occurs as a secondary process affecting a valve already injured and deformed by chronic sclerotic inflammation. It very rarely begins as a secondary result of chorea, scarlatina, or pyæmia, and is unknown as a consequence of Bright's disease, of gout, or of syphilis. Ulcerative endocarditis, like the benign form, is for the most part confined to the left side of the heart, but sometimes affects

* *Synonyms.*—Acute diphtheritic endocarditis (Eberth)—Malignant endocarditis (Osler)—Infective endocarditis.

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the pulmonary or tricuspid valves in addition. It only produces a similar affection of the arteries when an embolus lodges.

Its *diagnosis* depends, first, on the same physical signs as that of the benign form, for the mechanical effect of both on the valvular mechanism is the same; and secondly, upon the raised temperature, and the signs of infective embolism of distant parts. When we find a patient in a state of fever, and discover a cardiac bruit; when to this are added hemiplegia, aphasia, or other symptoms of cerebral embolism, albuminuria or hæmaturia, pointing to embolism of the kidneys, or increased splenic dulness, with a palpable tumour and tenderness in that region; or when an acute aneurysm forms in the arteries of the limbs—we may then safely diagnose ulcerative endocarditis.

Anatomy.—The first stages are like those of the benign form, but the fibrinous masses are found more abundantly, and are more easily detached. Hence embolism is particularly common.

When these vegetations are long and mobile, they come into contact with the surface of the ventricle, and there set up a fresh inflammatory process; so that the part touched is also soon covered with a patch of fibrin. In this manner a vegetation on one valve may bore a hole through another with which it comes in contact. There are few cases in which these secondary effects are not seen, and of the valves the mitral seems more likely to be affected than the aortic.

Subsequently the texture of the valves becomes involved, the tissue is loosened, and the valves become much altered in structure. The inflammation may go on to *ulceration*, and a rent or perforation may take place in the aortic or mitral cusps. At other times an aortic valve may be found partially detached from its base, or the chordæ tendineæ of the mitral ulcerated and broken, their loose ends floating about and covered with fibrin. As a consequence also of these inflammatory changes, thickening and adhesion or coalescence of the valves may take place, leading to obstruction at the orifices, or, on the other hand, retroversion of the valves, leading to reflux or regurgitation.

Acute ulcerative endocarditis is not so common an affection as the more chronic process. It often spreads by friction of fibrinous concretions. These rub against the wall of the cavity and there produce an ulceration followed by a further deposition of fibrin, or, proceeding further onward, invade the muscle until an abscess is formed. This finally discharges its contents into the heart and constitutes an *acute aneurysm*. It usually occurs at the root of the valve, and it may sometimes reach the surface of the heart, when if it bursts it sets up a fatal pericarditis. Although the fibrinous concretions spoken of are probably the usual instruments in the production of the aneurysm, the latter may also arise independently of them.

In some cases of ulcerative endocarditis the inflammatory products become detached, and infecting the blood, set up a fatal blood-poisoning; micrococci are frequently met with in them, as was first shown by Heiberg, of Copenhagen. The result is infectious embolism causing "pyæmia," abscesses of the spleen, kidneys, and other viscera, including the heart itself, and aneurysms of distant arteries.

Occasionally a diastolic bruit suddenly occurs in the course of rheumatic fever. This implies that the inflammation of the aortic valves has caused ulceration and laceration of one of the segments.

The *prognosis* of ulcerative endocarditis is always very grave. The

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patients frequently die from pyæmia, but sometimes they recover remarkably.

No efficient *treatment* is known; but quinine is given in large doses, and stimulants are usually exhibited.

VALVULAR LESIONS.—The effect of inflammation of the endocardium where it lines the cavities of the heart is of little practical importance, but not only do the valves appear to be more liable to inflammation than the rest of the endocardium, but a very little damage to their delicate structure will interfere with their mechanical action. It is not always easy to say precisely how the early stages of acute endocarditis produce this effect. One would scarcely have supposed that a few minute nodules of lymph upon the edge of a mitral curtain would prevent its closing effectually when the ventricle contracts. Probably we must explain it by calling to mind the difference between acute inflammation of the skin, the eye, or the throat as seen during life, and its scarcely perceptible effects in the dead-house. If we imagine the curtains red, swollen, and œdematous, it is easy to understand how they fail in the nice coaptation upon which their efficiency depends. This at all events is certain, that in acute endocarditis the only physical signs met with are those which denote incompetence of one of the valves. Obstruction from contraction of an orifice can only be produced by a chronic process.

When, as in the ulcerative form of endocarditis above described, the valve is destroyed or perforated, its inefficiency is easy to understand.

The effect of chronic or sclerotic endocarditis is permanently to pucker and deform the curtains so that they no longer perform their part. At the same time they become opaque and thick, less mobile and less flexible. Moreover, in the case of the mitral and tricuspid valves the chordæ tendineæ also thicken and contract, so as to prevent their floating up while the ventricle is filling.

Another process, however, is a frequent, one may almost say a normal termination of the same series of changes. The circumference as well as the free edges of the valves slowly contract after the inflammation is over, forming a true, fibrous, cicatricial tissue which gradually narrows the orifice affected. When calcareous is added to fibrous degeneration the narrowing is aggravated by the bulk of this new material. But beside the contraction of the whole aperture, the practically available opening for the passage of the blood is still more liable to be diminished by adhesions of the curtains or cusps, and this process is no doubt answerable for the most extreme cases of valvular stenosis. When this has occurred, the usual effect of the pressure of the blood-stream is to push the coherent valves into the ventricle or the outgoing artery, as the case may be, and thus form a more or less funnel-shaped septum with a perforated apex. In extreme cases this perforation becomes a mere pin-hole and the orifice is practically obliterated, a condition seen in the case of the pulmonary orifice alone.

Lastly, when severe ulceration and large fungated masses of fibrin are produced by septic endocarditis, the condition of the valve is liable to almost daily change. Regurgitation from extensive loss of substance of the curtains may be partially checked by deposit of fibrin, or this may happen still further to shackle the cusp which has escaped ulceration; or one or more chordæ tendineæ may ulcerate through and break, allowing the curtain to which they are attached to break loose like the sail of a ship and flap back into the auricle during systole.

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Lastly, destruction of the valves may go so far, as is particularly seen at the orifice of the aorta, that one or more cusps are wholly eaten away, so that it sometimes seems as if almost the whole of the blood which leaves the left ventricle at its contraction must return with diastole, and one wonders how the circulation can have been kept on, however imperfectly, for an hour.

Effects of valvular disease on the heart.—It may be laid down as a rule that obstruction in any cavity or passage causes hypertrophy in the muscular structure behind it, as seen in the case of the bladder from stricture or in the intestine from a chronic obstruction; in like manner stenosis of the aortic orifice produces hypertrophy of the left ventricle, and stenosis of the mitral hypertrophy of the left auricle. Regurgitation through a valve causes enlargement of the cavity, from its being continually over-distended; it follows, therefore, that the left ventricle will be dilated in aortic regurgitation, and the left auricle in mitral regurgitation: in the latter case the enlargement is not excessive, seeing that the blood is thrown back directly on the lungs, which help to bear the pressure.

Again, it is evident that any obstruction on the left side of the heart must affect the lungs, and so react on the right side of the heart; just as in chronic bronchitis, when the blood flows with difficulty through the lungs, the right ventricle of the heart becomes hypertrophied, and as a consequence the right auricle also.

If we take the case of mitral stenosis, we find that the left auricle, in order to propel the blood through the narrow orifice, has more work thrown upon it, and becomes in consequence much hypertrophied, and at the same time its lining membrane opaque and thickened; the blood in the lungs in like manner must be retarded, and the right side also becomes enlarged, as in bronchitis.

In advanced cases of mitral stenosis, owing to the whole current of blood being reduced to the measure of the mitral orifice, the left ventricle becomes somewhat diminished in size, and the whole heart smaller than in health.

In the cases of sigmoid disease in which there is an impediment to the flow of blood as well as regurgitation, the left ventricle becomes hypertrophied and dilated to a very great size. This impediment to the escape of blood from the aorta arrests the flow behind, and therefore the left auricle participates in the enlargement, and as a consequence the right side of the heart also. In such a case the whole heart is immensely enlarged, and is called "bovine." It frequently weighs forty-five ounces, and much heavier hearts are sometimes met with.

Effects upon other organs.—It will be convenient to mention in this place the changes which take place in other organs from the long-continued congestion. In the case of the *lung* this may be so great that blood is brought up during life, and the organ is found after death to have blood effused into the tissue; this may be scattered through the substance or seen as large round circumscribed dark masses, described by Laennec and other French writers as "pulmonary apoplexy." They have been compared to damson cheese in appearance. These usually occur at the lower parts of the right lung. If the blood do not actually burst through the vessels, the engorgement goes on until the capillaries are completely blocked and the alveoli of the lungs become almost obliterated; in consequence of

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this the lung becomes very dense, hard, and fleshy, and sinks in water, the cut surface being smooth. This condition is sometimes called "splenization." Sometimes, after a time exudation takes place, a large quantity of pigment is formed, the alveoli are thickened, and the lung becomes dark and granular; this is termed "brown induration." If more marked inflammatory products are thrown out, and some formation of connective tissue takes place, the consolidation which then arises, combined with the granular pigment, produces an appearance which has been termed "brown indurated pneumonia." A close examination discovers the remarkable changes which have occurred in the vessels of the lung from the long-continued pressure; sometimes they have become varicose, and atheromatous degeneration of the pulmonary arteries is frequently seen.

The *liver*, which from the long-continued engorgement becomes much enlarged, is found to be in the state described as "myristicated," or like the mottled section of a nutmeg. The arrested flow of blood in the hepatic veins produces a similar congestion of the portal circulation as well as a stagnation of bile in the ducts; the capillaries become choked, fatty degeneration of the cells takes place, and the secretion becomes arrested. The appearance of the liver is altered in a most characteristic manner, the fatty degeneration of the circumference of the lobule giving it a white border, and this white being mixed with the red of the blocked hepatic veins and the yellow of the obstructed ducts produces the myristicated appearance. It is still a question whether production of new connective tissue may also take place. Several authors speak of this condition going on to cirrhosis, so that in course of time a structural disease of the liver is produced. This, however, has never been thoroughly proved.

The *spleen* is found in cases of valvular disease to be hard, dark, and small, sometimes with fibrinous wedges from emboli. In cases of ulcerative endocarditis it is swollen and soft, as usual in pyrexia from any cause, and often contains congested wedges (infarcts) from the presence of septic emboli. These are sometimes found softened down into pyæmic abscesses.

The *kidneys* are large, hyperæmic, tough, and coarse-grained. In the end the congestion leads to a form of nephritis which, without producing the typical form of the large white kidney, may gradually cause shrinking, and thus simulate the appearance of the true cirrhotic kidney.

The *stomach* is intensely injected, often with petechial spots or hæmorrhagic erosions, and its surface is covered with mucus.

The *pancreas* is indurated, not obviously hyperæmic like the kidney and the spleen.

The *uterus* partakes in the universal venous congestion, so that its mucous membrane is red and velvet-like, and hæmorrhage sometimes takes place as if from returning menstruation.

Cardiac dropsy.—The effect of long-continued venous congestion in all loose and open cavities is to produce transudation of serum by mechanical pressure. The water, with the salts and albumen in solution, exudes through the walls of the vessels and collects wherever there are open spaces to receive it. This is not a process of true exosmosis, for serum albumen is a colloid; nor is it a process of inflammatory exudation, for there is no transmigration of leucocytes, nor is the transuded fluid coagulable. It is serum, but neither pus nor coagulable lymph. Such watery exudation or dropsy does not take place in the liver, spleen, kidneys, or brain, nor even in the spongy tissue of the lungs. By an exception which is not easy to explain

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satisfactorily, the result of the long-continued congestion of heart disease in these viscera is induration. The globe of the eye is too completely filled to admit of dropsy, nor do we find hydrocephalus in cardiac cases, nor yet œdema of the muscles. In the brain such a result is prevented by the communication between the lymph-spaces of the brain and the vertebral canal, in the muscles by the lymphatic circulation which is here probably at its best. Practically, therefore, the seat of cardiac as of other forms of dropsy is in the mesoblastic cavities lined with endothelium, which form the areolæ of the connective tissue under the skin, and are expanded into the great lymph-sacs of the pleuro-peritoneal cavity. A considerable amount of fluid is sometimes found on one side or other of the chest. Less frequently there is notable ascites, and in most cases there is a moderate amount of fluid in the pericardium. In the tunica vaginalis the quantity must be very small, for one does not meet with passive hydrocele in cases of dropsy.

Cardiac dropsy is as a rule less extreme than that produced by some forms of renal and hepatic disease. Such excessive anasarca as goes with the large white kidney and the enormous ascites of cirrhosis of the liver are scarcely ever observed. The mechanical character of cardiac dropsy is shown by its strict conformity to gravity. The face is occasionally swollen after lying down, but as a rule is free from œdema. The lower extremities are most affected, the peritoneum, and one or both pleuræ. The abdominal walls are not œdematous as in renal dropsy, nor does the scrotum swell. There is often considerable œdema of the arms, which may vary from one side to the other as the patient lies in bed. Such œdema of one arm where there is no local cause of pressure on the axillary vein is very characteristic of cardiac dropsy.

Other symptoms.—Next to the arterial anæmia and venous congestion with the dropsy which follows comes *dyspnœa*, which is often the first sign of cardiac disease. It depends upon deficiency in the lesser circulation. The air finds free passage to the air-vesicles and pulmonary capillaries, but the blood is not sent through them with sufficient rapidity to ensure complete aëration. Hence ensues, first, rapid breathing, and next deep and laboured respiration. This is, of course, increased on exertion, like dyspnœa due to whatever cause, and is most observable when the patient ascends a hill or goes upstairs. The form of dyspnœa which compels the sufferer to maintain an upright attitude is very characteristic of that due to cardiac disease, so much so that decided orthopnœa without dyspnœa, except in the recumbent attitude, should always suggest disease of the heart.

Cyanosis as the result of dyspnœa is always present, but is usually masked by the anæmia and wasting which accompany heart-disease. It is very rarely that we see the congested purple face and eyes and hands which are characteristic of severe capillary bronchitis and acute laryngeal obstruction. When marked cyanosis is present, the cardiac lesion is almost always a congenital lesion of the right side.

Congestion of the tip of the nose and the lobules of the ear may be seen, and sometimes of the chin and cheeks; that venous stasis is present is denoted by the frequent occurrence in the most chronic forms of valvular disease of clubbing of the fingers and toes. A further indication of chronic venous congestion in advanced cases of cardiac disease, particularly mitral regurgitation, is a liver enlarged and tender and sometimes pulsating; while renal congestion is shown by the scantiness, high colour, and abundant lithates of the urine, together with the presence of albumen.

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The patient suffering from heart disease, beside breathlessness, palpitation, and dyspepsia, suffers from disturbed and *sleepless nights*. As soon as he falls off he starts up in fear of suffocation. The best position for the patient is the dorsal one with the head and shoulders raised, a position unaccustomed, and therefore incompatible with the usual rest; again, for the production of sleep a quiet regular circulation through the brain is necessary, but this is often impossible when a heart is beating too frequently and with various degrees of force. Moreover, the medulla oblongata of the respiratory function is blunted; for although it is said that the spinal system never sleeps, the statement is true only in a sense; during ordinary sleep the deep breathing certainly indicates a change in the respiratory process. In disease of the heart a similar change, probably due to an altered vascular state of the centre of the vagus in the bulb, immediately produces suffocative symptoms and awakens the patient. The peculiar form of respiration known as Cheyne-Stokes respiration is often present, ascending and descending respiration, marked by a series of respirations becoming shallower and shallower until they altogether cease; then after a long pause the series begins again (cf. *supra*, vol. i, p. 945).

Hæmoptysis.—The above are the chief subjective symptoms complained of, but before long the disturbance of the circulation gives rise to external signs also. In mitral disease, in a certain number of aortic cases which have advanced to the last stage, and in various primary affections of the muscular tissue of the heart, the venous circulation becomes obstructed, and the changes already described ensue in the various organs of the body. The cardiac apnœa becomes aggravated by *congestion of the lungs;* the patient has a cough, and begins to expectorate mucus tinged with blood, and sometimes enough comes up to be called hæmoptysis.* On examining the patient, beside discovering mucous râles, we find that the lower part of the chest is imperfectly filled with air, and perhaps there is dulness on percussion at the base of both lungs; as a rule it is the lower lobe of the right lung which first becomes airless, but no satisfactory explanation of this prevalent engorgement of the right side has yet been forthcoming. At this time the lung is in the condition above described as "splenization," and if blood has been expectorated there are probably effusions of blood in its tissue—so-called pulmonary apoplexy. The coloured mucus implies an intense congestion of the bronchial membrane also.

Hæmatemesis is a much less frequent symptom of valvular disease, and is seldom large in amount. The blood vomited is dark, venous, and more or less discoloured; sometimes digested, so as to resemble brown coffee-grounds.

The physical signs of valvular disease.—Palpation and percussion determine the size of the organ and the probable condition of its several cavities, and this furnishes us, as we have seen in the last chapter, with the best evidence we possess of their dilatation or hypertrophy. Dilatation and hypertrophy

* Dr Shaw, then Medical Registrar at Guy's Hospital, found, from an analysis made for the second edition of this work, of 262 adequately reported cases of valvular disease, that hæmoptysis was most frequent in those of mitral stenosis, much less so in those of aortic regurgitation, with or without obstruction, and least frequent in those of uncomplicated mitral regurgitation. Hæmoptysis occurred in 29 per cent. of the whole number of cases; in 45 per cent. of those with mitral stenosis, and in less than 20 per cent. of those without it. Or, put another way, of the total number of cases of hæmoptysis (76 out of the 262) 45, or more than 59 per cent., were cases of mitral stenosis.

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are most frequently the results of valvular lesion, and therefore aid in its diagnosis; but the direct evidence of the presence and nature of the affection of the valve depends upon auscultation.

Occasionally immediate auscultation is practised, as, for instance, in listening to the cardiac sounds in a man under examination for insurance, when there is no reason from the pulse or any other cause to suppose that the heart is otherwise than healthy. But for all careful or critical observations the stethoscope is needful, and it is well to employ the double as well as the single stethoscope for the purpose, resting it upon the uncovered cardiac region, and taking care that it is not in contact with the patient's dress.

The point of impulse is first ascertained by inspection or palpation, and we then listen at that spot for the first sound, next at the junction of the third left costal cartilage with the sternum for the second sound, and afterwards trace any abnormal murmurs from the apex to the axilla and the back, or inwards to the ensiform cartilage, and from the base upwards towards each clavicle and down the sternum.

The sounds of the heart may be modified in many ways, without there being what is technically called a murmur.

(1) They may be *faint* or absent, due sometimes to the feeble action of the ventricle, sometimes to displacement of the heart, so that its sounds are not to be heard in a normal situation; sometimes to pericardial effusion or emphysema, bringing an ill-conducting medium between the organ and the ear. But often the cardiac sounds are feebly heard when the heart is perfectly healthy, owing to the thickness of the walls of the chest from great muscular development, or from subcutaneous fat.

The first sound of the heart is frequently deficient in loudness in cases of hypertrophy of the left ventricle; at the same time it is often prolonged and indistinct, so that it becomes impossible to say whether a faint bruit, an impure or prolonged first sound, or a murmurish sound, is the most appropriate description. A change in the opposite direction to a higher, clearer, and more musical character makes the first sound resemble the second, and is often observed in cases of cardiac dilatation.

(2) Increased *loudness* of the sounds, while they continue normal in quality, seldom points to organic disease. It is the result of violent action of the heart, and is constantly observed with the palpitation due to great exertion and temporary dyspnœa, or to that of mental excitement. When this sign is present the patient should be allowed to sit quiet or to lie down for a few minutes before proceeding with auscultation.

The second sound is increased in loudness, and acquires a sharp, ringing, almost metallic character when the tension of the pulmonary or aortic cusps is much increased. This "accentuated" second sound, as it is called, is due either to high blood-pressure in the aorta compared with the left ventricle, or to high blood-pressure in a pulmonary artery compared with the right ventricle. Accordingly it is met with in chronic Bright's disease with high arterial tension, in mitral disease with low intra-ventricular tension, and in pulmonary obstruction with increased tension in the pulmonary artery.

(3) If the tension between the two great vessels of exit from the heart is very different in degree, the pulmonary valves may close before or after those of the aorta, and thus the second sound becomes *reduplicated*. Reduplication of the first sound at the apex undoubtedly occurs, but in a well-marked and unmistakable form is decidedly rare. Apparently it is often

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confounded with a prolonged first sound, or with the first sound and a short presystolic murmur. There are considerable difficulties in explaining it by asynchronism in the contraction of the right and left ventricle; but the most recent experimental inquiries by Professor Roy, of Cambridge, seem to show that this is no impossibility. Clinically its most important significance is as a sign of mitral stenosis. *Intermission* and *irregularity* of the cardiac sounds have been already discussed in connection with the pulse (*supra*, pp. 8, 9).

*Cardiac murmurs.**—We now pass to the *bruits* or abnormal sounds which are added to the first and second sounds. Sometimes they replace them entirely; sometimes one can more or less succeed in distinguishing the normal sound from the murmur which accompanies it.

The recognition of these murmurs was among the firstfruits of Laennec's discovery. He was naturally much struck by their occasional loudness and curious varieties of *quality*, and carefully described several of these varieties by comparing them to well-known noises. The commonest and most characteristic is the bellows murmur or *bruit de souffle*, which exactly answers to the description of a continuous blowing sound, and is not unlike some forms of bronchial or amphoric breathing. Another frequent murmur has a higher pitch and muscular quality, and was named by Laennec the *bruit d'oboe*. A harsh, grating, interrupted murmur he compared to the noise made by a coarse file scraping wood, and named it *bruit de râpe*. Another quality was aptly designated by the term *bruit de scie*, or sawing murmur.

These comparisons, and many others which might be used, are chiefly of use for identifying a particular murmur. They tell us nothing or almost nothing with respect to the condition of the valves.

Nor is the *loudness* of a cardiac murmur of great importance; if distinctly heard, however faint, the message it conveys is the same as if it were loud, and sometimes an extremely loud murmur is of little practical importance. The same murmur will become faint or even disappear when the heart's pulsations are feeble, as in syncope, or first become audible when its beats are rendered quicker and more forcible by movement or by excitement.

We must, however, remember that, in the *healthy heart* when palpitating from mental excitement, or when working under stress—as when a healthy young man runs a race, or a stout and elderly man goes too quickly up a staircase—a murmur may be developed which disappears after a short repose. It is only by repeated examination, and by the help of the pulse and other symptoms, that it is possible to decide between the cardiac lesion which under favourable conditions is without a bruit, and the healthy heart which, when overtaxed, produces one.

The *length* of the murmur is also of no great practical moment. It renders it, however, much more easy to be certain of, for a very short bruit may turn out to be a prolonged first or a reduplicated second sound.

The really important points to be observed in a cardiac murmur, as Skoda seems to have first definitely taught, are its *locality*, and its *rhythm*.

* *Fr.* Bruits anormals.—*Germ.* Herzgeräusche. It is worth mentioning that the French term *bruit* is applied to the natural sounds of the heart; so that what is called a "murmur" in English is always distinguished in French as a *bruit anormal* in general, or as a *bruit de souffle, bruit de râpe, bruit d'oboe*, &c., in particular. *Bruit de souffle* is literally a "blowing murmur;" the ordinary English equivalent, "bellows murmur," is a translation of the French *bruit de soufflet*, a phrase used by Laennec, and stated by Littré to be identical in meaning with the former.

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Locality.—Murmurs produced at the *mitral* valve are not transmitted directly to the surface of the chest unless they are unusually loud. The great depth at which this orifice lies explains the fact. They are conducted by the solid and uniform walls of the left ventricle to the point at which it is pressed against the chest wall in the systole. They are therefore apex-murmurs. Possibly owing to the conducting power of the fifth rib they are audible in the axilla and along the wall of the chest to the angle of the scapula.

Murmurs produced at the *aortic* orifice are audible directly over their seat of origin at the upper border of the third right costal cartilage at its junction with the sternum. If produced in the aorta by narrowing of its orifice, they are conducted by its walls to the chest wall in the second and first right intercostal spaces. If produced in the left ventricle by leaking of the sigmoid valves, they are sometimes confined to the aortic cartilage and the third space immediately below it, but sometimes are conducted by the walls of the left ventricle to the apex, and more frequently down the sternum as far as the ensiform cartilage.

The far less frequent murmurs connected with the right side of the heart are localised as follows. A bruit produced at the *tricuspid* orifice is audible over the sternum between the right and left fourth costal cartilages, and from that point downward to the ensiform cartilage and immediately neighbouring parts of the thorax. A murmur produced at the orifice of the *pulmonary* artery is audible at the junction of the third left costal cartilage with the sternum and between the second intercostal spaces; it is carried upwards and to the left by the wall of the chest, so as to be audible in the second and first intercostal spaces; if produced in the right ventricle by pulmonary leakage, it may be conducted a short distance down the sternum.

Time.—The rhythm of a cardiac murmur determines its connection with the contraction or dilatation of one of the chambers of the heart. A *systolic* murmur is one which coincides with the impulse and the first sound—that is to say, it accompanies the systole of the left and right ventricles. It often extends beyond the period of the normal first sound into the interval between it and the second, but it always begins at the moment of impulse.

A *diastolic* murmur is one which coincides with the second sound, the closure of the aortic and pulmonary valves, and the dilatation of the two ventricles. If it occurs after the second sound and separated from it by an appreciable interval, although it still falls within the period of ventricular diastole which occupies the cardiac interval, yet it is for convenience named not diastolic, but post-diastolic—that is, occurring subsequently to the diastolic or second sound.

Lastly, if a murmur beginning in the cardiac interval is not separated from the first as well as from the second sound, but runs on until it meets the former, it is named a *presystolic* murmur.

Tactile thrill.—The sonorous vibrations which are perceived by the ear are, when very loud, accompanied by a palpable vibration or tactile fremitus, just as we found that loud sonorous rhonchi can be felt as well as heard. This tactile vibration or cardiac thrill when well marked is extremely easy of detection, and gives a sensation to the hand which Laennec aptly compared to the tactile vocal fremitus which is felt when one lays one's hand upon the chest of a purring cat (*frémissement cataire*).

A tactile thrill, like an audible vibration, may obviously be localised to the apex or base of the heart, and its rhythm may coincide with the contrac-

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tion, with the systole, or with the diastole. It most frequently appears accompanying an apical and presystolic bruit.

Theory of cardiac murmurs.—Laennec's notion was that when one of the natural orifices was narrowed the blood rustled in passing through, and it has been generally supposed that the rougher the surface the louder would be the noise. But comparisons taken from water running in contact with air do not apply to the passage of liquids in closed and completely filled tubes. It does not appear experimentally that roughness of surface produces audible vibrations, nor do we clinically find that roughening of the lining of an artery can be detected by the ear. Auditory vibrations when in the vascular system are now believed to depend exclusively upon the formation of a *veine fluide*—that is to say, a narrow stream of liquid passing through an orifice or relatively smaller channel into a more spacious one. It can be imitated by filling a glass tube with water, and then injecting coloured liquid into it through a narrower tube or a constricted portion. The thin coloured stream passes into the wider space at first without mixing, then turns round and forms currents which eddy backwards upon the walls of the larger tube and set up vibrations in these walls, which, if ample enough, may be heard by the ear and felt by the finger. Accordingly every cardiac or vascular murmur may be probably referred to the same physical cause.

The merit of stating this theory in opposition to those of Laennec and his successors belongs to the late Sir Dominic Corrigan of Dublin, who, writing in 1829, says, "When an artery is pressed upon, the motion of the blood in the artery immediately beyond the constricted part is no longer as before. A small stream is now rushing from a narrow orifice into a wider tube, and continuing its way through the surrounding fluid. The rushing of the fluid is combined with a trembling of the artery, and the sensation to the sense of hearing is the *bruit de souffle.*"

The conditions for the production of a fluid vein are demonstrable in almost every case of valvular disease accompanied with a murmur. If one of the cardiac orifices is contracted, the blood is forced through it into a wider space beyond; if one of the valves is rendered inefficient, the blood leaks through the aperture under the pressure which closes the valve into the space which it last quitted. In cases of dilatation of the right or left ventricle, the valves, although perfect, no longer suffice to close the enlarged orifice; leaking occurs with the formation of a fluid vein, and a murmur is again the result.

The chief difficulties in the practical application of this theory are as follows. In some cases, particularly of tricuspid regurgitation in cases of emphysema, we do not hear the systolic murmur we should expect. Again, a murmur is sometimes audible at the apex in cases of hypertrophy from Bright's disease where there is no evidence of regurgitation. Moreover, murmurs heard in the great vessels are not easily brought under the same hypothesis. The ordinary systolic basic bruit audible in the region of the pulmonary artery in cases of marked anæmia is certainly independent of contraction of the orifice or lesion of the valve, and it is rather assumed than proved that, the trunk of the artery being temporarily dilated, the orifice which leads to it becomes relatively narrower, and therefore capable of producing a fluid vein. The well-known *bruit de diable* in the internal jugular vein must be explained by temporary dilatation of part of the vessel and relative narrowness of the part behind. The arterial murmurs which the stethoscope detects in anæmic patients probably depend upon

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compression of the vessel by the instrument, causing a constriction and a temporary fluid vein at that point.

The systolic bruit audible over an aneurysm or in the placenta of a pregnant uterus may be referred to the same cause without straining the anatomical facts. The difficulty is rather to explain why a murmur is so often absent in cases of aneurysm than to explain its presence as the result of a fluid vein.

No other theory of cardiac and vascular murmurs is supported by physical facts, or is nearly so applicable to clinical experience; but it must be confessed that we sometimes meet with difficulties in its application. It is possible that though roughness of the lining surface of a vessel is incapable of producing sonorous vibrations, it may reinforce those produced by a fluid vein; and we still seem to need some further physical explanation of the undoubted fact that in cases of anæmia murmurs are readily produced and are unusually loud.

In any case it appears to be certain that the vibrations which reach our ear are those of the walls of the vessel, and not of the contained blood; that they are excited not at the seat of obstruction or of leakage, but immediately beyond; and that they are transmitted not by the stream of blood, and certainly not in the direction in which it flows more than in the opposite direction, but by the wall of the vessel or of the chamber of the heart which is thrown into vibration.

Having thus described the general pathology, symptoms, and physical signs of valvular disease, we will now notice the special peculiarities which belong to each lesion.

1. *Aortic stenosis with obstruction.*—This as an uncomplicated lesion is one of the most rare. It may occur under two conditions: as a result of rheumatic fever in youth, when, if not complicated by regurgitation, it is probably most often recovered from; and secondly, as a result of valvular atheroma in later life. Beside aortic regurgitation it may then be complicated by more or less extensive atheroma and dilatation of the ascending aorta. The physical sign is a systolic basal murmur propagated towards the right clavicle.

The pulse is small, and, by an exception to most cases of valvular disease, slow. The left ventricle is usually somewhat hypertrophied, but is not apt to dilate. The general symptoms are not severe, but fatal syncope sometimes occurs.

2. *Aortic incompetence with regurgitation.*—This also occurs in two conditions; as a result of rheumatism in young adults (it is rare in children), and as a result of atheroma in elderly people. It is commonly associated with aortic obstruction in both cases, and in the latter with atheroma, dilatation, or fusiform aneurysm of the aortic arch. The physical sign is a basic diastolic bruit replacing and continuing the second sound, often very audible at the ensiform cartilage, and occasionally at the apex.* It is sometimes

* Dr (now Sir) Balthazar Foster suggested an explanation of this aortic apex-bruit in the fact that different cusps suffer in different cases, and that if the left sigmoid is affected, the blood falls directly back into the ventricle and the bruit is heard best at the apex; whereas if the right or posterior sigmoid valve is diseased, the regurgitant blood impinges on the septum, and the bruit is carried down the right side of the heart. Dr Sibson attempted an explanation by supposing that the sinus venosus of the right ventricle is a better conductor of sound than the left ventricle, and so a bruit passes directly through it.

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musical but more often sawing in character, and is often very long and extremely loud.*

The rate of the pulse is usually increased, the artery is large and compressible, the tension low. In systole it fills rapidly, and rapidly subsides again. It is characteristically the quick, short pulse—*pulsus celer;* and is also in systole the strong, forcible pulse, in opposition to the weak pulse which is difficult to feel. It is, however, a weak pulse in diastole, weak in resistance, soft, compressible, the opposite of the hard, cord-like pulse. It exhibits the extreme of difference between systole and diastole, the greatest amplitude of arterial pulsation, the opposite from the characteristic pulse of chronic Bright's disease, in which there is little increase of calibre in systole, and little diminution in diastole. This very striking pulse of aortic regurgitation was recognised as its effect by Corrigan, and is still often named after him. It has also been described as a "locomotive," a "collapsing," and a "water-hammer" pulse.† The strong pulsation in the arteries is due to the reflux into the left ventricle during diastole. The elastic arteries are expanded with the ventricular systole, but instead of the column of blood being sustained by the resistance of the aortic valves, a portion of the contents of the ventricle which were pumped into the arteries returns, so that the blood-pressure is unsustained and falls low in diastole. The characters of the pulse are made more obvious by raising the patient's arm, since gravity then assists the rapidity of the collapse in diastole, and it is probable that not only in the aorta but in its branches for some distance a true reflux occurs; that the flow of blood is not only slackened or brought to a standstill at the moment of collapse, but actually moves back towards the heart.‡

Beside the throb and the collapse which are so striking to the touch, the eye detects the same exaggerated pulsation in the great vessels. As the patient sits before us the throbbing carotids almost tell us the nature of his disease before we feel the pulse or use the stethoscope. The same visible pulsation is seen in the brachial artery at the bend of the elbow. It often enables one to diagnose aortic incompetence when examining the retina, from the obvious pulsation of the arteries seen with the ophthalmoscope. It extends to the smaller arteries and even the capillaries, so that we can sometimes observe a visible flush with each pulsation in the nails of the hand and upon the skin of the forehead, particularly if we cause relaxation of the vaso-motor nerves and injection of the capillaries by gently rubbing the skin.

* The second sound of the heart was ascribed to tension of the aortic valves by Sir Robert Carswell, 1831. In 1832 Dr Billing, in a paper at the Hunterian Society, published in the 'Medico-Chir. Review,' April 1st, 1833, states for the first time in clear language that the first sound is caused by the tension produced in shutting the auriculo-ventricular valves; and the second sound is caused by the tension produced in the shutting of the sigmoid valves. Rouanet came to the same conclusion in the year 1832 ('Analyse des bruits du cœur,' August, 1832). In the same year, 1832, Corrigan discovered the mechanism and diagnosis of aortic regurgitation.

† The comparison is to the suddenness of the fall of a column of water *in vacuo.*

‡ If this is a fact, it explains a sign which is not frequent, but undoubtedly may occur, namely, the presence of a diastolic murmur over the femoral or brachial or carotid arteries in these cases of aortic regurgitation. It is improbable that any cardiac bruit is transmitted by the walls of the arteries for more than a very short distance from its place of origin. A systolic arterial bruit appears to be always caused by compression of the vessel by a stethoscope producing a temporary stenosis, and thus a fluid vein and a diastolic bruit may be explained by the same temporary contraction, if we suppose that in the cases in which it is heard there is an actual reflux in diastole.

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Although the signs just mentioned denote incompetency of the aortic valves, it does not follow that they are diseased, as their want of closure may arise from dilatation of the ascending aorta. For example, a yielding of the sinuses of Valsalva just above the aortic valves has been proved to interfere with their perfect action.

The regurgitant aortic bruit is most frequently combined with an obstructive one. The pulse has then the characters just described as those of the former lesion. There is audible at the base a very characteristic to-and-fro—that is systolic and diastolic—double murmur, one of the loudest, least variable, and easiest to recognise of all physical signs; of the two murmurs the obstructive or systolic is usually the softer and shorter, the regurgitative or diastolic the longer, louder, and higher pitched. It is often audible in every part of the chest, and almost alone among cardiac murmurs can sometimes be heard by the ear at a short distance from the chest, or even, it is said, by a person standing opposite the patient's bed.

The systolic bruit is often carried a considerable distance along the vessels, and sometimes the diastolic also. In the femoral artery, for example, a double bruit may sometimes be heard. If not, it may frequently be produced by using pressure with the stethoscope.*

The symptoms of aortic regurgitation (and of regurgitation with obstruction) are those of arterial anæmia rather than of venous congestion, for so long as a mitral valve is competent there is but little increased pressure in the left auricle and pulmonary veins or in the right side of the heart. Accordingly in uncomplicated cases we find the patient pale but without dropsy, albuminuria, or jaundice. He suffers from headache, and is liable to faintness and giddiness, he has severe dyspnœa on exertion and marked orthopnœa; but while tranquil and under favourable conditions of nutrition he suffers comparatively little. This depends upon what is known as *compensation.*

The ventricle is habitually over-full during diastole, receiving the reflux of blood from the aorta, as well as the natural onward stream through the mitral orifice. It also has the obstruction at the aortic orifice to overcome in systole. This habitual increase of pressure produces dilatation, and also hypertrophy. A moderate amount of the former change is probably beneficial in enabling the heart to accommodate an increased quantity of blood, and by help of the latter the force of the ventricular contraction is increased, so that on the whole the blood-pressure in the arteries is kept up, just as by a more vigorous use of the handle we can obtain a free supply of water from a pump with a damaged valve. The stream of blood through the aortic orifice is diminished in calibre, but its velocity is increased, and thus the amount discharged in a given period of time may be as large as it was before the valve was injured. The compensation is, however, liable to come to an end in more ways than one; first, owing to insufficient supply of food, indigestion, anæmia, or other causes independent of cardiac lesions which interfere with muscular nutrition. The left ventricle suffers perhaps not more than the muscles of limbs, but unlike them it is necessary to the organism, and then comes what appears to be the natural final end of abnormal hypertrophy, wasting and degeneration. It has been supposed that the increased blood-pressure in the left ventricle during diastole which

* Dr Austin Flint, of Philadelphia, maintained that a presystolic murmur may sometimes be heard in cases of aortic regurgitation. He believed it is due to the vibration of the mitral segments even when the valve is inactive, owing to the over-engorgement of the ventricle from the reflux of blood into it from the aorta.

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is the result of aortic regurgitation, would lead to dilatation rather than hypertrophy, and that the increased blood-pressure in the same cavity during systole in cases of aortic obstruction, would lead to hypertrophy rather than to dilatation. This is probably true; but, in the first place, when passive dilatation has taken place, the ventricle will at the end of the cardiac pause be over-full of blood, for it will contain the contents of the left auricle, and the amount which has leaked back into it from the aorta. It will thus have more than its due volume of blood to expel with the next systole, and will thereby be stimulated to hypertrophy. On the other hand, we see from a study of the effects of obstruction in other hollow canals, that dilatation as well as hypertrophy may be the result. When the pylorus is obstructed we find the stomach dilated as well as hypertrophied. Above the stricture of the œsophagus or of the large intestine, there is a dilated pouch with thickened walls, and the bladder in cases of stricture is often found dilated as well as hypertrophied. Moreover, in the case of the heart itself we have an instructive example of the effects of obstruction without regurgitation in cases of chronic Bright's disease. The precise nature of the obstruction in the systemic circulation is, we shall see, still a matter of controversy, but of its existence there is no question. Here, then, we have increased blood-pressure in the aorta, giving the ventricle increased work to empty itself in systole. But the semilunar valves are perfect, and the ventricle is empty during diastole. The result is, as we should expect, hypertrophy of the ventricle walls, and this may continue for an indefinite time; but in certain cases, and perhaps in the majority, if the process is continued long enough the walls begin to yield, and instead of, or in addition to hypertrophy, we find dilatation of the ventricle after death, as well as its appropriate symptoms during the patient's life.

It has been thought that much of the compensatory hypertrophy we have been discussing does not bring increased power to the ventricle, because it is not true muscular hypertrophy, but is accompanied by fibroid or fatty degeneration. This, however, seems to be very doubtful; the most typical cases of fatty degeneration do not occur with hypertrophy of the heart, or with any form of primary cardiac disease, and fibroid degeneration we have seen is also an independent process; hypertrophy of the heart is a true muscular hypertrophy, and the muscle is often found in a perfectly healthy condition. When this is not the case, what we do see is that the muscle is pale, soft, and flabby—looking, in fact, like the skeletal muscles in a patient who has long lain ill. Except in cases of atheroma of the coronary arteries it is probable that the nutrition of the cardiac muscle depends almost entirely upon that of the body generally.

The importance of the condition of the ventricle is obvious from what has been stated, and of late years there has been a disposition to make light of valvular lesions, in comparison with the state of the walls of the heart. But this might easily lead to practical mistakes. A valvular lesion is always a serious thing. It is permanent, and, however good a compensation may be, is always liable to produce its full effects should the compensation at any time fail. On the other hand, hypertrophy or dilatation and fatty degeneration of the heart are very rarely primary conditions, and they derive their importance in most cases from the fact of some antecedent lesion in the valves or elsewhere. The clinical recognition of hypertrophy and dilatation of the left ventricle, as well as of the other cardiac chambers, has been already discussed in the last chapter.

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Mitral disease.—At this orifice we have again the two conditions of obstruction and regurgitation, but, though frequently combined, they are more often met with separately than are the corresponding aortic lesions.

3. *Mitral stenosis with obstruction* may begin in one or more attacks of rheumatic endocarditis, followed by gradual thickening, sclerosis, and contraction of the edges of the opening. The semilunar orifice of the natural valve is converted into a short, straight, narrow slit, to which the not inappropriate name of the button-hole mitral has been given; or the two curtains may cohere, and, yielding to the pressure of blood from the hypertrophied auricle at the end of the cardiac diastole, may project in the form of a fibrous cone into the ventricle. This is called the funnel-shaped mitral.

The degree of obstruction may be roughly measured by the number of fingers which the orifice will admit, or more accurately by inserting graduated cones as far as they will go, or, again, by measuring the edge of the orifice after it has been cut open.

Sometimes a precisely similar contraction of the mitral orifice is found in persons who have never suffered from rheumatism, nor from chorea, scarlet fever, or other probable cause of acute endocarditis, and the whole process seems to be insidious in origin and gradual in progress. A certain proportion of these cases occur in later life, especially in women, and are associated with chronic Bright's disease and atheromatous arteries. They appear, therefore, to belong to the group of degenerative changes which are more often associated with disease of the aortic valves; and this view is confirmed by not infrequently finding calcareous nodules in the thickened fibrous tissue of the mitral curtains. Other cases, again, are met with in young adults and even in children with no history of rheumatic inflammation. Many of these are probably, notwithstanding this, rheumatic in origin; but the synovitis and the fever were, as is usual in children, so slight as to have been forgotten or overlooked. But it is possible that other cases date from before birth. This was the belief of the late Dr Peacock, and it is difficult to disprove; but the extreme rarity of mitral stenosis in *early* childhood makes it improbable.

The result of the narrowing of the mitral orifice is to diminish the supply of blood to the left ventricle, and to increase the blood-pressure in the left auricle. The latter hypertrophies in response to the call, and by a more energetic contraction succeeds in emptying its contents into the ventricle before systole begins. The process of dilatation, however, very soon accompanies that of hypertrophy, and since there are no valves guarding the entrance of the pulmonary veins the increased pressure in the auricle soon begins to tell upon the pulmonary circulation. Ultimately the right side of the heart and the systemic venous circulation may in time become affected. But this is a slow process, and in cases of mitral obstruction uncomplicated with regurgitation only takes place, if at all, after many years.

The physical signs of mitral obstruction are of great clinical and physiological interest. The lesion was recognised by Laennec, and he stated that a rasping murmur, or rather, as he expressed it, "a modification of the natural sound of the heart into a sound like that of a file," is sometimes due to a narrowing of one of the orifices of the heart, and is then much more loud than when it is caused by the heart being too full of blood. In the former case he noticed that one could sometimes feel a thrill, and also that the altered sound of the heart is much prolonged. He supposed both sounds of the heart to be of muscular origin; the one due to contraction of the ventricles, the other

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to that of the auricle. But he had not discovered the means of distinguishing the exact seat and nature of the lesions by means of the locality and the rhythm of the abnormal sound. When the experiments of Hope and Williams, and the clinical investigations of Skoda and Corrigan had laid the foundation of accurate valvular pathology, it was still believed that endocarditis, like pericarditis, produced a sound of itself; while the presence of anæmic and other functional bruits still prevented the mechanism of those produced by valvular lesions from being understood. In 1845 Dr Latham taught that murmurs are distinct from the natural sounds of the heart and differently produced; but instead of ascribing them to auricular contraction, like Laennec, he thought they resulted from unusual vibrations communicated to the particles of the blood by obstacles which it encounters in its passage through the heart. He understood, however, the doctrine of regurgitation, and believed that practically all mitral murmurs were systolic in rhythm and regurgitant in origin.* It was then generally believed that an apex systolic murmur denoted mitral disease, but whether the valve would be found too small or too large was quite uncertain. Dr Hughes, writing in the same year, 1845, states the different mechanism of obstructive and regurgitant murmurs and the methods of diagnosing each clearly and accurately, but he also failed in recognising the application of his rules to mitral obstruction. Those who more boldly carried out what they were taught naturally arrived at the conclusion that mitral stenosis would produce an obstructive murmur, apical in position and diastolic in rhythm; but this was seldom or never recognised by the bedside, and seems to have been admitted for the sake of symmetry. Practically, physicians recognised only one mitral murmur, systolic in rhythm, and produced by either obstruction or regurgitation. In 1845, Skoda stated that with narrowing of the mitral valve the second sound is replaced by a *long* murmur; but, in 1843, Fauvel had shown that the characteristic sign of mitral stenosis is an apical murmur which is neither systolic nor diastolic, but, adopting a phrase previously used in another sense by Gendrin, presystolic in rhythm—that is to say, it is not separated from the first sound by any interval, but, beginning during the cardiac pause, it runs up to and unites with the first sound and the impulse. It therefore occurs during the ventricular diastole, but at the end of that period, and coincides with the auricular systole which immediately precedes that of the ventricle.

Apart from its characteristic rhythm, the presystolic mitral murmur is remarkably loud, harsh, and often grinding in quality, rarely blowing or muscular. Moreover, it becomes louder as it goes on, and is suddenly cut short at its loudest by the first sound and impulse. Again, its locality is very limited, so that one is surprised to hear so very loud a sound, only audible in the immediate neighbourhood of the apex-beat. Lastly, it is accompanied by an accentuated second sound at the base, and not infrequently by the tactile fremitus which we have seen that Laennec recognised and named *frémissement cataire.*† This apical cardiac thrill, like the murmur which it accompanies, is presystolic in rhythm and ends in the impulse.

The explanation of the murmur is that the hypertrophied left auricle, in

* "The cases are so rare in which either the diastolic murmur alone, or the diastolic and systolic murmurs together, can be fairly imputed to the mitral valve, that they are a sort of clinical curiosity" ('Lect.,' vol. i, p. 38, 1845).

† "Quand (l'orifice rétrécie) est à gauche, on sent quelquefois à la main un frémissement analogue à celui qui accompagne le murmure de satisfaction que font entendre les chats lorsqu'on leur passe la main sur le dos" (Laennec, 'Ausc. Méd.,' tom. ii, § 634).

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forcing the last portion of its contents through the narrowed orifice into the ventricle, furnishes a fluid vein of sufficient velocity to produce audible and tactile vibrations of the ventricular walls, which are heard and felt at the point where it touches the surface of the chest.*

No cardiac murmur is more easy to recognise than the presystolic bruit of mitral contraction when these characters are present. Nevertheless the diagnosis of this lesion is often difficult, and for the following reasons:

First, it is often accompanied by mitral regurgitation; and a presystolic bruit ending, not in a first sound and interval, but in a systolic murmur, becomes difficult of detection, and is often taken for a long regurgitant bruit alone. One must try to recognise the difference of quality of the former and latter parts of this continuous murmur and to fix its precise rhythm by placing the finger upon the apex-beat, or by first applying the stethoscope so lightly that we only feel the impulse without hearing the sounds, and then increasing the pressure until it conducts the murmurs which then fall into their proper place, the one before and the other after the impulse. One must never attempt to get the time by feeling the radial pulse, for this may be delayed. The carotid may be used for the purpose more safely; but the direct palpation of the heart by the finger or the ear, as just recommended, is the safest and easiest plan.

Secondly, the presystolic murmur is often absent in cases of undoubted mitral stenosis and where it has been heard distinctly a short time before. On a single occasion the lesion of the valve may therefore pardonably be overlooked. Sometimes, however, one can feel a presystolic thrill when one cannot hear a presystolic bruit, or the accentuated second sound may help us, or the apical reduplication of the first sound; and our conjecture will be all the more probable if the patient is a female and if there is no history of rheumatic fever.

In not a few cases of mitral stenosis, instead of the characteristic, rough, long, and ingravescent *crescendo* presystolic murmur, a sound is heard which follows the second after a short interval and ceases before the first.† It is audible at the apex and *diastolic* in rhythm, *i. e.* it occurs during the diastole of the ventricle. But it differs from the presystolic murmur in that it does not run up into the first sound and impulse; beginning after the second (or diastolic) sound it occupies the period of ventricular pause,

* The above explanation of the presystolic murmur is that offered by Dr Gairdner in 1861 ('Edin. Med. Journ.,' vol. vii, p. 438), and is generally accepted in this country and abroad. It was ably defended by Dr Fagge in his paper in the 'Guy's Reports' for 1871, and also in his article in 'Reynolds' System of Medicine.' It is, however, right to mention that the otherwise unsupported opinion of the late Dr Barclay, that the murmur is produced by the ventricular systole, has been revived in an elaborate article by Dr F. C. Turner in the 'St Thomas's Hospital Reports' for 1876; and still more recently ('Lancet,' 1889) by Dr Dickinson with characteristic skill. The decision rests partly on physiological considerations, and partly on one's own auditory perceptions; and on both grounds the writer adopts the conclusions of Fauvel, Traube, and Gairdner. Whatever its physiological explanation, no one doubts that this famous bruit indicates mitral contraction. The recognition of a presystolic murmur as a sign of mitral stenosis is, as shown by the author in the paper above mentioned, undoubtedly due to Fauvel, whose article appeared in the 'Archives Générales' in 1843.

† It is sometimes called "*post-diastolic*," because it is heard, not *with* or replacing the second (diastolic) sound, but *after* it. It is well to note that the term "diastolic" may be applied in three senses: to the period of diastole of the ventricle, to the diastole of the auricle, or to the second sound.

Hope, writing in 1832, says, "When the mitral valve is contracted, a murmur accompanies and sometimes entirely supersedes the second sound, being occasioned by the obstructed passage of the blood from the auricle into the ventricle during the diastole of the latter."

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and ceases before the first sound, from which it is separated by an appreciable interval.

The probable explanation is that in these cases the left auricle, being well hypertrophied, succeeds in emptying its contents through its narrowed outlet before the ventricle contracts, so that, as in health, there is a short interval between the auricular and ventricular contraction. When the bruit is presystolic, the auricle does not succeed in expelling its contents before the ventricular contraction at once reverses the direction of the inflowing blood-stream, and ends the obstructive murmur by the first sound or by a regurgitant systolic murmur.*

Whatever is the explanation, this diastolic, or post-diastolic apex-bruit, when associated with other signs of mitral stenosis, is a sure evidence of that lesion. The difficulty is to distinguish it from a diastolic aortic règurgitant bruit, when exceptionally most audible at the apex. The character of the latter bruit, which is usually less harsh and more blowing or musical, the evidence of dilatation of the left ventricle, and the peculiar character of the pulse are the best marks of distinction from that of mitral obstruction.

Another frequent sign of mitral stenosis is *reduplication of the sounds* of the heart. It is obvious that if the two sides of the heart did not act together, four sounds might be heard instead of two. If, however, this want of synchronism occurred, it is still doubtful whether the first two sounds could be completely separated; so that the double sound is probably due to the second being heard twice. The heart's action is thrown out of gear by the hypertrophy of the left auricle, and by the increased pressure on the right side of the heart. Under these circumstances, the two ventricles not acting together, the aorta and pulmonary artery are thrown out of order, and a double sound is produced by the closure of the sigmoids.† This double sound, or, combined with the first, this triple sound of the heart, is sometimes heard under other conditions of impediment to the circulation, but it is most common in the case of mitral stenosis. It is called in France by Bouillaud's name, *bruit de rappel*, and is like a hammer rebounding on the anvil.

The contraction of the auricle is sometimes not equal to the production of a bruit in stenosis, and then the case is characterised by the almost total *absence of the first sound.* This may arise from weakness of the auricle or from less pressure being exerted on it from behind. A thrill might still be felt, since a less number of vibrations than would produce a sound might be palpable to the hand.

The effect of mitral stenosis on the heart will naturally be to produce dilatation and hypertrophy of the left auricle (and ultimately of the right

* Dr Humphry Rolleston has published a valuable criticism of this explanation, and his objections are not without force; but those against any view which ascribes the diastolic apex-murmur to contraction of the ventricle are still more cogent. It is very difficult to say beforehand what derangement of mechanism may take place when the delicately-adjusted normal relations of blood-pressure in the cavities of the heart are altered. It certainly appeared unlikely that the right ventricle should beat asynchronously with the left, but Dr Roy's recent observations seem to establish this as a fact. That the hypertrophied left auricle drives the blood through the narrowed orifice with increased velocity, that the auricular systole will be prolonged in time, and that it must precede and be ended by the ventricular systole, seems to be certain.

† Dr George Johnson has suggested that one of these sounds may be not valvular, but due to the auricular systole. Dr Sansom thinks that it may occur from a tension of the mitral valve at the time when the blood is thrown back during the diastole of the ventricle and that therefore it is not a normal second sound.

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side), while the left ventricle has less rather than increased work, and either continues of normal size, or may become somewhat atrophied, apart from comparison with the rest of the heart.

The pulse is generally, in uncomplicated cases, normal, but sometimes the volume is small and the beat feeble. As in most cases of valvular disease, it is more or less accelerated in time. If it is markedly compressible or irregular, this is probably due to concomitant mitral regurgitation.

The general symptoms are those of palpitation and dyspnœa without anasarca. The only one which is at all characteristic is hæmoptysis, which is certainly more frequent with uncomplicated mitral obstruction than with mitral regurgitation or aortic disease.

4. *Mitral incompetence* is the most frequent of all valvular lesions, but unlike the last three it is not always of organic origin. Hence we must distinguish (*a*) temporary and functional incompetence; (*b*) primary organic incompetence; and (*c*) incompetence associated with mitral stenosis or aortic regurgitation.

(*a*) Functional incompetence appears to depend upon the fact that the auriculo-ventricular orifice is not like that of the aorta or fibrous ring, but is formed by the muscular lip of the ventricular cup. These fibres, as Ludwig has shown, contract during systole so as to diminish the calibre of the opening. If this is not the case the curtains do not suffice to close it, and regurgitation ensues.* Want of due contraction of this muscular ring is probably the cause of mitral regurgitation in many conditions, when a temporary systolic apical bruit is heard. It is, however, from the nature of the case impossible to prove this, and there are some difficulties which must be recognised. One is that these functional apex-murmurs are not audible in the axilla, nor at the angle of the left scapula; whereas the physical conditions for the production and conveyance of a fluid vein would seem to be the same, whether the leakage is caused by the curtains being too small or by the orifice being too large. Again, an apical systolic murmur is sometimes heard in cases of chronic Bright's disease, not only when there is dilatation of the left ventricle, but when there is pure hypertrophy without dilatation and with unaffected valves. If we find an apical bruit in cases of anæmia along with a systolic basic bruit, we attribute the latter to the pulmonary artery and the former to dilatation of the mitral valve; but why such dilatation should sometimes occur and more often be absent in severe cases of anæmia is hard to explain. The apical systolic murmur of chorea is generally considered to be functional in character, and if so its mode of production is obscure (cf. i, 731).

A systolic apex-murmur is believed by some authorities to be sometimes pericardial in origin, a true friction-sound, but it is sometimes audible in cases of adherent pericardium where there is no valvular lesion to be found after death. The difficulty of explaining these apical murmurs is increased by the obscurity which still rests upon the nature of the normal first sound of the heart. If it is due, as taught by Billing and some other physiologists since, to tension of the mitral and tricuspid curtains, we may strictly apply the theory of the fluid vein to explain systolic murmurs. If, as Laennec supposed, and as some good authorities still believe, it is due to contraction of the ventricular muscle, its length and quality may be sup-

* If a ligature is fastened round the arterio-ventricular groove in a living animal, it slackens with each systole, as the writer has found in repeating Ludwig's experiment.

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posed to be variously modified with or without regurgitation. If, as seems most probable, it consists of two elements (the valvular tension which exactly corresponds to that of the sigmoid valves in the second sound, and a *bruit musculaire*, the addition of which gives the specific character distinguishing the first sound from the second), we then have greater scope for explanation of the various systolic apical murmurs—but also greater difficulty in determining the true explanation in each case.

It has often been supposed that a temporary or functional murmur may be produced by failure or irregularity in the action of the musculi papillares. Addison, for instance, suggested a spasmodic disorder of these muscles as an explanation of the choreic bruit, but we have no evidence of the occurrence of such a phenomenon; and in cases of extremely irregular cardiac action no murmur is to be heard. It must be added that none of the older explanations of either functional or organic murmurs which referred them to changes in the blood itself are compatible with physical facts, and though their admission would explain some difficulties, it would introduce many more. Apart from temporary imperfection in the contraction of the ventricle, there may be permanent muscular dilatation which will cause leaking at the mitral valve, the curtains being healthy. This occurs sometimes as a primary dilatation with more or less hypertrophy, such as was described in the last chapter, more frequently as a secondary result of increased blood-pressure in the left ventricle, consequent upon primary disease of the aortic valves or more distal obstruction in the systemic arteries, as in chronic Bright's disease. It may also occur as a more rapid process after scarlatina and other febrile disorders, particularly in children. This permanent dilatation with the consequent incompetence is marked by a systolic, apical bruit audible in the axilla, and leads to all the symptoms to be presently noted which follow the primary, organic lesion of the mitral valve. Hence it is often extremely difficult in an advanced case of aortic regurgitation, or of chronic Bright's disease, with mitral symptoms superadded, to say whether the latter are due to independent structural changes in the valve or merely to consecutive dilatation. Practically the question is of minor importance.

(*b*) We now come to the primary organic disease of the mitral valve which leads to regurgitation. Like other valvular lesions, it may depend upon acute, infective, or ulcerative endocarditis, upon subacute rheumatic inflammation, or upon chronic sclerosis with atheroma. In the first case the symptoms and prognosis are those of the general disorder, and have been already discussed (p. 56). The third condition rarely leads to uncomplicated regurgitation; if it affects the mitral valve at all, it produces stenosis. Mitral imperfection is by far the most frequent result of rheumatic endocarditis. It is most common in children, but may exist at any age.

Its first sign seems to be the appearance, on the auricular surface of the curtains and close to their edge, of a slight ridge of fibrinous exudation, often continuous, but often in separate nodules, which are arranged with remarkable regularity, forming a series of minute granules of equal size running along the opposite edges of the two curtains. It is somewhat difficult to understand how at this stage regurgitation is produced, but the presence of a typical regurgitant murmur leaves no doubt of the fact. Somewhat later the whole surface of the valves on the ventricular as well as the auricular aspect, and also the chordæ tendineæ become affected with the same plastic

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or serous inflammation. The inflammation may clear up and leave the valves uninjured, or only somewhat thicker and more opaque than before, like the pleura after recovery from pleurisy; but more often contraction takes place, the delicate curtains are shortened, or puckered, or folded, and the chordæ tendineæ become shortened and adherent or matted into a single deformed mass. Perforation never occurs except from septic inflammation supervening, but the valves often become so shrunken and tethered that they are as incompetent as if destroyed by ulceration.

The physical sign of mitral regurgitation is a bellows-murmur, systolic in rhythm, apical in locality, and audible in the axilla and at the angle of the left scapula.

With reference to the locality of the regurgitant mitral bruit, it does not appear that any sound produced in the heart is "propagated in the direction of the blood-stream," for sonorous vibrations do not necessarily travel in the direction of the current of translation. The direct or regurgitant murmurs are produced, not at the narrow passage, but beyond it, where the "fluid vein" is formed. Accordingly, a direct mitral bruit having its origin in the ventricle is propagated to the chest wall, where the ventricle strikes it, *i. e.* at the apex: a regurgitant mitral bruit being formed in the left auricle is conducted by the auriculo-ventricular continuity to the apex, and, by the excellent conducting power of the ribs, along the axilla; a direct aortic bruit originating in the first segment of the aorta is conducted by its walls to the part of the chest wall nearest to it, *i. e.* to the third right rib, and upwards towards the right clavicle; and a regurgitant aortic bruit being formed just beyond the valves is usually best heard where the sounding-board formed by the sternum receives and conducts it. A direct pulmonary murmur, being formed in the pulmonary artery, is conducted, not by the stream of blood, but by the solid walls of that vessel, in the direction in which it passes upwards and to the left. A regurgitant tricuspid murmur is not favourably situated for conduction, being formed in the right auricle, and is best heard either immediately over its place of origin or where the left auricular appendix comes in contact with the chest wall.

The pulse is characteristic: compressible, frequent, and irregular; not small and not dicrotic. Intermission is not common, and grouped beats are quite exceptional. It is the most irregular of all irregular pulses.

The second sound is *accentuated*, *i. e.* louder and "brighter" than usual, owing to the increased blood-pressure in the pulmonary artery.

(*c*) Frequent as mitral incompetence is as a primary uncomplicated lesion, its frequency is much increased by the fact that, when the valves are thickened and the orifice contracted, their accurate adjustment during systole is often impossible. Hence a large proportion of cases of stenosis are sooner or later complicated by mitral regurgitation. When this is the case, as when mitral regurgitation is consecutive to disease of the aortic valves, the clinical features of the case assume the character which belongs to cases of primary mitral incompetence.

These are, speaking generally, those common to advanced cardiac disease as described above (pp. 59—62). There is dyspnœa and orthopnœa, though the latter is usually less severe than in aortic cases; there is anæmia, but often modified by venous congestion of the lips, cheeks, chin, nose, and ears, and sometimes by an icteric tinge due to congestion of the liver. Hæmoptysis is probably less common than in either mitral stenosis or

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aortic disease. Venous congestion with dropsy is the most characteristic symptom, while arterial anæmia with syncope and liability to sudden death is less marked than in aortic cases.

The left auricle, being habitually over-full, dilates with but little hypertrophy. The pulmonary veins share in the change, and each pulsation of the left ventricle is felt in the pulmonary capillaries. The lungs become congested, the blood-pressure in the pulmonary artery rises, and the right ventricle finds difficulty in expelling its contents. As a result, it hypertrophies to some extent, and dilates much more; its walls also become dense and tough. This may be the effect of hypertrophy, but it is perhaps partly due to congestion of the muscular tissue, due to the pulmonary circulation sharing in the general venous congestion. The tricuspid valve probably yields as soon as the ventricle begins to dilate. Its safety-valve action comes at once into play, no doubt with relief to the heart; but the arrangement which is beneficial under the temporary congestion of the right side, which occurs during exertion in a normal heart, is now apt to be permanent. The right auricle dilates as much as the left, and in chronic cases often more, since the increasing obstruction in the pulmonary capillaries leads to comparative diminution of pressure in the left ventricle and auricle.

The unguarded openings of the two cavæ allow the congestion of the right side of the heart to be at once transmitted to the systemic veins; each contraction of the right ventricle produces a wave which passes straight into the two cavæ and their branches, not only checking but reversing the natural current of the blood. This is seen in the external jugular veins, which become dilated and pulsate with each beat of the heart, an important sign of tricuspid regurgitation. This can be easily distinguished from the normal or exaggerated pulsation, which accompanies inspiration, by its following not the pulmonary but the cardiac rhythm.*

The face and neck are generally preserved from dropsy, owing no doubt to the great assistance to the venous circulation afforded by gravity; but the arms are often found swollen, particularly over the dorsum of the hand, and the right or left limb will suffer according to the patient's position in bed.

The inferior cava is more rapidly and severely affected than the superior. The valveless hepatic veins which join it just before it enters the right auricle suffer immediately; the centre of each hepatic lobule becomes congested, the liver enlarges and is usually tender to the touch, and after a time biliary excretion is hindered, and a more or less decided jaundiced tint appears in the eyes and the skin; occasionally the hepatic channels become so dilated that the whole organ palpably throbs with each pulsation of the right ventricle, by the same mechanism, as was just noticed in the case of the external jugular vein (see a paper by Dr F. Taylor, 'Guy's Hosp. Rep.,' 1875, p. 377). Even before there are symptoms of hepatic congestion the stomach begins to suffer, dyspepsia and distressing flatulence greatly adding to the disturbance of the heart and the distress of the patient. After death the mucous membrane of the stomach is found swollen, deep red, often with minute ecchymoses, and covered with tenacious mucus. The intestines seem to be the least affected in the portal region; but the spleen from con-

* Dilatation and pulsation of the internal jugular veins was recognised by Lancisi as a sign of dilatation ("aneurysm") of the right ventricle. Corvisart disbelieved this statement, but Laennec reasserted it ('Ausc. Méd.,' ii, 263–4).

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tinued congestion becomes (not swollen, as in cases of malaria, but) dense, dark coloured, and crisp to the knife.

The kidneys show their congestion by secreting scanty, high-coloured urine with raised specific gravity and an abundant deposit of lithates. Albumen is often present, but hæmaturia from cardiac disease of any kind is decidedly rare, more so than hæmoptysis or hæmatemesis. Hæmorrhoids if present are aggravated, but this is a less conspicuous feature of disease of the heart than it is of cirrhosis of the liver. The congestion of the lower extremities leads to anasarca, which begins about the ankles and dorsum of the foot, and spreads backward to the thighs. It is remarkable that the scrotum almost always escapes. Ascites is almost constant among the later symptoms of cardiac dropsy, but is seldom so large as in cases of hepatic obstruction. Some amount of pleural and pericardial effusion is almost always found after death, but there is seldom enough to be detected during life, and still more rarely to call for treatment.

Valvular affections of the right side.—These are comparatively rare and unimportant.

5. *Pulmonary stenosis with obstruction.**—This is the least rare of primary organic lesions of the right side. It is almost always congenital, though due in most cases to intra-uterine endocarditis, and will be described with other malformations.

When, however, we hear a systolic basal murmur traceable upwards and to the left—that is to say, in the second and first left intercostal spaces—it very seldom indicates congenital dextro-sigmoid stenosis. The murmur is temporary, not permanent, and is unaccompanied by signs of dilatation of the right side of the heart or of cyanosis. Occasionally it is heard in cases of phthisis, when it is probably produced by slight traction or pressure on the pulmonary trunk by pleural adhesions, and intensified by solidification of the lung. Much more frequently this systolic pulmonary murmur is heard in cases of extreme anæmia, in chlorosis frequently, in the anæmia of rheumatic fever and of Bright's disease occasionally; in some cases of phthisis, and frequently in cases of Addison's idiopathic anæmia, of leuchæmia, and of Hodgkin's disease. A similar murmur is occasionally audible in the aortic area, and the arterial bruit heard in the great vessels of the neck, in the abdominal aorta, and wherever a superficial artery can be pressed by the stethoscope in anæmic subjects, is clinically if not physically related. The explanation above given (p. 66), by which anæmic murmurs are reduced to examples of a fluid vein, though not completely satisfactory, is more so than any other that is offered. The quality of the anæmic pulmonary bruit is sometimes soft and blowing, but now and then decidedly harsh. In children a dextro-sigmoid systolic bruit may sometimes be heard, which is produced by the pressure of the stethoscope on the yielding parietes of the chest.

* It is unfortunate that we have no word for the valves which guard the entrance to either the pulmonary artery or the aorta. The adjective pulmonary is naturally taken to apply to the lungs rather than to their artery or its valves, and the adjective aortic rather to the trunk of the great artery than to its valves. Some years ago the writer proposed to appropriate the terms semilunar and sigmoid by an arbitrary convention to the aortic and pulmonary valves respectively. Dr McAlister, of Cambridge, has suggested the words dextro- and lævo-sigmoid for the same purpose. Some such addition to our nomenclature would be of much practical value, but it seems almost hopeless to introduce changes of this kind, however much needed.

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6. *Pulmonary or dextro-sigmoid imperfection* is the rarest of all valvular lesions. The only cases seen by the writer are those in which ulcerative endocarditis has affected the right as well as the left side of the heart. Its sign would be a diastolic basal murmur, traceable down the sternum from the third left costal cartilage.

7. *Tricuspid stenosis* with obstruction of the passage of blood from the cavæ and right auricle to the right ventricle and lungs is a rare condition, and usually, when it occurs, is a complication of mitral stenosis.

Dr Bedford Fenwick has collected forty-six cases of tricuspid contraction, of which the majority occurred in young women and only five in men ('Path. Trans.,' 1881, p. 48). In half there was no rheumatic history giving a clue to their origin. Every case was accompanied by a corresponding condition of the mitral orifice.

The physical sign of tricuspid stenosis would be a presystolic murmur audible at mid-sternum between the fifth costal cartilages, and traceable as far as the ensiform cartilage.

8. *Tricuspid incompetence with regurgitation.*—This is extremely common either as a normal or almost normal adaptation to relieve temporary pressure on the right side of the heart. It probably occurs in every healthy man who runs at his top speed for 100 yards, or who dives, and continues as long as he can under water.*

It is also the probable explanation of the temporary systolic murmur which is frequently heard when apparently healthy persons have hurried upstairs to be examined for assurance; or in young men who fear that they have disease of the heart, and are naturally excited under examination. This murmur always goes with accelerated pulse, and often with irregularity and palpitation of the heart. It speedily disappears after the excitement is over.

More permanent tricuspid regurgitation is undoubtedly present as above described in chronic cases of mitral incompetence, and also in those of dilatation of the right side of the heart, which are consecutive to chronic bronchitis with emphysema; for in all such cases there is not only general venous congestion and dropsy, but after death the tricuspid orifice is found dilated, and the valves incompetent. But it is remarkable how seldom we can identify a bruit as the result of this regurgitation. In primary cardiac cases it may probably often be masked by an aortic or mitral murmur, and in dropsy and cyanosis of pulmonary origin rapid breathing and bronchial rhonchi may obscure it, or emphysematous lungs may prevent its reaching the ear. Nevertheless, its rarity remains a difficulty. Possibly when there is decided dilatation of the tricuspid orifice the leak is so wide that it nearly approximates to the dimensions of the auricle itself, and thus the conditions of a fluid vein would not be fulfilled. This explanation would agree with the much less frequent, but still undoubted absence of a mitral murmur in some cases of regurgitation, and with the constancy of a diastolic bruit in those of aortic regurgitation, where the difference in calibre between the largest gap in the sigmoid valves and the left ventricle in diastole must be far greater.

If carefully sought for, we may occasionally detect a tricuspid bruit supervening in a case of chronic mitral disease; and its presence is sometimes recognised in the later stages of bronchitis with emphysema, of

* For the explanation of this mechanism see the late Mr Wilkinson King's papers on the "Safety-valve Functions of the Tricuspid, or the Moderator Band of the Right Ventricle," 'Guy's Hosp. Reports,' vols. ii and vi of first series.

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cirrhosis of the lung, or of extremely chronic fibroid phthisis. Its characters are systolic rhythm, and a limited seat in the mid-sternum or over the ensiform cartilage. Its quality is usually that of a bellows murmur.

Frequency of single and combined valvular lesions of the heart.—The relative *frequency* of the several valvular lesions of the heart is not easy to determine: partly because during life it is often a question whether a murmur is due to functional or organic disease; partly because, even after death, there may be a question as to the perfect competence of a valve; and partly because there are so frequently combinations of more than one valvular lesion. Clinically, an apex systolic bruit is undoubtedly the most frequent; a to-and-fro basic bruit comes next, and a presystolic murmur third.

Mitral regurgitation, therefore, is by far the most common form of heart disease, as judged by physical signs; but mitral stenosis is decidedly more frequent in adults as shown in the deadhouse, and probably before adult life the same is true after the seventh and eighth year. Mitral is more common than aortic disease in women, and mitral stenosis more common in women than in men.

Aortic lesions rarely follow mitral, but usually end with mitral regurgitation. Mitral stenosis clinically precedes regurgitation; but, as Dr Goodhart suggests, it is possible that in childhood a puckered and leaking valve may gradually thicken and contract. Tricuspid stenosis is very rare except as a complication of the same condition of the mitral valve. Aortic and mitral regurgitation often go together; aortic stenosis is seldom seen without aortic regurgitation; and mitral stenosis, if the case goes on to its full term, is sooner or later complicated by mitral regurgitation.

From the *post-mortem* records of Guy's Hospital, Dr Shaw has ascertained that out of 147 cases the mitral valve alone was diseased in 41, the aortic alone in 26, and both in 69. On the right side there were 14 cases of tricuspid stenosis, 7 of its dilatation and thickening, and 5 of disease of the pulmonary valves. In only 3 cases were there lesions on the right side without the left being also affected.

Prognosis.—According to Dr Wilks, "as regards the relative gravity of the valvular diseases, probably aortic obstruction is the least serious; next to this, stenosis of the mitral valve, which with a compensating hypertrophied auricle may endure for years. Regurgitant diseases are far more serious, but mitral regurgitation is less so than aortic, for the latter condition often leads to sudden death."

This opinion is corroborated by Peacock and by Bristowe, but there are authors who regard mitral stenosis as more serious than mitral incompetence.

Undoubtedly the gravest of all valvular lesions (excluding those complicated by acute ulceration, "malignant" endocarditis, which proves fatal not so much by its mechanical effects as by its poisonous character) is aortic incompetence with dilatation of the left ventricle. But well-compensated aortic leaking may go on with care for years, and when mitral dilatation and incompetence become added, the danger is rather diminished than increased.

The least grave single valvular lesion is aortic stenosis, with moderate

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compensating hypertrophy. Still, with inelastic arteries, these cases are occasionally cut short by sudden and fatal syncope.

Intermediate in danger between the two forms of aortic valvular diseases are mitral obstruction and regurgitation. The former alone is more often met with accidentally, without symptoms; and in all likelihood will last longer. The latter alone causes severe symptoms, but of all valvular lesions is most efficiently relieved by appropriate treatment. The most common mitral cases are those of stenosis with (usually secondary) regurgitation, and these are the gravest; next to cases of aortic incompetence they offer the shortest prospect of continued life. The danger of sudden death is greater in uncomplicated mitral stenosis than when there is regurgitation either primary or secondary.

Other considerations, however, modify the prognosis derived from the anatomical character of the valvular lesion. One is that of age, another is mode of life as to exposure, intemperance, and overwork. A rheumatic lesion is, other things being equal, of better outlook than one of atheromatous origin, partly because the former occurs in younger, the latter in older patients. Another important consideration is the state of nutrition of the cardiac walls. A healthy hypertrophied auricle will often overcome the impediment of a narrowed left ostium; and a healthy hypertrophied ventricle will compensate a narrowed or even a leaking aortic valve. But when anæmia, fatty degeneration, and dilatation befall the muscle, then the hydraulic effects of the valvular defect become apparent and too often ingravescent.

Of lesions of the right side, pulmonary stenosis is almost always a congenital defect; it leads to cyanosis, and life is rarely protracted beyond childhood, except in the slighter forms of the disease, and even then the patients usually die as young adults from phthisis. Occasionally, however, they may live to be fifty years old. Tricuspid stenosis is almost always a concomitant of the same change in the mitral orifice, and probably, if recognised during life, it ought not to add to the gravity of the prognosis. Tricuspid regurgitation is very common, but only as the result either of pulmonary obstruction or of primary valvular lesions on the left side of the heart. It is probably a beneficial event, by allowing the high blood-pressure to be distributed through the systemic veins and relieved by anasarca.

It is only in the latter stage of aortic valvular disease that dropsy and general venous congestion with albuminuria and jaundice occur, when the left ventricle and the mitral valve have yielded and become dilated. Hæmoptysis and epistaxis belong particularly to its early stages. Severe cardiac pain, urgent dyspnœa with orthopnœa, excessive palpitation, are miseries which also attend aortic incompetence. Hæmoptysis is more common from mitral stenosis than from mitral regurgitation.

Syncope and sudden death are usually and justly associated with aortic rather than mitral disease. In mitral cases sudden death occurs more often with obstruction than with regurgitation. Probably atheroma of the aortic orifice, with considerable stenosis and incompetence, is the form of valvular disease which most often leads to absolutely sudden death. Bad cases of aortic regurgitation occurring in younger patients as the result of rheumatism produce comparatively slight symptoms while well compensated; but when dilatation occurs, the mitral valve gives, pulmonary congestion and dropsy soon follow, and the case becomes a mixed one: in such cases death may

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still supervene by terribly sudden syncope, but there has been ample warning beforehand.

With regard to *duration* of valvular disease, judging by the patient's age at the time of death, Dr Shaw finds that of 95 fatal *aortic* cases at Guy's Hospital (26 without and 69 with concomitant mitral lesions) 1 died under ten years old, 14 between ten and twenty, 40 between twenty and forty, 33 between forty and sixty, and 7 above sixty. Of 41 fatal *mitral* cases, none died under ten, 6 between ten and twenty, 14 between twenty and forty, 20 between forty and sixty, and only 1 above sixty. The general experience is that the duration of aortic disease is considerably less than that of mitral, *i. e.* the time of death is earlier.

Taking clinical instead of pathological data, he finds that among 80 patients (excluding cases of ulcerative endocarditis), the shortest period which elapsed between the appearance of marked cardiac symptoms and death was in *aortic* cases two weeks, in *mitral* four. The average period (not very important, since it may be so much influenced by a single exceptional case) was only two and a half years in "mitral" cases in general, one year and a half in aortic cases with mitral regurgitation also, and as much as four and a half years in simple, *i. e.* well-compensated aortic cases. In cases of mitral stenosis without symptoms or sign of regurgitation, the average duration was three and a half years.

In the most favourable form of valvular disease, aortic stenosis, Peacock records a case exhibiting the most extreme degree of obstruction in which compensation was so perfect that the patient died at seventy-five after the operation for strangulated hernia. With aortic regurgitation the same accurate observer records as the longest period of survival after the existence of the lesion was ascertained, five or perhaps seven years. This period, however, is often happily exceeded.

With respect to *sudden death*, Dr Shaw finds that there were 34 cases in the hospital in the ten years 1875–84, from all causes, excepting injury and hæmorrhage. Of these, in 14 the cardiac valves were found normal, in 20 they were diseased; the aortic in 13 and the mitral in 7, and in all these seven cases the left ostium was *constricted*. Of the above 14 cases with healthy valves, the cardiac muscle was degenerated in 11, viz. fatty in 8 and fibroid in 3.

So far as prognosis is related to the *cause* of the cardiac lesion, it is better in rheumatic than in atheromatous cases. The most favourable cases, apart from the nature and extent of the lesion, are those in young subjects whose hearts have been damaged by rheumatic endocarditis, but whose nutrition and renal depuration are vigorous.* The worst cases are those of ingravescent atheroma with confirmed gout and granular kidneys.

As to *sex*, the prognosis is better for women than for men, because in the majority of cases their lives are easier and more tranquil.

Apart from the nature of the lesion, the most important element of prognosis is the kind of life which the patient is able and willing to live. Hard work and exposure, dissipation, starvation, and drink bring otherwise favourable cases to an early fatal result. Moderation in all things, a gentle life, an equable temper, and exemption from attacks of rheumatism, from

* There is in early life a special power of repair and self-adjustment in the heart which warrants our expressing a more cheerful prognosis than would be justifiable in cases of cardiac disease occurring in a grown person (West). (In children,) as long as the cardiac lesion gives rise to no symptoms the prognosis is very favourable (Eustace Smith).

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bronchial catarrh and pneumonia, from muscular strains, and from the excitements of passion, are the conditions which prolong otherwise unfavourable cases to advanced age, and that not unfrequently after a far from useless or unhappy life.

On the whole, the hospital student will find that in private practice he will succeed far better with the same treatment than he could anticipate from his previous experience. Sir Andrew Clark, Professor Gairdner, and others have lately published remarkable facts as to the long duration of life in cases not only of cardiac murmurs, but with conclusive symptoms of organic disease of the heart ('Brit. Med. Journ.,' Feb. 5th and 12th, 1887).

The two following cases have come under the writer's observation:

An old gentleman, the subject of atheroma of the left sigmoid valves with obstruction and regurgitation, was advised to live with the precautions necessary to his condition, and from about sixty-five to nearly eighty continued to enjoy good health. He was of a placid temper, and so obedient a patient that he would let a train start before his eyes rather than hurry to catch it. He died at last from cerebral hæmorrhage.

A young man of thirty-one had been the subject of cardiac disease since an attack of rheumatism when a child. He had been educated at home, was never suffered to play like other boys, and at college lived the same careful and moderate life. When the writer saw him he was married, with healthy children, in active business, with good breath and healthy complexion. There was a loud diastolic basic murmur and a splashing pulse, with scarcely any signs of dilatation or hypertrophy, *i. e.* the nearly normal ventricle was sufficient for the regular calls upon its power.

From the point of view, however, of insurance, the risk is great and difficult to estimate; so that few offices will accept on any terms "lives" which are believed to be weighted with organic disease of the cardiac valves.*

Treatment.—The objects of treatment in valvular disease of the heart are—first, to spare the damaged organ as much work as possible, to avoid therefore all strains on the circulation, whether of muscular exertion or mental agitation; secondly, to maintain the nutrition of the part and particularly of the left ventricle in the best condition, so that (with or without compensatory hypertrophy) it may suffice to make up for the damaged valve; thirdly, to moderate the rapidity and increase the force of the cardiac contractions, and to lengthen the period of repose afforded by the interval between one beat and the next; lastly, to remedy the final stage of venous congestion and dropsy by mechanically relieving the circulation of the load of stagnant blood or exuded serum.

The first indication is met by enjoining a quiet, self-restrained life, with avoidance of the exposure which leads to bronchitis, and the intemperance which may end in Bright's disease; the second by prescribing animal food with moderate use of stimulants, adding tincture of steel, or any other preparation of iron which suits the patient better. In some cases arsenic is even more useful, and acts in much the same way, by improving the

* The writer may be allowed to refer to a paper "On some Points in the Prognosis and Treatment of Cardiac Disease," read before the Hunterian Society in the present year ('Brit. Med. Journ.,' November, 1890).

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blood and the muscles. To fulfil the third indication, we possess a direct cardiac moderator, making irregular action rhythmical, rapid ineffectual pulsation slower and more efficient, and feeble contractions strong, steadying the pulse, and raising the blood-pressure in the systemic arteries. This invaluable drug is digitalis. We use either the dried and powdered leaves of foxglove, or the infusion and tincture prepared from them. The condition most favourably influenced by digitalis is one of mitral regurgitation, with a dilated heart and a rapid, irregular, compressible pulse. In disease of the aortic valves with compensating hypertrophy of the left ventricle it is of little use, and when there is much dilatation it may be dangerous; but when primary sigmoid disease or mitral stenosis becomes complicated with mitral regurgitation, and the pulse acquires a "mitral" character, digitalis is invaluable.

In all cases of cardiac dropsy, it is our most important remedy. Besides its direct action on the heart, it is a powerful diuretic, probably by raising the blood-pressure in the renal artery. For this object it is much assisted by combination with mercury and squill. The pill so composed—"the infallible pill," as Addison called it—is the most valuable of remedies for cardiac dropsy. Other diuretics may also be usefully prescribed—acetate of potash, cream-of-tartar lemonade (the "imperial drink"), sweet spirits of nitre, decoction of broom-tops, and resin of copaiba.

Diaphoretics are not of much service in these cases, and if the kidneys are unfortunately diseased we must depend on the action of hydragogue purgatives. In other cases more gentle aperients help the action of the diuretic drugs.

When there is great ascites, it is well to tap at once, and to prescribe the pill when the kidneys have been thus relieved. For general dropsy acupuncture is a still better remedy. The patient should be got out of bed and the effused serum allowed to gravitate into his legs—a great relief in itself. They should then be oiled and punctured with a needle or lancet quite through the skin into the lymph spaces beneath; the benefit of the free flow of serum thus obtained is often most striking. The patient finds his breathing easier; he can sleep again and take food; the kidneys begin to secrete; the liver is relieved of its congestion; and a man who was at the point of death by suffocation is able to go about again free from dropsy, and in comparative health.

Occasionally engorgement of the lung will come on so quickly, and the whole venous system with the right side of the heart be so blocked, that *venesection* is called for, and may be practised with the greatest benefit; the indications for this treatment are orthopnœa, lividity of countenance, great distress of breathing, and pulsation of the jugular veins. After a few ounces or perhaps a pint of blood has been removed, the lividity will pass off, the breathing become tranquil, and often refreshing sleep ensue. Moreover, diuretics and digitalis will now act with effect, which before they could not do.

For a most distressing symptom of heart disease—sleeplessness, opiates may be given, not only with safety, but with the greatest benefit. One sixth or one fourth of a grain of morphia may be taken at bedtime, or, if it produce gastric disturbance, it may be injected hypodermically. If, from the presence of renal disease or any other cause, opiates are unsuitable, we may use Hoffmann's anodyne (sp. ætheris comp.) or full doses of hyoscyamus. Chloral hydrate is counter-indicated, but paraldehyde may

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take its place. Of all hypnotics except opium, the writer has found chloralamide and sulphonal most useful.

Extracts and tincture of the flowers and stem of lily of the valley (*Convallaria majalis*) have been recently revived as a remedy in similar conditions to those in which digitalis is valuable. The drug is undoubtedly efficacious in raising blood-pressure, and thus producing diuresis. It appears to be devoid of danger, and sometimes proves useful when digitalis fails or is counter-indicated. It was employed for dropsy in the seventeenth century, long before Withering introduced the use of digitalis, and it is still a popular remedy in Russia.

Citrate of caffeine is another useful cardiac remedy, and is much praised by some good physicians.

Strophanthus, an African arrow poison (*S. hispidus*, nat. ord. Apocynaceæ), was introduced by Dr Fraser. A tincture is prepared from the seeds, which yield an active glycoside, strophanthin. Fraser found that this drug increased the length of the systole in animals while slowing the rate of pulse, and that it was more powerful than digitalis. It is said not to have a cumulative effect. After thorough investigation in the laboratory it was tried in cases of cardiac, particularly mitral, disease (in doses of five or ten minims of the tincture or more), and with good results. Its remedial action is like that of digitalis, and it sometimes appears to succeed better, at least for a time; but at present strophanthus has shown no title to supersede the older remedy, and not infrequently fails where digitalis succeeds. See Prof. Fraser's paper (in the 'British Medical Journal,' November 14th, 1885), and one by Dr Quinlan (*ibid.*, August 27th, 1887).

In aortic cases, where digitalis is useless or counter-indicated, we depend chiefly upon iron in the early stages, and on ammonia, senega, and ether in the later.

One important object in these cases is to prevent flatulent dyspepsia by careful diet, and by the use of soda and nux vomica, bismuth, tincture of cardamoms, peppermint, and other carminatives. Strong coffee, wine, and brandy are often very useful.

In many cases of disease of the aortic valves, usually before general dilatation of the heart and dropsy have set in, there are developed symptoms like those of angina pectoris. For this severe paroxysmal pain nitroglycerine and nitrite of amyl are the appropriate remedies, with repeated small bleedings from the arm. Belladonna is sometimes given in these cases with good effect; also iodide of potassium, which has a double application in disease of the heart, relieving pain in the same way as it does in aneurysm, and a diuretic in cases of cardiac dropsy.

Of late years Prof. Oertel has advocated treatment of disease of the heart by a system of carefully arranged gymnastics, including ascent of hills. This plan has been opposed both in Germany and elsewhere, and it appears not to be supported either by physiology or by practical experience. The heart can never be an idle muscle, and when there is a valvular lesion present it is stimulated by the difficulty to increased exertion. To add to this exerting stimulus seems unnecessary, if not injurious; and to urge a patient to tax his heart beyond what he can do with comfort is meddlesome practice; we have ample experience of its bad effects from what we see in patients whose necessity compels them to practise Oertel's cure without knowing it.

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CONGENITAL DISEASE OF THE HEART.—This most frequently depends on a primary defect of development, but also almost certainly upon intra-uterine inflammation, sometimes perhaps of a true rheumatic character.*

Malformation from arrest of development without evidence of endocarditis.—When the heart consists (like that of a fish) of only two cavities, the auricle and ventricle, without any septum making a right and left division, life is rarely sustained after the cessation of the placental circulation at birth. One infant, however, with this defect is recorded to have lived seven days, and two others three days.

With two completely formed auricles and one ventricle, from which an aorta arises and supplies both lungs and body (as in a frog), life may be prolonged for weeks or even months (see Dr Peacock's collection of cases in his monograph on 'Malformations of the Human Heart,' 1866); and when the pulmonary artery and aorta are normal, adult life may be occasionally reached even though there is no trace of a septum in the ventricle.

A mere imperfection of the septum between the two ventricles (as in the chelonian reptiles) is a comparatively unimportant defect of development. It almost always occupies the "undefended space" between the base of the left ventricle and the sinus of the right. Persons with this abnormality have lived to adult age in apparent health, and died of some independent disorder.

Results of intra-uterine endocarditis.—It appears from twenty-three cases tabulated by Schipmann that this is rare before the fourth month of fœtal life. By far the most common and important effect of intra-uterine inflammation of the heart is *stenosis of the pulmonary orifice.* The limitation to the right side of the organ really comes under the same law as the corresponding limitation to the left side in extra-uterine life; in both cases the cavity which has most work to perform suffers most. Why the tricuspid valve should so frequently escape is less clear; probably it may depend on part of the incoming fœtal blood being diverted to the foramen ovale. Cases, however, of congenital stenosis of the right auriculo-ventricular valve are recorded.

The effect of this obstruction, occurring before the septum between the ventricles is completed, will be to perpetuate the aperture of communication in the undefended space, the ductus arteriosus will remain permeable, and the foramen ovale will continue unclosed.

The first case of this remarkable condition recorded was by Sandifort at Leyden in 1777, and the next was by William Hunter at Glasgow in 1783. In each the patient was a boy who lived to the age of about twelve years. Numerous cases have been since observed, in most of which the aorta communicates with the right as well as the left ventricle. In the most extreme degree of the lesion the pulmonary artery is completely obstructed (*atresia*), so that not only some but all the blood which reaches the right ventricle is expelled through the aorta, and the lungs are supplied through the ductus arteriosus. The septum ventriculorum is open, and under such circumstances life may be prolonged for months or possibly for years. Sometimes the constriction is not at the orifice of the pulmonary artery, but at the junction of the sinus with the rest of the right ventricle, so that that cavity of the heart is divided into two, like the ventricle and bulbus arteriosus of a frog's heart. From the position of the opening in the undefended space above described, there is still free communication between the

* The most important contributions to the pathology of this subject are by Rokitansky and Kussmaul in Germany, and by Peacock in England.

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right and left ventricles, and so the results are much the same as those which accompany moderate stenosis of the pulmonary orifice.

Lastly, the effects of endocarditis of the right side may be combined with one or any of the remarkable *transpositions* which occur in the origin of the great vessels from the heart. These combinations have been particularly investigated by Kussmaul, and are of great interest from a morphological point of view, but they are not frequent enough to be of clinical importance. Moreover, as we shall see, the physical signs and symptoms of congenital lesions of the heart are rarely capable of leading to more than a general recognition of their presence, and we must be content to base further diagnosis upon the age which the patient has attained, and our knowledge of the relative frequency of the several lesions.

Prognosis.—In many cases it is remarkable how adaptation and compensation by dilatation and hypertrophy prevent the results which beforehand would appear inevitable. The greatest mortality occurs in the first days or weeks of extra-uterine life. All the worst cases end in death before the infant is a month or two old, and if he survives the first year there is good probability of his being reared. With the development of the body at puberty fresh stress is put upon the heart, and often severe symptoms then first show themselves. Even if adult life be reached, the dangers of exposure to cold producing bronchial and pulmonary inflammation, and of muscular exertion breaking down the compensatory power of the heart are such, that of those who survive childhood few live beyond the age of thirty. The same factors, exposure and exertion, sufficiently explain the undoubtedly worse prognosis for males. Among the longest survivals in cases of (usually very moderate) congenital pulmonary stenosis, two cases were fatal at thirty, one at thirty-seven, two at forty-four, one at fifty-seven, and two at sixty.

Rokitansky believed that cyanosis protected from tuberculosis, Lebert thought it favoured consumption. In fifty-six cases of malformation of the heart with more or less cyanosis, where patients reached the age of eight, Peacock found that nine died of tubercular disease of the lungs.

When puberty is attained (and from thirteen to twenty-five) phthisis is undoubtedly the most common cause of death, a remarkable contrast to its rarity in stenosis of the orifices of the *left* side of the heart. The chief peculiarity of the disease is the frequency of severe hæmorrhage. It does not often run a rapid course, and tubercles of the larynx are said to be rare.

Symptoms.—The most important clinical results of congenital disease of the heart are dyspnœa and cyanosis; to these anasarca succeeds, and the other results of venous congestion and arterial anæmia, which have been detailed in the present chapter.

Cyanosis, or the blue disease (*morbus cæruleus*), was once supposed to be characteristic of congenital cardiac lesions, and to depend on mingling of the arterial and venous streams of blood. So William Hunter originally taught, and he was followed by Meckel, by Gintrac and Bouillaud, by Hope, Williams, Walshe, and Chevers. But it is not an uncommon result of chronic bronchitis, especially of bronchiectasis in children; and, on the other hand, it may be absent for many years, notwithstanding grave malformations of the heart. Still, in its extreme form it is seldom seen except in cases of malformation; and if absent it often appears on exertion or crying.

The question has been much discussed whether cyanosis, if admitted not to be due to actual mixture of venous and arterial blood, depends more upon non-aëration of the blood or upon venous congestion. The latter

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view was taken by Louis and Cruveilhier, Rokitansky, Stillé, and Peacock. Both conditions coexist in most of the cases now under discussion; but of the two, deficient aëration is probably the more constant. See a valuable paper by Dr Lees in the 31st volume of the 'Pathological Transactions.'

Cyanosis is most marked in the lips, the tip of the nose and ears, the fingers and toes; in bad cases the whole face is of a leaden colour, the eyes are bloodshot, and the mucous membrane of the mouth purple in tint. The blueness is increased by exertion, by coughing, and by cold. The fingers and toes are clubbed and the nails incurved. Though the patient is usually comfortable while at rest, exertion brings on dyspnœa. Orthopnœa is less constant and marked than in acquired disease of the heart.

The patient suffers much from the winter's cold, and in marked cases the hands and feet feel chill and clammy even in warm weather. The body is often well nourished and the secretions normal, while the muscular strength and mental faculties are usually not perceptibly impaired.*

In 101 cases analysed by Peacock, he found that symptoms were first noticed at or shortly after birth in 74, within the first year in 15 more, between one and two years after birth in 4, between two and five years in 5, and in the remaining 2 cases at thirteen and fourteen.

The *physical signs* of congenital pulmonary stenosis are as a rule—(1) increased cardiac dulness transversely and to the right from hypertrophy of the right ventricle; (2) a long and loud bellows murmur, systolic in rhythm, basic in position, but often unduly diffused, and frequently accompanied with a tactile thrill. The form of the chest, the seat of the apex-beat and the impulse, the character of the second sound, and the rate, volume, and regularity of pulse when the patient is at rest, are usually unaffected. But the pulse is liable to be easily rendered frequent, irregular, feeble, or intermittent. Occasionally a diastolic bruit has indicated pulmonary regurgitation; and, perhaps more often, there has been no abnormal sound whatever.

Treatment.—The first indication is to keep up the temperature of the patient's body. A cyanotic child should be wrapped in cotton wool, and the room kept at an equable temperature. If it survives infancy, it should be clothed from head to foot in flannel, and carefully shielded from exposure to inclement weather, even a degree of cold or of wind which is only healthful to an ordinary child. Fits of crying should be as much as possible prevented during childhood, and all exertion should be carefully restricted. Such children instinctively sit quiet by the fireside, although unfortunately they are sometimes of an excitable temper.

The convulsive attacks to which cyanotic persons are subject are often relieved by the application of a few leeches to the temples or below the ears; but great care should be taken that the loss of blood does not exceed the required amount (Peacock).

Cardiac palpitation, dyspnœa, bronchitis, and flatulence must be treated by the same measures as are found useful in acquired disease of the heart.

* Dr Peacock, however, says that children who are the subjects of congenital cardiac diseases have generally feeble mental power, and are usually thin. He quotes the following graphic description by Dr Wm. Hunter of his original case (1763): "Though he was remarkably thin, he had not the look of being emaciated by consumption; on the contrary, it appeared to be his natural habit. If a man had never seen any of the canine species but the bull-dog, he would be struck at the first sight of the delicate Italian greyhound. This young gentleman put me in mind of that animal, and when I looked at his legs especially, I could not but think of the legs of a wading waterfowl."

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CARDIAC DISEASE IN CHILDREN.—Apart from the congenital lesions just described, children are frequently the subjects of acquired disease of the heart, and in them it presents certain peculiarities of practical importance.

The parts involved are almost always the pericardium and the endocardium. Primary disease of the myocardium and lesions of the aortic valves are rare. Except in cases of chorea and in exceptional cases of adherent pericardium, there is scarcely any condition but organic lesions of the valves which produces a bruit. Even with considerable anæmia, it is very rare to hear murmurs in the heart, the arteries, or the veins. The pulse is naturally frequent in children, and even irregularity of the heart's action may occur in health. The apex-beat is usually higher in childhood than in adults, and sometimes a little further out.

Ætiology.—The origin of valvular disease as observed in childhood is in one of the following causes :—(1) Congenital lesions from malformation or intra-uterine endocarditis of the right side. (2) Rheumatism. This is common in children, but the synovitis and pain are commonly slight and the fever moderate, so that it is easily overlooked. On the other hand, there is great likelihood of the heart suffering, since there is no doubt that fewer children affected with (acute or subacute) rheumatism escape cardiac inflammation than adults attacked in the same way. (3) Chorea. This is often combined with cardiac murmurs, and these are probably organic, but chorea is so often preceded by rheumatism that certainly many, and perhaps the large majority, of these cases are due to the latter cause. (4) Scarlatina. Valvular lesions may not very infrequently be traced to an attack of scarlet fever; but this disease is so often followed by multiple synovitis of, in most cases, true rheumatic character, that here again most post-scarlatinal cases of endocarditis are really due to the latter malady. (5) Occasionally cardiac disease in children has followed measles, enteric fever, smallpox, or diphtheria.

In the great majority of cases, disease of the left side of the heart in children means previous rheumatic endocarditis. Girls are more liable to valvular disease than boys (167 to 81 of Dr Goodhart's cases).

Acute ulcerative endocarditis with infective emboli may occur before puberty, usually as a second attack on valves previously damaged by rheumatism. Atheromatous degeneration of the valves is unknown in childhood.

Anatomy and locality.—The nodules of coagulated lymph, the puckering, contraction, and adhesion of the valves, are what has been already described in the adult. Parrot (quoted by Dr Eustace Smith) has observed minute hæmatomata and fibrous nodules on the mitral curtains in infants dying under a month old; these gradually disappear without leading to further changes.

Incompetence is a more common result of endocarditis than obstruction in children, but the latter is a far from infrequent lesion of the mitral orifice. An apex systolic bruit is the commonest physical sign, and the lesions are probably, in order of frequency—mitral insufficiency, mitral obstruction, aortic regurgitation, and aortic obstruction.

The resulting hypertrophy and dilatation of the cavities does not differ from that seen in adults.

Symptoms.—It is remarkable that we rarely see the familiar type of either aortic or mitral disease in children, except in the final stages of their illness. The aspect of a child with cardiac disease is rather that of phthisis. He is pale and thin, with dilated pupils, a delicate skin, and a

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quick, easily excited, but usually regular pulse; he has often a short, dry cough, and gets out of breath when he runs.

Sudden death from disease of the heart (or, indeed, from any cause) is very rare in children. The last stage of their illness is marked by dyspnœa and exhaustion, or sometimes by general dropsy.

Prognosis and treatment.—For the most part, moderate valvular lesions are well compensated in childhood, and for many years there may be no symptoms whatever. Even when pallor and dyspnœa on exertion lead to an examination of the chest and discovery of the malady, it is often long before palpitation appears, and longer still before signs of venous congestion are added to those of arterial anæmia. The period of puberty and the five or six years that follow it are very critical, and the greater exposure and liability to the strain of labour and of emotion which adult life brings are unfavourable, so that few who have acquired serious lesions of the valves in childhood reach middle age. Some of the worst cases are those of adherent pericardium.

The general treatment consists—first, in fostering the nutrition of the patient by helping the appetite and digestion, and giving a due amount of meat with, in most cases, the addition of a little ale or port wine; secondly, in protecting him from damp and changes of temperature, while at the same time seeking to increase his power of resistance by constant woollen clothing, by well-ventilated dwellings and bedrooms, and by life as much as possible in the open air; thirdly, in prohibiting violent games, and all but the most equable and moderate bodily exercise; lastly, in giving the heart long periods of physiological rest by sleep.

In these cases residence in the south of Europe during the winter and spring, and, for those who are old enough, a long sea voyage, if possible on a sailing vessel, often prove most salutary.

Of drugs, steel is the most constantly useful. Digitalis, in small and long-continued doses, is next in importance. Strophanthus has not served the writer so well with children, and sometimes entirely fails to reduce the number of pulsations. In one case of advanced mitral regurgitation with dropsy, in a girl of about twelve, convallaria acted as a most useful diuretic after digitalis had apparently lost its power.

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PERICARDITIS

Ὅταν τὸ ὕδωρ πνίγῃ, τί δεῖ ἐπιπίνειν;
Proverb quoted by ARISTOTLE ('Eth. Nic.,' lib. vii, cap. 2).

Rarity of idiopathic pericarditis—its usual antecedents—Anatomy—fibrinous, serous, hæmorrhagic, and purulent effusion—Physical signs—Symptoms—Diagnosis—Prognosis and events—Adherent pericardium: its effects: its recognition—Hypertrophic pericarditis and pulsus paradoxus—Hydropericardium—Treatment of pericarditis and its results—Paracentesis.

INFLAMMATION of the pericardium has been recognised as an anatomical condition from the time that autopsies were systematically made; and attempts were made even before the discovery of auscultation to diagnose it during life—for the most part unsuccessfully. Laennec recognised the characteristic pericardial rub and its likeness to that of pleurisy.

It is convenient to deal with the subject in the same order with respect to diseases of the heart as meningitis occupied with reference to those of the brain, and pleurisy to those of the lungs; but inflammation of the pericardium is much less closely connected with disease of the viscus it covers than inflammation of the pleura, pia mater, or peritoneum.

Ætiology.—Pericarditis is very rarely idiopathic, *i. e.* it is not set up as a primary disorder by exposure to cold, like pleurisy. Indeed, the pericardium is too deeply situated to run such a risk. Moreover, the heart is not, like the lung, liable to acute inflammation, which might spread to the membrane covering it. If we exclude cases in which pericarditis is set up by wounds or direct mechanical injuries, it can, in most cases, be traced to one of two causes—either to a general malady, or to a previous local disease of a neighbouring part. The great majority of cases of pericarditis are secondary to either rheumatism or Bright's disease.

Professor Bäumler, now of Freiburg, published three cases of idiopathic pericarditis in adults, in the fifth volume of the Clinical Society's 'Reports,' and the writer once saw an uncomplicated idiopathic case with Dr Dalton, of Norwood, in a healthy man about forty. Acute pericarditis is said to have followed prolonged bodily exertion—for example, a long march; or exposure to cold, when it has perhaps been secondary to pleuro-pneumonia. Scarcely any acute disease is more rapidly fatal than double pleuro-pneumonia with pericarditis, but in these cases the pulmonary symptoms override those of the pericardial inflammation.

(1) In patients under forty years of age rheumatism is by far the most frequent cause of pericarditis, and in children under puberty almost the only one. After middle life it is rarely due to this cause, but the writer once saw an old gentleman of seventy-three who died of rheumatic pericarditis. It occurs as a rule a day or two after the pyrexia and synovitis have appeared. Its advent is almost a guarantee of the accuracy of

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the primary diagnosis, for pericarditis does not occur in cases of gout, of osteo-arthritis, or of gonorrhœal synovitis.

Pericarditis is sometimes the first manifestation of acute rheumatism, pain and swelling of the joints coming on only when it has existed for two or three days. Hence, when a patient dies of pericarditis after a very short illness, one cannot exclude the possibility of its rheumatic origin. This explanation applies especially to children, in whom such rheumatic pericarditis preceding any affection of the joints is much more common than in grown-up persons.

(2) Next to acute rheumatism, Bright's disease is the most common cause of pericarditis. It may occur in the more acute tubal form, or in chronic interstitial nephritis. In persons above fifty the latter is by far the most frequent cause of pericarditis.

(3) In Russia pericarditis has been often observed in the course of *scurvy*. Gout has also been mentioned as a cause, but this has probably only acted indirectly, through the renal disease which so often complicates it. Pericardial effusion with some inflammatory lymph in addition to the serum of dropsy is not uncommonly discovered after death in cases of cardiac dropsy, but usually when the kidneys as well as the heart are diseased. *Tubercular* pericarditis is not uncommon in the deadhouse, particularly in children, but is not often observed at the bedside, and occurs chiefly when several serous membranes together are attacked by tubercle.* Formerly *pyæmia* was said frequently to give rise to this and to other serous inflammations, but it is now believed that pericarditis occurs in pyæmic cases only when suppuration has first attacked the heart's substance.

(4) Pleuro-pneumonia (of the left side especially) is often associated with pericarditis; inflammation attacks both organs simultaneously, or may pass from the lung to the heart. Mediastinal suppuration, caries of the ribs, and a mammary abscess or pyæmic abscess of the heart are other causes of pericarditis. Also malignant growths of the mediastinum, or even of the breast, liver, or stomach.

Anatomy.—The morbid changes characteristic of pericarditis are those of a serous inflammation affecting an endothelial surface. In an early stage the membrane becomes minutely injected and loses its lustre. Then coagulable lymph appears upon its surface, often first around the roots of the great vessels. As this increases in amount, it forms thick concentric layers of fibrin, which may be stripped off in succession from the heart. The parietal pericardium likewise becomes lined with a similar false membrane, and between the two surfaces there is more or less serous or seropurulent liquid. In consequence of the incessant movements of the heart the surfaces now become remarkably roughened. Sometimes they bristle with a number of papillæ; more often they look honeycombed, and so may resemble either the second stomach or the paunch of a ruminant. Another good comparison is that made by Laennec, and afterwards by Hope, who say that the surface looks as if butter had been squeezed between two flat pieces of wood, which then were suddenly separated. So shaggy does the heart sometimes look in these cases that it was formerly known as the *cor hirsutum*. The progress made by pathology between 1750 and 1850 is shown

* Dr Mussex, of Philadelphia, has recorded a remarkable case of acute tubercular pericarditis, with effusion of sixty-four fluid ounces of bloody serum ('Trans. Coll. Med. Phil.,' November, 1888).

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by the fact that Haller described this "hairy" heart as occurring especially in bold and adventurous men. The thickness of the false membranes in pericarditis is far greater than is at all frequent in pleurisy or peritonitis.

In other cases pericarditis leads to the effusion of serum with only flakes of lymph. When the disease is acute this cannot much exceed the amount that it is possible to inject into the sac after death, viz. twelve to eighteen ounces; when this limit is reached the diastole of the ventricles is interfered with and great distress is produced, terminating in the rapid death of the patient. But in chronic cases a much larger quantity of fluid may accumulate in the pericardium; more than three pints have been found.

The serous effusion is sometimes tinged with blood, particularly in cancerous and, it is said, in scorbutic cases.

Suppuration appears to be much less frequent than in other serous sacs, but this probably depends chiefly on the preponderance of rheumatism as a cause, for rheumatic inflammation, whether of the joints, the pleura, or the pericardium, is seldom or never purulent. Purulent or sero-purulent pericarditis is not uncommon in cases of Bright's disease.

In very rare cases the fluid in pericardial effusion has been known to undergo decomposition, with the evolution of fœtid gas. This condition, *hydro-pneumopericardium*, has been observed when suppuration or malignant disease has extended to the pericardium from a mucous surface, as that of the œsophagus or stomach.

Inflammation often spreads from the pericardium to adjacent parts. Thus pleurisy may be set up, especially on the left side. In other cases the mediastinal tissues become affected, so that the parietal pericardium is fixed to the sternum by dense adhesions (cf. p. 98). The areolar tissue above the heart may participate in this change, and the left innominate vein may have its coats greatly thickened and its cavity plugged with coagulum—a point of some clinical interest, since it leads to œdema of the left arm without general dropsy.

Another and a very important extension of pericarditis is to the heart. The outermost strata of muscle are then found soft, and of a pale yellow or dull greyish-red colour. On microscopic examination it is found that superficial fatty degeneration of the myocardium has taken place.

Signs.—The recognition of pericarditis in practice turns upon the discovery of its characteristic physical signs, for the general symptoms, taken by themselves, are uncertain guides. The earliest sign is generally the friction-sound or *rub*. It is true that in some cases, even before this can be heard, the onset of pericarditis may be suspected from the heart's action becoming disturbed and "tumbling," and the first sound noisy and prolonged; but only the detection of a rub converts this suspicion into a certainty. The murmur, like an endocardial bruit, always accompanies the first or second cardiac sound, or both. It is never diastolic; sometimes it is systolic in rhythm, but more often double—a to-and-fro sound; or it may give the impression of a continuous churning going on without any complete intermission. Its quality varies like that of a pleuritic rub, from a harsh, dry, tearing sound, to a soft murmur like a râle, and in certain cases it is decidedly musical. The rhythm of the heart is often changed to a very characteristic three-beat or cantering cadence—the *bruit de galop* of Laennec. The seat of a pericardial rub is usually the base of the heart, and it is never propagated beyond the cardiac limits.

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It sometimes lasts for weeks; in other cases it rapidly disappears, after being audible for a few days, or only for some hours. This often is due to the fact that the two serous surfaces have become separated by fluid, and consequently no longer rub together as the heart moves.

The presence of this fluid is indicated by special signs, the most important of which is an increase in the area of the cardiac dulness; and in practice it is generally found that the augmented dulness is first discoverable at the base of the heart. Instead of the percussion-note in the third left interspace being but little less resonant than at the corresponding point of the opposite side, it becomes completely dull, and this dulness often reaches as high as the second rib, and sometimes even higher. When the quantity of fluid is considerable, the left lung is pushed to one side and compressed; and dulness on percussion may exist over so large a part of the left side as to cause the case to be mistaken for one of pleuritic effusion. This was the case with a patient of the writer's, a woman, in whom the gradual but at last enormous distension of the pericardial sac was ascribed to concomitant pleural effusion on the left side. Another sign of pericardial effusion, if extensive, is bulging of the præcordial region with widening of the intercostal spaces; and occasionally the diaphragm may become so depressed that the epigastrium bulges forwards, and the left lobe of the liver is displaced downwards.

Comparatively small quantities of fluid may suffice to separate the heart from the chest wall, and its impulse may consequently be diminished or imperceptible; but this sign is far from being constant, for it is remarkable how large a collection of fluid may fill the back of the pericardial sac, while the heart seems to float up in contact with the chest wall. Even with extensive effusion, an impulse can often be felt in the fourth interspace, slightly external to the line of the natural apex-beat.

The positive value of the physical signs above enumerated is very great; friction-sound is conclusive as to the presence of lymph; and increased dulness upwards, if developed during an attack of acute illness, proves that liquid has been poured out into the pericardial sac. On the other hand, it is by no means certain that the absence of a pericardial rub can be regarded as disproving the existence of acute pericarditis when some other severe disease is present. One may fail to discover pericarditis a few hours before death in cases of double pleuro-pneumonia, and yet the heart may be found covered with recent lymph.

Symptoms.—These vary remarkably in different cases; and in Bright's disease pericarditis is often altogether latent. Pain in the cardiac region and in the epigastrium may be most intense and agonising, and may radiate widely over the chest, and down the left arm to the elbow; while pressure over the heart or on the pit of the stomach may cause the greatest distress. But in other instances the patient feels no pain, nor is there any tenderness. It was maintained by Bouillaud and by Addison that pericarditis is painful only when it is associated with pleurisy, the pericardium itself being insensitive both in health and disease; but pleurisy also may exist without marked pain.

When pericarditis is acute, and particularly when there is large and rapid effusion of fluid, there is much *dyspnœa;* the patient can hardly speak for want of breath and tightness of the chest; his features are anxious and drawn; his nostrils dilate with each inspiration; he generally sits up or reclines on his back, with his head raised; but it is remarkable

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that when copious effusion has occurred he sometimes lies by choice flat in his bed, with scarcely a pillow, since the least elevation of the head produces a tendency to syncope. In other cases, however, there is marked orthopnœa.

The heart's action may be regular or irregular, quiet or greatly disturbed and attended with distressing palpitation.

The pulse in acute pericarditis may be quite unaffected. Usually, however, it is more frequent than normal, and in acute cases with large effusion becomes irregular or intermittent.

The earlier writers mention the occasional occurrence of violent cerebral disturbances in acute pericarditis. Maniacal delirium, rapidly fatal, has sometimes been the principal symptom; and the case has been regarded as one of cerebral inflammation, until the autopsy showed that the pericardium was the seat of disease. But similar cerebral symptoms occur in acute rheumatism, independently of pericarditis, when the temperature is greatly raised; and this fact renders it doubtful whether, when pericarditis is present, it is really concerned in the production of the delirium. The same may also be said of the choreic movements occasionally observed in pericarditis, for there is a very close relation between rheumatism and chorea. Apoplectiform stupor, hemiplegia, and convulsive attacks may probably be traced to embolism of the cerebral vessels from endocarditis. All the symptoms mentioned in this paragraph appear to be confined to cases of rheumatic pericarditis.

Dysphagia has been described in cases of pericardial effusion, and has been referred to the pressure on the œsophagus by the sac distended with fluid. Walshe disputes this opinion, and refers it to a nervous origin.

Diagnosis.—This is by no means always easy. A to-and-fro rub is usually unmistakable, for it has never the blowing character of an endocardial murmur, and its rhythm and seat are characteristic. But a soft systolic rub may resemble some kinds of valvular or functional bruit; or a pleural friction-sound may acquire a cardiac rhythm if its seat is the anterior margin of the left lung.

When effusion has occurred before the case is seen, diagnosis is often difficult. The best rule is to map out the limits of cardiac dulness on the surface of the chest, when its shape and daily progress will distinguish it from pleural effusion or mediastinal tumour.

Event.—It is rare for acute pericarditis to be the sole or even the direct cause of death. By Louis the average mortality was estimated at one in six cases; but in the pericarditis of acute rheumatism the immediate danger is far less than this. In Bright's disease, death often follows quickly upon the occurrence of pericarditis as a complication. Sometimes fluid effusion accumulates so rapidly as directly to hamper the heart, apparently by interfering with its diastole.

In favourable cases the symptoms gradually subside, and generally disappear in from twelve to twenty days; the fluid effusion diminishes by absorption, and if the corresponding surfaces of the heart and parietal pericardium are still roughened by lymph, a *redux* friction-sound may be heard on auscultation. After a time the lymph is also absorbed; but before this occurs the pericardial surfaces are often more or less extensively glued together and become permanently adherent. There is difference of opinion as to whether such adhesions occur in every case of pericarditis

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in which lymph has been effused. Some of the best observers think so, but the fact is open to doubt.

Adherent pericardium.—The physical characters of pericardial adhesions vary greatly in different cases. Sometimes, especially after the lapse of a long time, they are reduced to a mere film of connective tissue, which the fingers can tear through with but little difficulty. In other cases they are exceedingly tough, so that the only way to denude the heart is to strip off pericardium and adhesions together from the muscular fibres. Again, they may be uniformly of great thickness, or they may include masses of altered lymph, accumulated in certain parts of the pericardial sac, and especially round the great vessels. Lastly, the lymph may in course of time undergo calcification, and thus the heart be enclosed in a bony case.

This condition of obliteration of the pericardial sac by adhesion did not escape the notice of the older pathologists, but they supposed it to be a congenital defect.* Since its real nature has been understood, its clinical importance has often been doubted. Probably this depends on the kind of adhesions: if thin and areolar they appear not to hamper the heart's movements in any way, but a thick mass of hard fibrous tissue closely surrounding the organ may give rise to serious symptoms. By Hope and others it was maintained that an adherent pericardium always tended to cause hypertrophy of the heart's substance; but it is now known that this was a mistake. If one or more of the chambers is hypertrophied in such a case, this is the effect of some previous disease or of a coexisting valvular affection. On the contrary, the presence of thick pericardial adhesions is often associated with atrophy of the ventricular walls. This may in part be the result of the myocarditis which often accompanies pericarditis rather than of the adhesions themselves, but it is precisely in such cases that an adherent pericardium most often gives rise to symptoms, and that it can (if ever) be detected by physical signs.†

The early auscultators attempted to diagnose adherent pericardium by various signs which were long ago shown to be fallacious. One of these was the occurrence of systolic depression at the site of the cardiac impulse, while one or two intercostal spaces above this recede at the same time. Slight retraction of the spaces close to the sternum during the systole is by no means uncommon even when the pericardium is healthy; but it appears probable that obliteration of its cavity may generally be inferred when a considerable region of the chest wall is drawn in. Still, however, we may doubt whether this can occur without the pleura over the heart being adherent and the left lung being withdrawn from its natural position; and if so, it might perhaps be met with as a result of old pleurisy apart from any pericardial adhesion.

This systolic drawing-in instead of impulse was first carefully investigated by Skoda. He showed that it depends upon the natural movement of the heart in systole downwards, forwards, and to the left being hindered. The atmospheric pressure is then unsupported, and slight depression in the fifth space ensues. Probably for this result there must also be some adhesion or atrophy preventing the ear-shaped process of the left lung from expanding and filling the space. Another cause of the same phenomenon

* The pericardium is, as a rare anomaly, congenitally absent. Dr Mathew Baillie described this condition in the first volume of the 'Med.-Chir. Trans.' See also his 'Morbid Anatomy,' p. 13.

† See the late Dr G. H. Barlow's remarks in the 'Guy's Hosp. Reports' for 1847, and Dr Wilks's article in the same 'Reports' for 1871.

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is dense adherence between the visceral and parietal pericardium, an between the parietal pericardium and the chest wall. This may be th result of rheumatic pericarditis, but is more likely when there has bee extensive thickening of the anterior mediastinum in addition.

Basal adhesions are much more effectual in producing this result tha apical, as was proved by Weiss and Friedreich.

Some observers have endeavoured to diagnose pericardial adhesion fro the fact of the heart's dulness not being diminished during inspiration, o from the position of the organ remaining unaltered when the patient lies o different sides; but these signs are very uncertain.

The clinical effects of tightly adherent pericardium are most marke while the heart is still undergoing growth. Hence it is of much more prac tical importance in children than in adults. Apart from the physical sign just discussed, the want of development of the heart leads to weakness o the circulation, and to the arterial anæmia and venous congestion which we saw to be the ultimate results of all organic cardiac lesions if not otherwise cut short. The type of disease is mitral rather than aortic, and there is often considerable dropsy.

Accordingly, if we find a child with symptoms of chronic cardiac disease, but no murmur to be heard, and particularly if he has suffered from rheumatism, we may with great likelihood diagnose an adherent pericardium. Frequently in such cases there is valvular disease as well, and the pericardial adhesions make the result far more serious.

Mediastinal implication.—A remarkable pathological condition was first recorded by Griesinger in 1854, in which not only is the pericardium found adherent and enormously thickened, but also the adjacent anterior mediastinum, forming with it a continuous mass of indurated fibrous tissue. He observed in this case the "pulsus paradoxus," or *pulsus inspiratione intermittens*—an irregular and frequently an intermittent pulse which becomes imperceptible with each inspiration. Moreover, the distended jugular veins, instead of collapsing as usual with the free entry of blood to the right auricle which follows the expansion of the thorax, became fuller with each inspiration, apparently from the dense fibrous adhesions of the mediastinum dragging upon the innominate veins and superior cava. Kussmaul described three similar cases in 1873, when the fact had been apparently forgotten, and explained the diminution and cessation of the radial pulse as due to stretching and thus narrowing of the aortic arch by the same adhesions. Similar cases of chronic hypertrophic pericarditis, with the same intermission of the radial pulse and fulness of the veins during inspiration, have been described by Traube, Bäumler, and other observers, both in Germany and in England.

Milkspots.—We frequently find the visceral pericardium thickened and opaque in patches, on the prominent parts of the right ventricle in front or of the left behind, or on the auricles. These "milkspots" or "corns," as they have been termed, are not adherent to the parietal layer, and are not produced by acute pericarditis. They are the result of friction, and are seen most often on an hypertrophied heart, and when there is more than usual friction, as from a soldier's old-fashioned cross-belt. They are probably of no clinical significance.

Hydropericardium.—The pericardium may be distended with fluid under two conditions—excessive inflammatory exudation, and passive effusion as part of general dropsy. In the latter case it probably is seldom recognised

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during life and seldom gives rise to serious symptoms. Moreover, since large pericardial effusions are rare except in cases of renal dropsy, it will commonly be found that (as in hydrothorax) the effusion is not pure serum, but shows by the presence of fibrin that the process is active as well as passive. Accordingly, we may regard hydropericardium as usually the result of inflammation, and not of mere dropsy. An ounce or two of clear serum is often found in cases of anæmia, dropsy, and wasting diseases, and has no clinical significance. Beside the increased area of pericardial dulness, reaching the second and third left intercostal spaces and having uncompressed lung at the back of the chest, it has been observed by Professor Bäumler that when the sac is thus full of liquid effusion, the normal respiratory waves of blood-pressure become so much increased as to be perceptible in the sphygmographic tracing of the pulse.

When the fluid has distended the sac, the normal cardiac dulness is increased as in dilatation with hypertrophy of the heart, but it is increased upwards and to the left, not downwards, except in the last stage of enormous accumulation. It is then that orthopnœa, stridor, irregular pulse, and præcordial oppression are most marked.

Such cases appear for the most part as sequelæ of rheumatism or Bright's disease when the acute symptoms have passed off, and then the diagnosis is not difficult, particularly if the case has been watched from the beginning. If seen first when the effusion is already extensive, diagnosis is more difficult, and most of all when there is also effusion in the left pleura.

Treatment.—The treatment of pericarditis, according to modern practice, would have been regarded as very inadequate by the earlier auscultators, who sought to recognise the disease at the earliest possible moment, in order to combat its progress by antiphlogistic measures. It is most instructive to peruse the graphic and confident description by a writer of literary skill like Latham ('Lectures on Diseases of the Heart,' xii—xv) of the signs of acute pericarditis, of the bold treatment of the disease by bleeding and by mercury, and of its rapid subsidence under these measures. At the present time few would regard the danger as imminent, and probably none would believe that the classical treatment would avert it. Venesection is now rarely employed in this affection, and if leeches are used it is only with the object of relieving distress and dyspnœa, for which purpose they have undoubted value. Mercurials are scarcely ever prescribed: the influence of mercury on inflammation is denied by many of the most competent observers, and pericarditis offers little opportunity for testing its value, since the natural duration of the disease varies greatly in different cases. The therapeutical measures which we now adopt are as follows:—The patient is kept as quiet as possible in bed; the præcordial region is covered with a poultice, a thick layer of cotton wool, or a hot flannel; light fluid nourishment is given, with a saline or effervescing mixture, and opium or chloral or henbane is prescribed for pain and restlessness.

Nevertheless, not only may a few leeches applied over the sternum in the early stage of pericarditis relieve distress and perhaps limit the inflammation, but, when there are symptoms of embarrassed circulation with orthopnœa and distress, an irregular pulse, arterial anæmia, and venous congestion, the abstraction of four or five ounces of blood from the arm is found in some cases to give remarkable relief, and probably is never

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injurious. But the remedy is used to meet a special complication, and no with the intention of curing the disease.

Some physicians apply a blister as soon as a pericardial rub is audible But beside the practical inconvenience of this treatment, its benefit is mor than doubtful, and in cases of Bright's disease blisters should, if possible, b avoided. To relieve pain and quiet the heart's action, Dr Bäumler, fron experience in his own case, much prefers the application of a bladder of ice

When, however, large effusion has taken place, a blister, quickly followe by a poultice so as to promote free effusion of serum, is undoubtedly efficien in hastening absorption; at any rate, it has been followed by a rapid dimi nution of the dulness which for several days before had remained much more extensive than natural. Iodide of potassium is frequently given, with the hope of favouring absorption, and its efficacy in some allied affections is perhaps sufficient ground for employing it as a diuretic.

The efficacy of a blister was well shown in two cases under the writer's care. One was a boy of seven or eight, with such urgent symptoms of distress from distension of the pericardium in rheumatism that it was intended to tap him. By the prudent advice of a senior colleague a large blister was first tried, and (when that did not rise satisfactorily) blistering fluid with a large poultice afterwards, and complete success followed. The other was a girl of fourteen who had a (rheumatic ?) pericardial rub with no symptoms whatever. She was therefore left without treatment for a week without the least change in the physical signs. A blister was then applied, and the rub disappeared in twenty-four hours.

Paracentesis.—When urgent dyspnœa and threatening suffocation arise from the presence of large effusion, the operation of tapping the pericardium should be considered; its performance is justified by the imminence of the danger or by the failure of a large blister to relieve. This operation was suggested more than two centuries ago by Riolanus, but it appears to have been first practised by Romero, of Barcelona, in 1819, and with success (quoted as the first of fifty cases, in a dissertation on "Paracentesis Pericardii," by Hindenlang, 1879). Aran injected iodine into the pericardial sac after removing about two pints of fluid, and the patient recovered.

A hypodermic syringe may first be inserted to remove all doubt as to the diagnosis. A slight incision is then made, and the trocar is passed gently into the fourth or fifth left intercostal space, about an inch away from the sternum so as not to wound the internal mammary artery.

The operation has now been performed in a sufficient number of cases to make it trustworthy; and when, as sometimes happens, the pericardium is found after death enormously distended with pus, we regret that paracentesis had not been carried out. One of the most successful cases of paracentesis pericardii was published by Dr Samuel West. The patient was a lad of sixteen, who had been suffering from increasing dyspnœa for three weeks before he applied for admission to the Victoria Park Hospital. The physical signs led to the belief that a large pericardial effusion existed, and he was accordingly tapped. Fourteen ounces of pus were withdrawn. In a few days the same amount was again taken away; but the fluid still re-forming, an incision was made into the sac through the fifth interspace and a drainage-tube inserted. Two quarts of purulent fluid were thus removed, and the patient gradually but completely recovered ('Med.-Chir. Trans.,' vol. lxvi).

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DISEASES OF THE BLOOD-VESSELS,

INCLUDING ANEURYSMS AND OTHER THORACIC TUMOURS

Ἀρτηρίας ἀναστομωθείσης, τὸ πάθος ἀνεύρυσμα καλεῖται . . . . . διαγινώσκεται δὲ τὰ τοιαῦτα παθήματα τῶν σφυγμῶν τῶν ἐργασαμένων ἀρτηριῶν.—GALEN, *De Tumoribus*, cap. ii.

AORTIC ANEURYSM—*Definition of the term—Varieties—Anatomy—Origin—Symptoms—of aneurysm of the ascending aorta—of the arch—of the descending aorta—treatment—Abdominal aneurysms—origin—diagnosis—treatment—Dissecting aneurysms—Statistics of aortic aneurysm.*
INTRATHORACIC TUMOURS—*of the mediastinum—of the lung.*
THROMBOSIS AND EMBOLISM—*Causes of coagulation—Effects—Pulmonary embolism—Fibrinous infarction—Cases of recovery.*
EXOPHTHALMIC GOITRE—*History—Ætiology—Symptoms—Pathology—Event—Treatment.*

THE physical conditions under which the thoracic organs are placed render the detection of tumours within the chest more difficult and less certain than in either of the other great cavities of the body. For the walls of the thorax being formed by a bony cage, a tumour cannot be felt through them in the same way as through the yielding abdominal parietes. And the contents of the thorax being largely constituted of the soft and yielding lungs, the effects of pressure are not, as in the interior of the cranium, perceptible while a tumour is still small; and they may be absent long after it has attained a large size. These reasons explain the fact that it is sometimes impossible to carry the diagnosis of a case beyond that of "intra-thoracic tumour," using this term in its widest sense, to include aneurysms as well as new growths; and they justify, from a clinical point of view, the collocation of aneurysms and new growths in the present chapter. It will be convenient also to treat in this place of vascular bronchocele, and of the pathological conditions of intra-vascular clotting so far as they bear on internal medicine.

INTRATHORACIC ANEURYSMS.—An aneurysm* may be defined as a circumscribed tumour containing fluid or solid blood, communicating directly with the canal of an artery and limited by the tunic which is called the sac. It has long been questioned among authors whether the term should include cases in which the whole calibre of the vessel is dilated, and whether in the more circumscribed form it should have any reference to the number of

* Aneurysm (*ἀνεύρυσμα*, from *ἀνευρύνω*, to widen out, to dilate) is a term used by Galen and Paulus Ægineta to denote what we should now call a traumatic, false or secondary aneurysm. Its Latin equivalent is *dilatatio*, and it was applied by Corvisart to dilatation of part or the whole of a chamber of the heart. The word occurs in its modern restricted application at the end of the seventeenth century (Cook's 'Marrow of Chirurgery,' 1675; Salmon's 'Ars Chirurgica,' 1699; Blancard's 'Lexicon Medicum,' 1702); but aortic aneurysms were first investigated by Morgagni and Valsalva.—*Fr.* Anévrisme.

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arterial coats involved. As regards the former question, the terms *aneurysm* and *dilatation* are used as expressions of degree; a mere bulging of one side of the aorta would be spoken of as a dilatation, whereas a general dilatation of the whole circumference of the vessel, if limited in length, would be called a *fusiform* aneurysm. Not infrequently one side of the aorta may be so much enlarged as to produce symptoms by pressure on neighbouring parts, and then the term "aneurysmal dilatation" is often used. It is justified by its practical and clinical significance, since it applies to a case where the symptoms are referable both to the impeded circulation due to dilatation, and also the mechanical pressure of a circumscribed aneurysm. When we speak of "aneurysm" without qualification, we imply the existence of a circumscribed swelling of the *sacculated* kind, communicating with the vessel by a comparatively narrow aperture.

The division of sacculated aneurysm into two kinds, *true* and *false*, according to the number and nature of its coats, is useless, not only because the terms have been used in opposite senses, but also because the same aneurysm may in the course of time vary with respect to the number of these tunics. Scarpa, one of the earliest and most original writers on the subject, ascertained the fact that in most saccular aneurysms some of the coats are wanting; and he called these "true," in distinction from mere dilatations of an artery with all its tunics complete, or "false" aneurysms. Subsequent writers used the terms true and false as indicative of the perfection or absence of some of the coats of the vessel. It is better, therefore, to avoid these terms, or to use the word false as synonymous with *diffused*—that is, to denote the case of an artery in which the blood has burst through the real coats, and becoming effused in the tissues around, has formed out of them a pulsating swelling with new walls.

Anatomy.—Probably most aneurysms arise from a morbid softening of the inner coat, whereby a bulging occurs which pushes the middle and outer tunics before it. Before the aneurysm has reached a large size, it is found that the inner coat has become much attenuated or in parts atrophied. It may be sometimes seen over the whole of the inner surface, and continuous with that of the artery; in other cases it ceases abruptly at the neck of the sac, or only patches and shreds of it are discoverable over other parts of the interior. Frequently we find the whole inner coat smooth, but it differs from its original condition; the endothelium is absent, and it is intimately connected with the coats below. While the internal and middle tunics are thinned or destroyed, the outer one becomes much thickened, and in many cases constitutes the real sac of the aneurysm.

In the *fusiform* aneurysm with smooth walls there is no tendency for the blood to coagulate, but in the circumscribed or *saccular* form, with its narrow orifice and roughened interior, coagulation is ever ready to occur. This is promoted by the sluggish motion of the blood within it. When these more circumscribed aneurysms are examined, the interior of the sac is seen to be filled wholly or partially with fibrin: this is arranged in concentric layers,—the outer thin, hard, pale, and adherent to the arterial coats; whilst the inner are thicker softer, and darker. This shows that the deposition has taken place from the coagulation of blood, and not from any exudation from the walls of the sac. Nor does there seem to be any vascular connection between the sac and the fibrous clot. The mode in which this lamination of fibrin occurs is not very clearly ascertained: in the cases of rapid cure by pressure the sac is merely filled with a uniform coagulum;

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hence we learn that the sac need not be filled layer by layer. Lamination appears to take place in the coagulum afterwards.

Ætiology.—Aneurysms arise from special and definite causes, and are not merely due to the general degenerative changes found in the arteries as part of senile decay; for though scarcely ever seen under the age of twenty they are occasionally met with in young men, and are clinically almost as rare in old age as in youth. They are by far most common in men in the prime of life, and are very rare in women.

The process which sets up an aneurysm is a true arteritis or inflammation of the deepest part of the intima, and is quite distinct from fatty degeneration of the endothelium, which is always superficial. Arteritis involves the middle coat, and then ruptures the endothelium, forming what used to be called an "atheromatous ulcer." The first histological products are leucocytes, then fatty and calcareous degeneration follows, producing a "branny" detritus (*atheroma*). (See Dr Moxon's paper in the 16th volume of the 'Guy's Hospital Reports,' Third Series.) Whether the term atheroma be used or Virchow's term Arteritis deformans, it is important to remember that the process is one of chronic inflammation followed by degeneration, not of primary fatty or calcareous transformation.

The origin of this arterial inflammation is to be sought first in the wear and tear, the strain on the arteries which is caused partly by mechanical stretching, particularly in the artery of the ham, in the subclavian and innominate, and partly by varying and excessive internal blood-pressure. Thus it may occur early in life as the result of excessive stretching of the arteries in walking or climbing, or in lifting weights, or in rowing; or as the result of great efforts made with the breath held, the thorax fixed, and the muscles contracting to the utmost of their power. The former cases will more affect the arteries of the limbs, the latter the aorta.

Again, the same increased internal strain from high blood-pressure may be produced by the imperfectly known conditions of chronic Bright's disease—not the arterio-capillary fibrosis which affects the smaller vessels, but the high tension which accompanies this change, and produces its effects on the aorta and its primary branches. Thus, atheromatous arteritis becomes connected with gout, with plumbism and alcoholic intemperance, and with chronic aortic and mitral stenosis. We cannot say that atheroma, still less that aneurysm, is the result of Bright's disease; but all these morbid conditions are found together, and have more than an accidental connection.

Lastly, atheroma appears as a purely senile change due to the normal and necessary wear and tear of the circulation.

As in other cases, we must consider the tissues as well as the irritant. The amount of strain which sets up extensive atheroma in one man has no such effect in another; thus we must regard aneurysm from the point of view of vulnerability of the arterial system, which is probably often hereditary.

A sudden strain or violent exertion may be the direct precursor of aneurysm, either by producing an actual rupture of the coats or by setting up an inflammatory process. It is a well-known fact that the thoracic aorta is very liable to be affected in those who take violent exercise, especially in those who use their arms in rowing. In such persons the vessels become much altered in configuration, the coats thickened, and the interior atheromatous; thus aneurysmal sacs are liable to occur. The aortic valves also are apt to be affected under the same conditions. These are the causes which

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are supposed to be especially in operation in soldiers, who are found to be very liable to aneurysm; but it must be remembered that, besides the drill and other exercises, syphilis may have a share in their production.

That violent exertion is productive of aneurysm is shown in the greater liability of *men* than women to the disease; also by the labouring classes being most obnoxious to it, particularly soldiers, sailors, porters, and others who work hard with their arms. Probably *lead*-poisoning may be an occasional cause, for it is remarkable that the workers in this metal not only suffer from gout, but from the diseases which so often accompany gout, as granular kidney and diseased blood-vessels; consequently they have been found to suffer not infrequently from aneurysms.

That *syphilis* may be a cause of aneurysm had been conjectured, and there can now be no doubt from careful observation in all countries that this is by no means an infrequent antecedent. It seems not to produce atheromatous arteritis, but a more patchy and more softening form of inflammation which leads to aneurysm without extensive deformity and thickening elsewhere. It is analogous to but not identical with the endarteritis obliterans described as affecting the cerebral vessels (vol. i, p. 556).

In the smaller arteries aneurysms have been demonstrated to arise in connection with the acute softening arteritis produced by a septic *embolism*. In a case under the writer's care in 1883, ulcerative endocarditis was seen to produce first embolism and then aneurysms of the radial and posterior tibial arteries successively. The patient was a young and otherwise healthy man, and he recovered of the acute disease, having only a well-compensated imperfect aortic valve. Each aneurysm was nearly as large as a marble. (See Messrs. Langton and Bowlby's important paper, 'Med.-Chir. Trans.,' 1887.)

Symptoms.—Dilatation or fusiform aneurysm of the ascending aorta is the result of atheroma, and leads to loss of elasticity in this part of the vascular system; the pulse is not sustained in diastole, and the shock on the smaller arteries is greater. Moreover, the same atheroma frequently befalls the aortic orifice, leading to valvular disease. Clinically this form of "aneurysm" is quite distinct from saccular aneurysm of the aorta, although anatomically they are, as we should expect, often found together. The symptoms of saccular aneurysm depend upon its size and position, and, as already stated, connect it clinically with a new growth within the thorax.

THORACIC ANEURYSM.—It is convenient to treat of thoracic and abdominal aneurysms separately, and to divide the former into those of the ascending, transverse, and descending thoracic aorta.

(1) Aneurysms of the *ascending aorta* are strikingly different from those of the rest of the arch on account of their tending towards the front of the chest, and therefore not implicating the important structures which aneurysm of the transverse and descending aorta must do.

(*a*) If the aneurysm form a pouch in the sinuses of Valsalva, the valves become involved, their closure may be prevented, a diastolic bruit be produced, and all the other signs and symptoms dependent upon regurgitation follow. These may indeed be the only symptoms, and none exist indicative of the presence of an aneurysm. The sac never grows to a great size; it usually bursts into the pericardium, and thus causes instant death.

(β) When the aneurysm is higher up, it will continue to increase, sometimes without much inconvenience, until its presence is apparent by a pulsating swelling or projection of the chest. Very often previous to its

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appearance pains in the chest have been experienced, but not always of a severe character. When it has come forward, it may be recognised as a bulging of the chest to the right of the sternum, and usually in the second space. When the hand is placed over it a distinct pulsation is felt; this is synchronous with the heart's action, as may be ascertained by placing the other hand over the apex. Sometimes the touch detects a thrill or *frémissement.*

If the swelling should project so as to be grasped by the hand, a distinct expansion is experienced. This expansive character of the swelling is the most important and distinctive sign of an aneurysm. Without it there is little certainty of its nature, for a pulsating tumour may be nothing more than a growth situated over an artery, and if a bruit is present it may be produced by pressure on the vessel; but no dilatation would be felt by the hand which grasps the swelling. The expansion may sometimes be well shown by covering the swelling with a piece of plaster in which a slit has been cut; if this be narrowly watched the slit will be seen to widen at every beat of the sac. If in the early stages a pulsation is felt, but not seen while looking directly at the chest, it may often be clearly observed by placing the eye on a plane with the patient's chest, either by looking over his shoulder when he is in the erect posture, or by stooping to a level with his body when he is supine. Or the stethoscope may be placed on the spot as a lever, and the pulsations recognised as they are magnified at its distal extremity.

On applying the ear a distinct throb or jar is communicated to the ear, and sometimes a systolic murmur is heard. This is, however, by no means always the case, for it depends upon the relation of the sac to the vessel, and is due to the existence of a constricted opening, through which the blood passes into a larger space beyond. It is very rare to hear a diastolic bruit; if it exists, it must depend upon some peculiar and exceptional circumstance, or may be a transmitted cardiac murmur from aortic regurgitation. As a rule the second sound is not only clear, but accentuated.

The heart is not usually enlarged from saccular aneurysm of the aorta; it is only when an aneurysm exists at the very commencement of the aorta that enlargement occurs, as in primary disease of the valves. The apex-beat is, however, often found somewhat lower than natural, owing to the direct downward pressure of a large aneurysm. Occasionally the tumour presses upon the superior vena cava, and then some enlargement of the veins of the neck may be observed, or the surface of the chest may be seen covered with distended veins. In some remarkable cases a communication has been formed between an aneurysm of the ascending aorta and the superior vena cava, in others between the aneurysm and the pulmonary artery. If large, the sac may press upon the trachea or bronchus, and impede the entrance of air into the lung. Sibilus and rhonchus might thus be produced, and if ulceration took place into the bronchus, hæmoptysis. If the aneurysm approach the axilla, the subclavian vein may be involved, and the nerves of the brachial plexus, so as to cause swelling and pain of the arm. Most frequently, however, aneurysms of the ascending aorta push forward, and absorbing the ribs or passing through the sternum, project outside of the thorax as circumscribed tumours—sometimes of a very large size and irregularly bossed; often of the size and rounded form of an orange. The skin may become thin and red, but the aneurysm very rarely bursts externally. When this is threatened, blebs form with thin sanguineous contents, the surface becomes discoloured, and at last a raw surface

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is formed, which from time to time exudes blood. Even then, however, the elastic corium long resists pressure, and after alarming hæmorrhage has again and again been stanched, the sac usually bursts inwards.

The percussion-note over an aneurysm is of course dull, the extent varying with its size and forward growth.

(2) In aneurysm *of the transverse arch of the aorta* the symptoms are more numerous and varied than those of the ascending part, owing to the fact of many other structures being implicated. An aneurysm at this part produces all the mechanical effects of a tumour. Hence new growths and aneurysms are constantly mistaken for one another, for if the latter be deep-seated pulsation may be altogether wanting. If we consider the close packing together of the aorta, trachea, bronchi, œsophagus, pneumogastric, recurrent laryngeal, and sympathetic nerves, it is evident that any tumour like an aneurysm forming amongst them must interfere with some of these structures. The vertebræ may be eaten away; or the pulmonary artery may be pressed upon and even opened. The parts usually involved, however, are the trachea and bronchus. These being for some time pressed upon, subsequently ulcerate until the mucous membrane is reached and an oozing of blood takes place. If the aneurysm contain much fibrin, and the solid portion be in contact with the air-passages, this oozing of blood or occasional hæmoptysis may continue for a long time, for weeks or months, and in some exceptional cases for years. Sooner or later, however, the blood breaks through in large quantities, and causes immediate death by stifling or by syncope.

The most frequent seat of aneurysm of the arch is at the origin of the innominate artery, and this vessel is often dilated so as to form part of the sac.

If the aneurysm form in the transverse aorta behind the sternum, it will probably soon show itself on the left side as a pulsating tumour about the second left costal cartilage. If there have been no symptoms, this, when first observed, may be mistaken for the left auricle. As in the case of the aneurysm of the ascending aorta, it may be seen and felt to pulsate, and occasionally a bruit is heard.

Should the aneurysm grow downwards beneath the arch no external signs of it are apparent, and the symptoms are all due to its pressure on the parts which surround it.

One of the most common of these symptoms is *dyspnœa*. This may arise either from pressure on the trachea or bronchus, or from implication of the pneumogastric nerves. In the former case there is evidence of the pressure by sibilus, and by mucous expectoration tinged with blood (cf. vol. i, p. 917). In other cases the difficulty of breathing is due to paralysis of the larynx or its vocal cords. It is usually the left vagus which is involved, and it is sometimes found after death closely incorporated with the coats of the sac, and perhaps much thinned. Under these circumstances the muscles of the larynx, especially the posterior crico-arytænoid, are much atrophied. The symptoms due to pressure on the nerve generally differ from those caused by direct pressure on the air-passages, the difficulty of breathing being paroxysmal and the cough peculiar. It is like that of a croupy child, ringing or brassy, and in the intervals the breathing may be free and natural. If the laryngoscope be used, it will be found that one of the vocal cords, generally the left, is paralysed and motionless; and sometimes, although only one nerve is involved, both cords become paralysed, causing great difficulty of breathing, and threatening suffocation (cf. vol. i, p. 871). It must be remembered that

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during the act of breathing the larynx is actively opened to let in air, and therefore if the muscles be paralysed it would close during inspiration.*

Pressure on the œsophagus may occasionally cause difficulty of swallowing; but more frequently pressure on the vagus or pulmonary plexus may set up an insidious form of *pneumonia*. Frequently after sibilus has been heard, febrile symptoms ensue, and it is found that one lung is becoming solidified. After death, the lung is then found to be hepatised or in a state of purulent infiltration, probably, as Sir William Gull and Dr Budd long ago supposed, by implication of the trophic nerves of the lung.

Pressure on the sympathetic nerve in the neck may affect the pupil of the eye, its paralysis causing *myosis*, or contraction of the pupil, in the same way as paralysis of the third nerve causes dilatation. The pupils ought, therefore, to be carefully examined in cases of suspected aneurysm. The left is the more often found contracted.

Sweating on the same side as the contracted pupil has been recorded in cases of aneurysm by Professor Gairdner ('Edin. Med. Journ.,' 1856), and also by Dr Bramwell (*ibid.*, 1878); but in the latter case the hyperidrosis had preceded the symptoms of aneurysm by years, aud in cases of undoubted injury to the cervical sympathetic, as well as of its experimental division in animals, the secretion of sweat is checked, not increased, on the affected side (cf. 'Journ. of Physiology,' vol. viii, p. 26).

As regards *pain*, this varies according to circumstances. As a rule it is a pain in the chest or down the arms which first brings the patient to the physician. This in course of time may become very acute, and is sometimes accompanied by actual weakness of the arm. The pain may have its origin in the aorta, and so, through the sympathetic and spinal nerves, is at last referred to various parts of the chest and upper extremities. If the aneurysm be situated at the lower part of the arch it may erode the spine, and so immediately involve the nerves passing to and from it.

It is very rare for aneurysm to run its course without pain, and in these rare cases it is most apt to appear early as a pulsating tumour.

Another important indication of the existence of an aneurysm may sometimes be found in the *pulse*, and especially in one pulse being smaller than the other. This may arise from three causes: the subclavian artery may be compressed by the sac from without; or it may come off from the sac and its mouth be closed by fibrin; or, more frequently, the artery is open, but the aneurysm, being deficient in the normal tension of the aorta, fails to produce due pulsation in the vessels coming off from it. It must be remembered that when the blood is delivered by the ventricle to the aorta, the elasticity of the latter does not prevent each shock being felt in the distant arteries; but if the aorta were converted into a large sac the impulse of the heart would be lost in this space, and the blood would flow out through the efferent vessels in a continuous stream. A case once in Guy's Hospital exemplified this: the pulses at the wrists became quite imperceptible, but the warmth of the hands and their vascularity showed that the blood flowed into them as before. It is the exception, however, for the pulse, as in this instance, to cease completely; more usually that in one wrist becomes more feeble than that in the other. If the aneurysm should involve the innominate, the right radial pulse would be enfeebled; if the left subclavian, the left. The sphygmograph is found useful

* On this subject see, however, Dr Bristowe's remarks in the 3rd volume of the 'St Thomas's Hospital Reports' (1872).

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in demonstrating on paper the difference between the perfect pulse of one wrist with all its parts, and the imperfect tracing of the other.

Retardation of the left radial pulse or in the left radial and carotid, compared with the arteries of the right arm and neck, may sometimes help to the diagnosis of an aneurysm of the transverse arch between the innominata and left carotid.

(3) Aneurysms of the *third part* of the arch and *descending thoracic aorta* may be taken together. They soon involve the bones of the spine, which they erode, and expose the spinal nerves. Pain, therefore, is one of the commonest symptoms, and may be the earliest. It is often very defined in the course of a particular intercostal nerve, and so the seat of the disease may be accurately determined. A descending thoracic aneurysm may also involve the lung, or press upon a bronchus, or compress the œsophagus. It may make its way backwards, and involving the ribs be felt at the back; it may even enter the vertebral canal, and compressing the cord produce paraplegia (cf. vol. i, p. 473). In the former case the pulsating tumour may be felt and seen, and a bruit be heard with the stethoscope.

Diagnosis.—The recognition of a thoracic aneurysm is sometimes easy and obvious, sometimes it is extremely difficult.

When a pulsating tumour can be seen and felt, it can only be an aneurysm, or an abscess or tumour which receives an impulse from the heart, or which is extremely vascular. With care it is almost always possible to discriminate between these possibilities.

When no tumour or expansion or even pulsation can be discovered, we must depend entirely on the symptoms of pressure above detailed. Of all of them pain is the most constant, and should never be carelessly put down to "neuralgia," still less to "hysteria" or to malingering.

The most frequent difficulty, after due care and observation have been used, is to distinguish between an aneurysmal and a solid intrathoracic tumour. Here we are helped by the sex and age of the patient. Aneurysm is unlikely in a woman, and almost certainly absent in a patient under twenty. On the other hand, its presence is probable in a labouring man, in a soldier or a sailor, and in a case where we find (with or without a history) the evidence of syphilis. Enlarged lymph-glands above the clavicle, or in the axilla, and pleural effusion, are almost certain indications of a new growth.

Results and prognosis.—It is very rare for aneurysm to undergo spontaneous involution. That this may occur is proved by some solid and cured sacs being discovered accidentally in the deadhouse or dissecting-room. They are found in bodies wasted by phthisis or cancer, or the aneurysm may in its growth compress its own artery, and thus cure itself.

With such rare exceptions, when the dilatation has once begun it goes on until the sac becomes thin and ruptures. Even if an aneurysm becomes contracted and solid, fresh sacs are pushed out from a neighbouring atheromatous surface.

Aneurysm of the first part of the ascending arch opens into the pericardium or into the pulmonary artery. In the latter case death may be long delayed and strange murmurs may be heard.

Aneurysms of the rest of the arch most often open into the trachea or the left bronchus, rarely into the œsophagus.

Aneurysms of the descending thoracic aorta sometimes open into the lung, but more often burst into the pleura by a gaping fissure, which never

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allows oozing as a mucous surface does, but empties the whole sac at once and kills by sudden syncope.

The duration of thoracic aneurysms after they are recognised must be reckoned by months rather than by years; but they may grow for two years or more before they prove fatal.

Death often occurs from implication of the lung causing pneumonia, sometimes from disease of the left sigmoid valves, and occasionally from some intercurrent disease.

Treatment.—The object of the physician in treating aneurysm of the aorta is the same that the surgeon has in view in treating aneurysm of a limb—to retard the movements of blood in the vessel and aid the deposition of solid fibrin on the walls. This is more likely to be effected when the opening of the sac is small and the interior roughened.

The method of cure is to lessen the rapidity and force of the circulation; and for this end the patient must be kept absolutely at rest in the recumbent posture, and subjected to a strictly limited diet.

The soundness of the principle on which this treatment is founded was well shown by the late Dr Moxon in an article in the 'Guy's Hosp. Reports' for 1866. He there remarks on the few and perhaps doubtful cases of spontaneous cure of aneurysmal tumours described by Porter (and also referred to by Heberden), and quotes Liston's case of an axillary aneurysm cured by a second sac forming above it and obliterating the subclavian artery; and proceeds to give an account of a case of aortic aneurysm cured by the supervention of cancer, and to quote six cases from the 'Pathological Transactions' in which consolidation of an aneurysm was found after death from phthisis, or other causes of anæmia and wasting.

The original method proposed by Albertini and Valsalva was bleeding the patient repeatedly and keeping him on the smallest amount of food. This treatment is deprecated by Tufnell, who, however, has revived the method in a modified form with remarkable success. The object, he allows, is to reduce the circulation and keep the blood in a highly coagulable state; but this is not accomplished by starvation. His experience has been mainly with abdominal and peripheral aneurysm, but the same method equally applies to aneurysms of the thoracic aorta. He allows three meals a day and a small amount of fluid, with absolute rest for at least two months. The value of rest in the recumbent posture he illustrates by the fact that in one patient the difference of the number of beats of the heart between the sitting and recumbent position was thirty-five per minute. This, he says, is equal to more than 50,000 in the twenty-four hours, and no remedy in the Pharmacopœia could produce so striking an effect. The diet he recommends is the following: for breakfast, two ounces of bread-and-butter and two ounces of milk or tea; for dinner, three ounces of mutton and three ounces of potatoes or bread, and four ounces of claret; for supper, two ounces of bread-and-butter and two ounces of tea: the total per diem being ten ounces of solid food and eight ounces of fluid.

Of the two constituents of treatment, *rest* and *low diet*, the former is the more efficient and indispensable. The patient must not sit up to take food, nor be moved to have his bed made or to relieve his bowels. Even suddenly turning over should be avoided, and he should move his arms as little as possible. Sleep should be procured when necessary by bromides or chloral, or, if there is much pain, by opium or by injection of morphia. The latter drugs are useful in the early part of the treatment by stilling hunger as well

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as pain, but after a few days the pain usually subsides and the patient sleeps comfortably.

The restriction of solid food is comparatively easy; that of liquid is more difficult. Thirst may be relieved by ice, by effervescing lozenges, or by sucking slices of lemon. While aiming to reduce the water taken to half a pint a day, the diminution must often be gradual, and sometimes may stop short of that limit without apparent harm.

After two or three weeks of this regimen the pulse sometimes becomes very rapid and feeble, with palpitation of the heart. The strict dietetic treatment must then be relaxed, opium with digitalis given for a time, and then the food cautiously diminished again. In certain cases all attempts in this direction are baffled, and it is then wise to let a patient eat and drink moderately, and to trust to the effects of rest and iodide of potassium.

In favourable cases pain rapidly subsides and then disappears, pulsation is less violent, the swelling diminishes, and after two months or more there may be neither physical sign nor symptom of the aneurysm. This occurred in a patient under the writer's care, when the sternum had been perforated in two places, and the sacs had both become filled with clot and firmly contracted.

As soon as the tumour has disappeared the patient's food should gradually be increased; and next he should be allowed to sit up. Lastly, he may be restricted from laborious occupations only, and suffered to walk about as usual; but it is wise to continue the iodide for several weeks, and to avoid all excesses in meat or drink, in bodily efforts or mental emotion.

Digitalis is often given, but probably without any benefit, and the same may be said of lead, ergotine, and many other drugs; the only one which can be said to be really efficient is the *iodide of potassium*. This is now given in large doses, fifteen to thirty or even sixty grains, three or four times a day for several weeks; and sometimes with marked success. Under its use the aneurysmal sac becomes hard, as if the remedy favoured the deposition of fibrin. Whilst this is taking place the pain is subsiding, which shows that the pulsation of the sac produces symptoms not due to the tumour alone; for when the latter has become hard and inert they disappear. It is true that the cured aneurysm is smaller than the active one, but this alone will not account for the subsidence of symptoms. The fact is well seen in cases of popliteal aneurysm where the pain and tenderness immediately cease after the arrest of the circulation through the sac.

Dr Balfour has published some remarkable cases of benefit by treatment with iodide, and Dr Bramwell recorded several others in the 'Edin. Med. Journ.' of 1878.

Sometimes, however, the case has gone too far for treatment before it is seen, or the patient may be intractable and perverse; or the treatment, after success for a time, is followed by more obstinate return of the disease; or the tumour threatens immediate rupture. To relieve urgent symptoms which cannot wait for the effect of rest, and indeed prevent it, the most efficient remedy is small and, if needful, repeated *bleeding*. The four or six ounces taken from the arm, recommended by the late Dr Hughes Bennett, rarely fail of effect. More than once a patient, who had suffered many days and nights of unrelieved pain and sleeplessness, with frequent attacks of agonising suffering, after ice-bags, morphia in large doses, and other means had failed, has obtained complete relief and sound sleep after a single venesection.

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For the radical cure of thoracic aneurysm, when the plan of treatment above described is inapplicable or has failed, various local measures have been attempted, but not with much success. One of these is *galvanism.* The poles of a battery have been passed into the sac so as to produce coagulation of the blood. The needles must be well insulated, and of course must not touch; but probably the best plan is to use the positive pole only, so as to procure the firm non-gaseous clot which forms on the anode, and to place the cathode as a large wet sponge on an indifferent part in the neighbourhood. This practice has been successful in the hands of Ciniselli, and partly so in those of Dr John Duncan ('Edin. Med. Journ.,' April, 1866), but is now generally abandoned.

Another plan, introduced by the late Mr Moore, is to pass several yards of fine iron wire into the sac. At Guy's Hospital this has been tried with imperfect success, and horsehair has also been used. The danger is setting up inflammation or sloughing of the sac. Only two patients have survived the operation more than a fortnight; and about twenty other fatal cases are recorded. In one case, under the writer's care, this plan was adopted in fear of rupture. The effect was to produce inflammation of the sac, to increase the patient's sufferings, and to hasten his end.

Ligature of the distal vessels has been several times performed, and in some cases with temporary benefit.

Dr Macewen, in an able paper on the treatment of aneurysm, has advocated the introduction of needles into the sac, so as to irritate its internal surface, and produce a firm parietal clot ('Brit. Med. Journ.,' Nov., 1890).

ABDOMINAL ANEURYSM.—Aneurysms occur more frequently at some parts of the abdominal aorta than at others. The most common site (in 133 out of 177 cases collected by Sibson) is just below the diaphragm, at the origin of the cœliac axis, which is often involved; they are more rare at the origin of the superior mesenteric artery, and are only seldom found lower than this point. The form of aneurysm here, as in the descending aorta in the thorax, is always saccular. Its anatomy agrees with what has been already stated of thoracic aneurysm.

Symptoms.—The first symptoms of abdominal aneurysm are generally painful feelings in various parts of the belly. These may be dull and aching in character, or lancinating and paroxysmal; they sometimes seem to encircle the body like a girdle. They may be due to the sympathetic nerves being involved in the sac, or the spinal nerves may be directly affected by the aneurysm eroding the vertebræ, or forward pressure of the sac may cause the pain.

If the patient have an aneurysm in the upper part of the abdomen, pulsation will be most usually found in the left hypochondrium, or at least to the left of the median line. A distinct tumour may be felt, expanding under the hand, or pulsation only; this may be so great as to give considerable movement to the stethoscope when placed upon it. If not only an impulse but also an expansion be felt, the diagnosis of aneurysm is at once established. Sometimes also a thrill can be perceived. On auscultation a systolic bruit will almost always be heard.

If the aneurysmal tumour be lower down, it can more easily be grasped and its nature made out. If it grow backwards, it may be detected in the loin, both by its pulsation and by the existence of a bruit; and this bruit cannot be produced by pressure of the stethoscope, as is frequently

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the case when it is heard in front in a thin and anæmic subject. Various other symptoms may be present owing to the pressure of the sac. Actual obstruction of the intestine has been recorded; but the constipation which is frequently observed probably arises indirectly. A very careful examination of the femoral arteries will sometimes show a slight retardation of their beat as well as a diminished tension.

The end of an abdominal aneurysm is generally rupture; this may take place directly into the peritoneal cavity, causing instant death, or behind the peritoneum, whereby the blood becomes effused into the areolar tissue, and a coagulum is formed for the time. This new or false sac soon, however, gives way, with the result of fresh and probably fatal hæmorrhage. A cœliac aneurysm sometimes opens into the stomach, causing profuse hæmatemesis and more or less sudden death; still more rarely into the transverse colon. Very exceptionally the coagulum becomes hard, and, being incorporated with the original aneurysmal sac, a cure is effected. Other exceptional terminations of abdominal aneurysm are sometimes met with, as in a patient in Guy's Hospital, in whom a loud whizzing bruit was heard in the lower part of the back, and who also had dropsy of the lower extremities. After death a small aneurysm was found communicating with the vena cava, so as to make an aneurysmal varix. The venous blood was retarded in its flow upwards, and so the dropsy was produced.

Ætiology.—The causes of abdominal aneurysm are the same as in the thoracic form. Violent exertion is no doubt a frequent cause, and this is the reason why it is so much more common in men than in women; for in women it is exceedingly rare. In several cases there has been well-marked syphilis, and no doubt can exist as to this being a frequent cause. Of three female cases in Guy's Hospital two were syphilitic. One of them occurred in 1853 under the late Dr B. G. Babington; the patient was a prostitute, and was being treated for syphilis, when she died suddenly from rupture of an abdominal aneurysm at the cœliac axis.

Diagnosis.—This is not always easy, since pulsating tumours may exist in the abdomen which are not aneurysmal, and bruits may be discovered in the abdominal aorta without disease of its coats. In anæmic women especially pulsation in the abdomen often occurs, and bruits are heard, which suggest aneurysm. Again, a tumour may be raised up by the aorta and pulsate, and a bruit may be heard, but these two signs are not sufficient to warrant the diagnosis of an aneurysm; nothing but the rhythmical expansion of the sac can afford proof. If the patient be placed on his hands and knees, and the tumour be handled, any falling forwards would be in favour of a morbid growth, for an aneurysmal sac would still remain in its place. A murmur audible over the first and second lumbar vertebræ is also a characteristic symptom.

Treatment.—The general treatment of abdominal aneurysm is the same as that of thoracic—restricted diet, absolute rest, and potassic iodide.

The opportunity sometimes occurs of using mechanical pressure as a local method. The plan was successfully carried out by Dr Murray, of Newcastle, in 1863, and has since been followed by others. He found that a very short period may be sufficient to cause coagulation in the sac. The aneurysm must of course be sufficiently low down. Unfortunately the more common position at the cœliac axis is too high to allow room for any instrument being applied above the tumour. In Dr Murray's first case ('Med.-Chir. Trans.,' vol. xlvii, p. 187) pressure was used for two

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hours, and again for five. The aneurysm was rapidly cured, and the man remained well for six years afterwards. It was found after death that the aneurysm came off from the aorta at the origin of the inferior mesenteric artery; this was obliterated, and the circulation had been carried on through collateral branches. This case was followed by one of Dr Greenhow's, in which one application was not sufficient; pressure was repeated again and again for a week, and at last a perfect cure resulted. Mr Durham reported a case about the same time of an abdominal aneurysm cured by pressure. This was kept up with the patient under chloroform for ten hours until pulsation of the sac ceased. The circulation also stopped in the femoral arteries, and coldness of the legs came on. Pulsation again occurred in the aneurysm, but in a less degree. The treatment was continued, but pulsation was not completely arrested in it for a month. Perfect recovery then followed.

Another case occurred in Guy's Hospital where pressure was kept up for some hours on the *distal* side of a high aneurysm. No apparent effect upon it was produced; but collapse came on, and in twenty-four hours the patient died of peritonitis. The duodenum was found bruised and covered with lymph. The sac contained a coagulum which appeared as if it had been deposited during life.

The bold plan has also been adopted of opening the abdomen, exposing the aneurysm, and inserting iron wire (Pringle and Morris, 'Med.-Chir. Trans.,' 1887).

Dissecting aneurysm.—Instead of expanding into an aneurysm, the aorta sometimes ruptures from disease. It is rare for the whole of the coats to give way simultaneously; more usually the intima is lacerated; the blood then finds its way between the tunics and produces a dissecting aneurysm; finally the adventitia ruptures, and causes the patient's death. When the blood is effused in this manner, it tears asunder the layers of the middle coat, so that part is found united with the intima and part with the adventitia. The following cases illustrate this remarkable form of false aneurysm.

A former sister of Petersham Ward, about sixty years of age, was seized a month before her death with a violent pain across the chest and abdomen, her heart became quick and tumultuous, and it was thought at the time that she was dying. She, however, rallied, had some slight febrile symptoms, and in a few days was doing duty in her ward. She had another similar attack and again quickly recovered; but two days later she fell dead whilst dressing. The autopsy showed the pericardium to be full of blood which had proceeded from a rent at the origin of the aorta. On the inside the rent was seen to be an inch long just above the valves, and through this blood had passed between the coats of the vessel; outside the external coat was ruptured, but this did not correspond to the internal opening. Some distance above this, and at the beginning of the transverse arch, was another fissure, an inch long and older in date; it had smooth edges connected by bands, and was quite healed. Proceeding from this was effused fibrin in the coats of the artery, which were separated from one another to the end of the abdominal aorta. The separation had occurred in the whole circumference of the vessel.

Another example was that of a gentleman about sixty years of age. After returning from the City and eating his dinner, he was seized with a

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severe pain in the chest, which continued all the evening until he went to bed. He was found afterwards dead at the side of his bed. The autopsy showed the pericardium full of blood, which proceeded from a fissure in the aorta. A laceration of the inner coat was seen to have occurred half an inch above the valve; it ran almost completely round the vessel. Blood had passed through and separated the coats throughout the arch, the descending thoracic, and the abdominal aorta. It had, however, taken only about two thirds of the circumference. The clot was quite recent. The laceration in the outer coat was at right angles to the internal one; it was an inch and a half long, and opened into the pericardium.

A third case was reported by the author in the 52nd volume of the 'Med.-Chir. Trans.' It occurred in a gentleman aged seventy-one, who had long suffered from signs of cardiac disease, and died very gradually. The rupture in the intima was less than an inch above the valve, and opened into a large aneurysm which communicated again with the aorta in the third part of the arch; but the false channel continued side by side with the descending aorta to beyond its bifurcation, and only ceased when it opened into the common iliac arteries.

Dr Peacock, who thoroughly investigated the subject of dissecting aneurysm ('Path. Trans.,' 1863), speaks of a variety occasionally met with, in which a long time elapses between the rupture of the internal and external coats, so that a distinct pouch may form like an ordinary aneurysm, and become in time lined by a smooth membrane. The third of the above cases is an example of the latter and rarer form.

Statistics.—During the ten years from 1875 to 1884 inclusive, there were ninety-two cases of death in Guy's Hospital from saccular aneurysm of the aorta, or nearly 2 per cent. of the total autopsies. An abstract of the *post-mortem* records was made by Mr Alfred Parkin for the writer, who has added eight of his own cases either antecedent or subsequent to that period, so as to make the number a hundred.

As to the *sex* of the patients, 93 were men and only 7 were women.

As to their *age*, one patient was eighteen at the time of his death, 5 were between twenty and thirty, 29 were between thirty and forty, 33 between forty and fifty, 25 between fifty and sixty, 3 between sixty and seventy, one was seventy, and one (a woman) was eighty-three.

Although all the cases were *saccular*, in no less than sixteen there were one or more other pouches, some of which were obsolete and solid; and in several others there was more or less diffused dilatation of the aorta. In three cases the inner coat only had ruptured and had formed a *dissecting* aneurysm.

The part of the aorta affected was in 55 cases some part of the arch, and in 28 of these in the ascending part before the origin of the innominate artery; in 7 it was the descending thoracic aorta; in 2 the sac was just at the orifice in the diaphragm, and in 15 it was below that boundary, 11 above and 4 below the origin of the superior mesenteric artery. It is, however, often difficult to say precisely whether a bulky sac, with perhaps some diffused dilatation around it, should be reckoned to one section of the vessel or another.

In 47 cases there was rupture of the sac (including six in which it had opened into the pulmonary artery), in 52 the sac had not ruptured, and death was caused by pneumonia, bronchitis, or pulmonary œdema, by syncope, by embolism, or by some independent disease.

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Of the thoracic cases in which rupture occurred, the sac burst into the right pleura in 4, into the left in 7; into the trachea in 4, the left bronchus in 4, and the left lung in one; into the œsophagus in 4, and the posterior mediastinum in one; into the pericardium in 4, and into the left auricle in one, beside the six above mentioned into the pulmonary artery. The abdominal aneurysms ruptured nine times into post-peritoneal tissues, once into the peritoneal cavity, and once into the stomach. In the whole number of cases there was none of external rupture, although in two the threatening signs described above had appeared.

INTRATHORACIC NEW GROWTHS.—These tumours are liable to be mistaken for aneurysms, or the reverse; and therefore, though pathologically so different, they may be conveniently considered here. From the same clinical point of view, the new growths that occur in the thoracic cavity may be divided into two groups: (1) those which are seated in the mediastinal tissues, or at least implicate the root of one or both lungs; (2) those which affect the lungs without interfering with their roots.

Mediastinal new growths.—These tumours differ in different cases in almost every particular,—in their starting-point, in their histology, and in their results. We are obliged to group them clinically under a single heading, since no distinction can be drawn between them during life.

As regards the *origin* of a mediastinal growth, it is seldom possible to come to any positive conclusion at an autopsy, when it has generally reached a great size and involved various tissues. The common supposition is that they often start from the bronchial and other lymph-glands; and this is doubtless correct for cases in which the thoracic affection is only part of Hodgkin's disease or general lympho-sarcoma, as well as for those in which it is secondary to a tumour situated in some other part of the body. But on more than one occasion we have found the glands only partially attacked; some have been infiltrated with the new growth, but others, though embedded in it, have retained their natural structure. In such cases the origin may be assumed to be the thymus or the mediastinal connective tissue and fat, or perhaps the pericardium, or the periosteum of the sternum or vertebræ.

As to the *histology* of mediastinal tumours, the vast majority of them are made up principally or entirely of small round cells, and are classified either as lymphomata or as round-celled sarcomata. But some specimens contain a large proportion of spindle-cell tissue, others consist of little but fibrous tissue, and some have been described as having an alveolar structure. In yet other cases mediastinal tumours have been found to be of a syphilitic nature, being formed of gummata embedded in a dense fibrous material. Lastly, some have been dermoid cysts, containing hair, bone, and occasionally teeth, beside a quantity of fat.

Among the *relations* which mediastinal new growths bear to the various structures contained in the thoracic cavity, by far the most important are those that concern the great systemic veins and the main air-passages.

It is almost a peculiarity of the affection now under consideration to produce *obstruction of the venous trunks* either of the superior vena cava or of one or other of the innominate veins, or of all three. In one case a tumour in the lower part of the thorax reduced the orifice of the inferior vena cava

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to a mere slit, through which the finger could scarcely be passed. As already observed, aortic aneurysms occasionally, but only occasionally, interfere with the veins (p. 105); and almost the only other cases in which the flow of blood through the superior cava is retarded or prevented are certain cases of heart disease in which, during an acute attack of pericarditis, there has been inflammation of the mediastinum with thrombosis of one or both of the innominate veins, ending in obliteration of the affected vessels (cf. p. 98). But when there is a mediastinal new growth, venous obstruction is a very frequent result. Sometimes the growth penetrates the coats of the vena cava, and fungates within it as a soft smooth mass. Sometimes it surrounds that vessel or one of the innominate veins, and causes extreme narrowing or even complete obliteration of the blood-channel. In either case there may be a consecutive thrombosis of the jugular and other tributary veins.

The clinical effects of these lesions are sometimes very marked. There may be great œdema of the arm and hand on one side or on both. The neck and the face may be enormously swollen and of a deep red or purple colour, with obvious over-distension of the veins. When the obstruction is limited to the superior cava, a collateral channel for the passage of the blood may be afforded by the azygos vein, which becomes dilated. But when both innominate veins are blocked, the intrathoracic vessels can do little towards carrying on the circulation, which then depends in great measure upon anastomoses between the superficial veins of the chest and back with those of the lower part of the trunk. The consequence is that the body becomes covered with dilated vessels, and may acquire a deep purple or claret colour. We can make out in which direction the blood has to flow, for when one of the veins is emptied by pressure along its course it fills far more rapidly from above downwards than from below upwards. Or, by passing a piece of string round the chest, we may see at once that the vessels above it remain full, whereas those below it become empty. But in many cases the appearance of the affected parts is in itself a sufficient indication. For it is a curious fact, doubtless dependent on the presence of valves in their interior, that obstructed veins are apt to become far more tortuous when the circulation through them is in the reverse, than when it is in the natural direction. Thus when the superior cava is blocked the veins may be zigzagged and varicose all over the chest and the upper part of the back, whereas those over the lower part of the body may be a little larger than natural, but almost straight. If there is obliteration of the inferior cava, on the other hand, the effect may be exactly the converse. Sir Thomas Watson, in his sixty-third lecture, records and illustrates by diagrams two cases in which this distinction was very manifest.

A man who died in Guy's Hospital from a mediastinal new growth in 1868, said that the first indication that anything was wrong with him was that a sensation of swimming in the head came on when he stooped, owing probably to venous congestion of the brain.

Obstruction of the air-passages is the effect of compression of the trachea or of the bronchi by mediastinal growths. The records of necropsies at Guy's Hospital contain very few cases in which these parts were found untouched by the disease. But sometimes it is noted that although their walls, even to the mucous membrane, were infiltrated, there was yet no narrowing of their calibre.

In most cases the growth extends along the bronchi into the pulmonary

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tissue. Sometimes it fills up a great part of the lung, forming large masses in its interior and even reaching its surface. For obvious reasons the disease is then either confined to one side, or at least far more marked on one side than on the other. Dr Powell, in 'Reynolds' System,' says that the left lung is invaded more often than the right, but among twenty-six cases taken from the records of autopsies at Guy's Hospital the numbers on each side are nearly equal.

The *physical signs* caused by mediastinal new growths are due chiefly to their size, and to their pressure on the air-passages and the lungs. Their bulk is sometimes enormous. In one case there was a solid mass that weighed ten pounds, and another in which the measurements in three dimensions were ten inches, seven inches, and five inches respectively. Such great tumours naturally cause considerable enlargement of that side of the chest which they principally occupy. The intercostal spaces may be widened and flattened, and the movements of the ribs much impaired. But, on the other hand, if the growth is attended with shrinking of the lung, the measurements may be less than those of the opposite side. In some cases the tumour protrudes above the clavicle, so as to be felt at the root of the neck; in others it bulges through one or more of the intercostal spaces. It may lead to some absorption of bone, but it does not appear as a rounded swelling, projecting far beyond the natural level of the ribs, as an aneurysm does. It may, however, pulsate more or less forcibly without true expansion, and a systolic murmur may be transmitted through it from the heart or the aorta.

As may be imagined, such large growths as these cause great dulness on percussion. This is commonly very marked over the sternum, and for a greater or less distance on each side of it. But it may also extend over the whole of the front of the chest on one side up to the clavicle, or to a variable distance over one scapula, or between the scapulæ, or even over the whole of one half of the chest from apex to base, including the axillary region. Not only is the percussion-sound absolutely toneless, but the sense of resistance to the finger may be extreme. The condition may therefore be exactly like that which would be produced by liquid effusion into the pleural cavity; and as a matter of fact such an error of diagnosis has been often committed, and repeated exploratory punctures have been made, which of course have led to no good result. The best way to avoid this blunder is to map out carefully and mark with ink or an aniline pencil the area of dulness in all directions, when it will be found to extend beyond the bounds of pleural effusion. But the difficulty is increased by the fact that when the pleura is reached by a new growth, effusion as a rule follows, and sometimes in large quantity. Hence the success of a paracentesis is no warrant for resting satisfied with the diagnosis that there was liquid in the serous cavity; that may be merely a complication of a far more grave disease. If there is altered blood in the liquid, giving to it a dark brown or reddish tint, the presence of a tumour is almost certain. But in some cases of mediastinal tumour the pleural effusion is straw-coloured, and exactly like that seen under other conditions.

It must be borne in mind that there are cases, especially where the growth is limited to the root of one or both of the lungs, in which percussion yields negative results.

The other physical signs of mediastinal tumours are very uncertain. Tactile vibration is sometimes increased, but more often lessened or abolished.

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It is, of course, only in the latter case that the disease is likely to be mistaken for pleural effusion. There may be more or less loud bronchial breathing, or a very faint vesicular murmur may be audible when the patient inspires, or there may be absolute silence. The stridulous and other sounds produced by narrowing of the lower air-passages by the pressure of a tumour have been described in the first volume (pp. 918 and 922). In many cases there are râles, of varying quality, not over the tumour itself, but over the parts of the lung to which the branches of an obstructed bronchus pass. To these signs may be added those of consolidation, when pneumonia ensues.

In many cases mediastinal growths invade the pericardium, generally at the base of the heart; and they may then spread downwards to a greater or less extent, both along the parietal layer of the serous membrane, and also in the walls of one or both of the auricles. The pericardial sac may either become closed by adhesions, or it may become distended with liquid effusion, which is often sanguineous, or it may be affected with inflammation, leading to the exudation of lymph or of lymph and pus. Of the great vessels, the aorta appears almost always to escape entirely, the venæ cavæ (at least the vena cava superior) are very often greatly narrowed, as has already been stated; the main divisions of the pulmonary artery and of the pulmonary veins are also in many instances pressed upon, so that their calibre is much reduced. At first sight it might appear probable that cases in which the pulmonary vessels are involved would be characterised by more intense dyspnœa than would otherwise be present. But in practice this is not observed, nor ought it perhaps to be expected, inasmuch as Lichtheim has shown by experiment that one fourth of the natural calibre of the pulmonary artery suffices to keep the lungs fully supplied with blood. Even when the heart is not invaded by a mediastinal growth it is often much displaced; generally it is pushed downwards and to the left, but sometimes, when there is shrinking and contraction of the left lung, it is dragged up so as to be felt pulsating not far below the clavicle. Although the aorta itself resists the pressure of the growth, it may happen that some of its branches are more or less narrowed, causing the radial pulse to be weaker and smaller on one side than on the other.

The œsophagus does not seem to be often occluded by mediastinal growths; at least in the reports of autopsies at Guy's Hospital there are very few instances in which this is said to have been the case, or in which dysphagia is noted as having been among the symptoms. One patient is reported to have brought up his food almost directly after attempting to swallow it; and at the autopsy the œsophagus was found pushed aside, but not invaded. In some rare instances the disease extends into the spinal canal, causing a variety of "compression-paraplegia."

One curious effect of a mediastinal growth which was observed many years ago, in the late Mr. Cooper Forster's ward, was apparently due to pressure upon the vaso-motor nerves. A woman, aged twenty-six, was admitted, in the summer of 1866, for a defective state of the circulation in her fingers, which were blue, cold, shrunken, and also very painful and tender. After a few weeks she was discharged, but she again came in under Dr Moxon in the following April, and died some months later. At the autopsy it was found that a growth infiltrated the fibrous tissue in front of the spine, and involved both the first dorsal nerve and the sympathetic trunk. Nothing abnormal was ever detected in the state of the pupils.

In other cases in which there is pressure upon the sympathetic on

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one side at the root of the neck, the corresponding pupil has been noticed to be smaller than the opposite one, especially when but little light reaches the eyes, so that both pupils should normally be dilated. The explanation is that when the sympathetic is paralysed, the natural balance is lost and the contracting fibres of the third nerve get the upper hand. In three cases of mediastinal growth, Rossbach is said to have further observed that both pupils dilated regularly with each inspiration; in two of these cases, in which there was a swelling above the clavicle, pressure upon this tumour caused the pupils to become widely dilated, while the frequency of the pulse also underwent a temporary alteration, being retarded in one instance and quickened in the other.

The other symptoms of mediastinal growths vary widely in different cases. There is generally more or less dyspnœa from the time when the patient first notices that anything is amiss with him. Very often he is obliged to sit up, even at night; sometimes the only position in which he can sleep is leaning forwards, or even lying on his face. The breathing is generally quickened to twenty-four or thirty in the minute.

Pain is commonly an early symptom, and sometimes (though not usually) it is very severe. It may be referred either to the side, or to the shoulder, or to the middle of the chest in front. Sir Risdon Bennett speaks of it as sometimes sudden and transitory, and attendant on physical exertion.*

Most patients have a troublesome *cough*, which is often described as "ineffectual," giving rise to little or no expectoration. But sometimes a viscid mucus is brought up, and occasionally this contains blood so intimately mixed with it as to give it the appearance of red currant jelly, a sign of some diagnostic importance. The spitting of pure blood, too, is not uncommon, and it may occur at the very commencement of the clinical history of the case, and more than a year before the fatal termination. Profuse hæmoptysis is seldom observed, but Dr Church has recorded in the 'Pathological Transactions' (vol. xix, p. 64) one instance in which four pints of blood were brought up immediately before death; the bleeding probably came from broken-down lung-tissue in the neighbourhood of the growth, rather than from the growth itself.

Cachexia is by no means a prominent symptom. The patient often looks well and ruddy for some time after he comes under treatment, and even at the last there is not often extreme emaciation. Pyrexia is generally absent, but Sir Risdon Bennett relates a case (*loc. cit.*, p. 121) in which the temperature had varied from 100° to 101·4°, but in which Dr Sutton, who made the autopsy, could detect no appreciable imflammatory changes, so that the only way of accounting for the febrile disturbance was by referring it to the active cell-growth that had been going on, not only in the lungs, but also in other organs of the body.

To complete this account of the occasional symptoms of mediastinal tumours, it may be noted that in two cases of intrathoracic dermoid cysts the patient expectorated hairs, in one of them for twelve years before death.

Occasionally no symptoms are present at any period of the case, which is cut short by disease of some other part of the body, most frequently by a similar tumour of the brain. Several instances of this kind have occurred

* Lumleian Lectures on "Intrathoracic Growths," p. 179. This refers rather to a sudden effort or injury producing a pain, to which the patient refers as his earliest symptom. The same author says, with reference to the later symptoms, "The amount of mere pain is seldom such as to call for the use of any large quantity of opium. But the distress is often very great" (p. 186).

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at Guy's Hospital, and they are of interest, not only in themselves, but also as showing how little dependence can be placed upon the duration of pulmonary symptoms as an indication of the real rate of progress of the disease. On the other hand, it must not be forgotten that even if we did theoretically know the time necessary for a growth to develop itself from its very commencement until it destroys life, we should still be unable, so long as we cannot fix the date at which it begins, to make any use of the knowledge in clinical practice.

In nine cases at Guy's Hospital in which the duration of the symptoms was noted, it varied from two and a half to eleven months. The extremes in each direction are probably afforded by two cases cited by Hertz, in 'Ziemssen's Handbuch:' one which proved fatal in a week from the first appearance of symptoms; the other in which life was prolonged for at least seven, and possibly for fifteen years.

Diagnosis.—It will have been gathered from the last section how difficult, and sometimes impossible, it may be to recognise the existence of mediastinal tumours. In most cases, however, the physical signs point to a mass within the thorax, not pneumonic or tubercular, and distinct from, though often complicated by, pleuritic effusion. The diagnosis is then between new growth and aneurysm. In the absence of pulsation we depend on probabilities. Severe pain, absence of pyrexia, and the fact of the patient being a man and having been laboriously occupied, incline to aneurysm. Insidious origin, œdema of one arm, effusion into the chest, and enlarged cervical or axillary glands, together with the fact of the patient being a woman, particularly a young woman, or a child, point decidedly to the diagnosis of tumour.

It may be laid down that, while pleuritic effusion should make one suspect tubercle in young subjects, and in older patients Bright's disease, we should also consider the probability of lymphoma or sarcoma.

Ætiology.—With regard to the *causes* of mediastinal growths very little is known. In one or two recorded instances they have been attributed to injuries, such as blows upon the sternum; but it may well be doubted whether this is more than a coincidence. A remarkable contribution to the ætiology of the disease was a paper by Hesse in the 'Archiv der Heilkunde' for 1878, where it is stated that in the mines of the Schneeberg 75 per cent. of all the miners—from twenty-one to twenty-four each year—die, generally about the age of forty, from "cancer of the lungs," spreading from the root. Prof. Ernst Wagner examined some specimens of the disease and found that the growth was a lympho-sarcoma. Only two explanations appear possible: one that it is the result of a tendency inherited and transmitted from generation to generation; the other, that it depends upon the nature of the minerals worked in the mines, which contain bismuth, cobalt, and nickel, with some arsenic and sulphur. With regard to the first suggestion it is noteworthy that the miners of the Schneeberg are recruited from among the sons of former miners; whether intermarriages are frequent does not appear. Dr Walshe mentions the cases of two brothers who were each affected with intrathoracic growths, and a parallel instance will presently be mentioned.

Most observers say that more men than women die of mediastinal growths, and this is confirmed by a collection of thirty-three cases from the *post-mortem* records of Guy's Hospital, the proportion being more than two to one. As regards age, the numbers for each decennial period from twenty to sixty are almost exactly the same; whereas it is generally

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stated that the disease is more frequent in persons between twenty and thirty than in those who are older. A few cases have been observed in children.

The prognosis in cases of mediastinal new growth is very grave. If recovery should take place in a case diagnosed as one of this disease, the general impression would be that a mistake had been made, and that the patient was really affected either with some inflammatory or syphilitic thickening of the intrathoracic structures, or else with aortic aneurysm. But we had one instance which goes some way towards establishing a different conclusion. It is that of a man named John Bullions, who was admitted into Guy's Hospital under Dr. Habershon on February 1st, 1867, with loss of voice, stridulous breathing, great swelling of the neck, œdema of the chest, and fulness of the veins. There was also slight deficiency of resonance on percussion over the right apex and over the root of the right lung behind. Under the administration of iodide of potassium he rapidly improved, and left the hospital on March 2nd, after which he returned to work. But on May 22nd he was readmitted with what was apparently an attack of erysipelas of the face and neck. This also quickly subsided, and from that time we lost sight of him. However, in 1871 another man, named Thomas Bullions, aged nineteen, came into the hospital and died of a mediastinal new growth, as was proved by the autopsy. Struck by his peculiar name, we inquired and found that the former patient was his elder brother, and was then in good health, though still rather short of breath. Were both cases of the same nature, like those recorded in the two brothers by Dr Walshe, or were they different?

In the *treatment* of this disease a faint hope of cure is by the administration of iodide of potassium, arsenic, or mercury. When there is great venous obstruction marked relief is often afforded by venesection, cupping, or leeches. To ease pain recourse may be had to blisters or mustard poultices, and also to the various anodynes; for the cough, Risdon Bennett recommends antimony in small doses with a sedative.

Pulmonary new growths.—Only those cases remain to be mentioned in which the lung is affected with a new growth that leaves its root free. Clinically they differ widely from the mediastinal cases just described. In many instances the patient up to the time of his death shows no indication of anything more than general cachexia, and it is only at the autopsy that the lungs are found to be studded with nodules of new growth. In other cases cough, dyspnœa, or hæmoptysis draws attention to the state of the lungs; and there are physical signs of the exact position of a tumour, beside evidence of fluid effusion into one or both of the pleural cavities.

They are almost always secondary. The position of the pulmonary circulation causes the lungs to be the most natural seat for all forms of secondary growth when infection takes place by the blood-current, excepting in those cases in which the primary tumour lies within the area of the portal system of vessels.

In most of these cases the breast or the stomach or some other part is known to be affected with a primary malignant growth. But the analogy of other viscera in which secondary tumours are apt to appear, such as the liver and the brain, would lead one to expect what occasionally does occur; a patient dies of the effects of growths in the lungs, and it is discovered for the first time at the autopsy that these growths were secondary to some

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primary growth in a distant organ. Such an occurrence, however, appears to be very rare. Once a young man came into Guy's Hospital under Dr Frederick Taylor with what appeared to be acute bronchitis, and died in a few days. At the *post-mortem* examination it was found that the lungs were full of sarcomatous masses, secondary to a like affection of the testis.

Primary growths in the lung are extremely rare, and even less can be said about them than about secondary growths. Cases in which there is only a single mass of considerable size, or in which one among several masses is obviously older than the rest, have often been described as examples of primary malignant tumour in the organ, starting perhaps from the cartilaginous rings of the bronchi or from the perichondrium. But it is uncertain whether the other viscera, and especially the different mucous membranes, have always been searched with sufficient care to justify the conclusion. The same doubt applies to some cases which have been recorded under the name of disseminated or miliary cancer of the lungs. One such was brought by the author before the Pathological Society in 1866. The patient was a man aged fifty, who died of an illness of two or three months' duration, but only two days after being admitted into hospital, with what appeared to be capillary bronchitis complicated with some pneumonia. At the autopsy the lungs were found full of round bodies like tubercles, but larger (some as large as hemp-seeds), and of a shining white appearance. The only growths discovered elsewhere were a few in the heart and in the liver. A somewhat similar case, in a girl of seventeen, is related by Sir Risdon Bennett; but the liver in that instance contained several very large tumours, so that the pulmonary affection was clearly secondary.

A still more remarkable form of disease is one of which an instance occurred in 1870. A man, aged thirty-six, died in Guy's Hospital after two and a half months' illness, which was attributed to damp and cold, and which appeared clinically to have been pneumonia of the right lung, accompanied with much effusion into the pleura. At the autopsy the lung was found much enlarged, nearly white in colour, but somewhat mottled, smooth, and shining in appearance, soft and cushiony to the feel, so that one might have imagined it to be generally emphysematous but for the fact that it was absolutely airless, every part of it sinking instantly when put into water. At the root of the lung there was obvious new growth, which probably was seated in the glands, but which had also involved the superior vena cava, and narrowed it considerably. Unfortunately, the lung was thrown away before any microscopical examination of it was made. But probably it was an example of a primary diffused carcinoma of the pulmonary tissue, such as is alluded to by Hertz (in 'Ziemssen's Handbuch') as "bearing a striking resemblance to grey hepatisation."

A remarkable case of multiple sarcoma of the lungs, chiefly affecting the surface, but not causing pleurisy, occurred in a woman aged sixty-four, who died in Miriam Ward, November 29th, 1886. There were malignant growths in the vertebræ, liver, and kidney, and a mass of diseased glands in the neck. These were taken for tubercular glands, and the pulmonary symptoms ascribed to phthisis by the writer, under whose care she was for a day before her death: there was considerable caseation of the bronchial lymph-glands; but the true nature of the case was chronic lympho-sarcoma, lasting at least eighteen months, and then becoming generalised. One point worthy of note was a rise of temperature from 99° to 105·8° F.

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THROMBOSIS AND EMBOLISM.—Thrombosis implies the spontaneous coagulation of the blood in a blood-vessel during life; embolism the carrying away of a clot to a distant vessel, which thus becomes blocked.*

Coagulation may occur under various conditions and from many causes. The problem presented by this remarkable process is one of the most difficult of physiology. Hunter and Hewson laboured at it, and in more recent times Buchanan, Alex. Schmidt, Hammarsten, Bizzozero, and Wooldridge have followed in the same path; but the exact nature of the change which transforms the soluble or suspended proteids of the blood-plasma into the insoluble and precipitated proteids of the clot, the part taken by the paraglobulin, the ferment, the leucocytes, and the blood-plates, all still await complete exposition. In practical medicine we are still compelled to follow purely empirical observation in this important subject.

Anything which renders the blood-current slower and brings it nearer to stagnation will favour its coagulation. The constant antecedent of coagulation while the blood is flowing is some change in the intima of the vessel; whilst this is smooth and in a state of vital integrity the blood remains fluid, but as soon as the endothelium is altered in structure, or any part of it removed, coagulation will take place. The effect of retardation of the blood-current is seen in the case of coagulation in the iliac veins of persons dying of phthisis and other wasting disorders, and the effect of disease of the intima in thrombosis within aneurysms of the arteries. There are, probably, other causes which favour coagulation, inherent in the blood itself. For example, thrombosis is often met with in diabetes, gout, the febrile and particularly the puerperal state. In these cases coagulation begins in a small vein, and then proceeds upwards, sometimes reaching to the vena cava.

The clot may set up inflammation of the lining of the vessel, endarteritis or endophlebitis. The clot then becomes organised and the vessel obliterated. Occasionally it may shrink and allow a passage of blood through it again. In other cases the clot softens and is changed into a milky or pus-like material.

Formerly it was supposed that suppurative phlebitis caused the clot, and that pus mingled with the blood, producing "pyæmia;" but we now know, owing to the researches of Goodsir and Virchow, that the leucocytes of a thrombus are white blood-corpuscles, not pus-cells; and that they are less numerous than was supposed (compare vol. i, p. 62). When pus does enter a vein, *e. g.* from rupture of an abscess, it sets up adhesive phlebitis, which usually seals it up, and the clot spreads thence towards the heart.

Phlebitis may be therefore the result as well as the cause of thrombosis. In the same way endocarditis will set up local thrombosis, particularly when the current of blood is slow.

Septic, *i. e.* bacterial thrombosis, produces ulcerative phlebitis, arteritis, or endocarditis; and septic inflammation of the heart or vessels produces very abundant thrombosis (p. 57).

True suppurative phlebitis is probably always the effect of septic thrombosis. We have seen a typical instance of it in disease of the internal jugular vein from disease of the temporal bone, and we shall meet with an equally clear example in suppurative pylephlebitis of the liver.

* Embolus (ἔμβολος, anything thrown in, a plug or stopper) was a term invented by Virchow to denote the transferred clots discovered by Kirkes and by himself.

Thrombus (θρόμβος) is classical Greek for a clot or curd, *coagulum*.

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Cases of thrombosis become most important when the coagulation reaches the iliacs or vena cava, for then, should a portion of the clot become detached and carried to the heart, and so on to the pulmonary artery, almost sudden death ensues. The most common example of this occurs in puerperal cases, when a clot has formed at the commencement of the vena cava by extension from the iliac veins. Some inflammatory process having occurred about the neck of the womb, coagulation takes place in the uterine sinuses and extends through the iliac veins to the inferior cava. This may have occurred with so very few symptoms that no suspicion of the coming fatal event is excited. It is usually about a fortnight after delivery, on the patient's rising from bed, that a portion of the fibrinous clot becomes detached, passes into the pulmonary artery, and causes almost instant death. If the clot should be small, and plug only one pulmonary artery or a branch, death may be postponed for an hour or two. In other cases the detached portions are still smaller, and not being large enough to close the trunk of the vessel, stick in some of the branches, and there produce further coagulation. This proceeds until the whole lung is blocked, and a fatal termination ensues in a few days. Under these circumstances it is, however, possible, though very rare, for the patient to recover (*cf.* p. 125).

Next to pulmonary embolism the most important and frequent cases of clots being carried free in the circulation are those met with on the arterial side of the system. A clot may form, for example, in an aneurysm, or in the left auricular appendix, or vegetations may become detached from the valves of the heart, and are then carried into the arterioles of various parts of the body. There may be sudden blocking of the vessels of the limbs leading to gangrene. For example, a boy under treatment for cardiac disease was suddenly seized with pain in the arm, which on examination was found to be pulseless; a man was attacked without warning by most excruciating pain in the leg, and the limb was found cold and with no pulsation in the arteries. It is remarkable that blood-vessels, which are regarded as insensible organs, should evince the most acute sensibility when subject to such stretching as occurs in embolism.

One of the subsequent events which take place in the vessels so blocked, if the clot is septic, is the formation of aneurysm (*vide supra*, p. 104).

When numerous septic clots are detached and carried by the circulation to all parts of the body, as in ulcerative endocarditis, local effects are not so manifest, but the blood becomes infected, symptoms of a typhoid or pyæmic character are set up, and the malady, if fatal, may be protracted through several weeks. In these cases the arterioles in various organs become blocked, leading to very characteristic formations in the organs, known as "infarctions." The vessel having become blocked and the blood flowing back into the emptied tissue, coagulation occurs in these arterioles; and if exudation takes place from them, a mass resembling a portion of fibrin is formed. Such "wedge-shaped masses" are met with at the edges of the lung, kidney, or spleen, in the midst of which the remains of the tissue of the organ may still be seen. They were described by Hodgkin in 1829, and afterwards by Kirkes and by Virchow. These may soften and so lead to further infection; or they may dry up and leave a cicatrix; or in the case of the lung they may become a focus for an inflammatory process.

The origin and pathology of ulcerative endocarditis has been already described (*vide supra*, p. 56). Instances of sudden death from pulmonary

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embolism need no illustration; and we have considered at length the effect of embolism and thrombosis of the cerebral arteries and of thrombosis of the cerebral veins (vol. i, pp. 459, 675). But cases of slow coagulation in the lungs with more lingering symptoms are not so common, and therefore an example may be given.

A medical man, fifty years of age, was seized on the 3rd of March with difficulty of breathing, and on the following day he was carefully examined by Dr Wilks. His shortness of breath was of that kind so frequently seen in cardiac disease: the patient was panting and breathless, the respiration was very quick, and on the slightest exertion it almost ceased, as happened more than once when he attempted to move out of bed. On examination of the chest nothing abnormal could be discovered; the sounds of the heart were healthy, its action quiet, and the pulse was 80. The patient had no difficulty in taking a full breath; indeed, he felt, he said, as if he must breathe too much. A suspicion of embolism caused an examination of the whole body, when it was found that he had had phlebitis of one leg, arising from an injury six months before. The patient also said that a month ago he was seized with shortness of breath, which passed off after a few hours. Dr Wilks concluded from this that he had embolism of the pulmonary artery. On the following day he was no better, and on March 6th Dr Fagge saw him, but could discover no physical signs of disease in the chest. On March 7th he was worse, and was beginning to spit up tenacious, rusty, and bloody mucus. On March 8th he was dying, gasping for breath and with great lividity of the extremities, but the heart was regular and there was no bruit. The autopsy showed embolism of both branches of the pulmonary artery. In the left was a large clot, tolerably recent, and in the right an older one adherent to the walls of the vessel. There was also pneumonic consolidation in portions of both lungs. The right femoral vein contained a thrombus of exactly the same character as that found in the pulmonary artery.

Cases of recovery from such pulmonary embolism are still more rare.

A young man, an officer in the army, was operated on for varicocele; he rapidly recovered from the operation, and had left his bed, but had not gone out of the house. During conversation one evening, February 9th, a fortnight after the operation, he suddenly called out, fell back, and gasped for breath. It was thought he was dying. A medical man saw him soon afterwards and found him cold, pulseless, and breathing heavily. About an hour afterwards he spoke; he was very pale, and the respiration was sighing, but it was thought that his pulse was perceptible. He lay all night quiet and calm, but pale, and the pulse was only just to be felt. On the following morning he had rallied, his skin had become warm, the breathing was tranquil, and the pulse 100. February 11th.—Some oppression of breathing with little pain; temperature normal. 12th.—Slight crepitation heard over lower part of chest on left side; heart normal. 13th.—Lying quietly, fills chest well and deeply. Crepitation over lower part of left lung in front and expectoration of a little bloody mucus. A slight murmur heard at base of heart. 14th.—Bruit more audible and traced up in course of pulmonary artery. 16th.—Bruit less marked and crepitation diminished. 20th.—Bruit gone and no râles heard, but in the spot where they existed respiration imperfect. He continued to improve, but spat up red, glairy, transparent mucus, and when he sat up, the pulmonary bruit became audible. March 15th.—Allowed to leave his bed, all physical signs having disap-

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peared; but he still spits a little coloured mucus. He was kept quiet for another month, when he was allowed to go out, and gradually recovered.

Two cases of recovery from pulmonary embolism have come under the present writer's notice: one in a patient seen with Dr. Hine, of Oxford, where it followed thrombosis of the femoral vein from an injury; the second during recovery from pneumonia, in a healthy man under the care of Dr J. H. Galton. In both cases the condition was recognised from the first, and in both the diagnosis was amply confirmed.

EXOPHTHALMIC GOITRE.*—The most remarkable functional disorder of the circulation is that which is characterised by the combination of palpitation of the heart and throbbing of the arteries, with staring eyeballs and an enlarged thyroid body. In Germany such cases are spoken of as examples of "Basedow's disease," because von Basedow in 1840 published a paper on the subject. The clinical picture had, however, already been described by Graves, of Dublin, in his 'Lectures' (originally published in 1835, according to Stokes, who himself recorded one of the earliest cases); and therefore Trousseau named it "Graves's disease." Still earlier records of the same affection have been found in the writings of Adelmann, and of Caleb Parry, of Bath (1825); but Flajani (1798) did not refer to this affection at all according to Dr Beigell ('Reynolds' System,' vol. v), who shows that Graves first described a typical case and saw its clinical significance.

Origin.—The complaint generally sets in very gradually. As a rule no definite cause can be found for it; but sometimes it has been traceable to a severe mental shock, and it may then develop itself rapidly. Trousseau speaks of a lady who, having one night been crying for a long time on account of her father's death, "suddenly felt her eyes swell and lift up her eyelids," while at the same time she had copious epistaxis, violent palpitation of the heart, and throbbing and enlargement of the thyroid; four days later the nature of the case was recognised.

Graves's disease occurs chiefly in young women, but a patient who died of it at Guy's Hospital in 1868 was a woman of fifty-eight, and Stokes saw it in a lady upwards of sixty years of age. It is said to have been observed in boys of fourteen and less, in a girl of seven, and in another girl only two and a half years old. The proportion of females to males is stated by von Graefe as six to one, by Eulenburg (in 'Ziemssen's Handbuch') as at least two to one; Trousseau's cases give it as fifty to eight, Henoch's as twenty-three to four, and Praël's as twenty-eight to one.

Persons who are anæmic or chlorotic are said to be especially liable to exophthalmic goitre; and Stokes mentions the case of a man in whom long-continued bleeding from piles apparently caused it. It frequently occurs in hysterical women and in neurotic subjects, including epileptic patients and lunatics. An instance is recorded of it in a boy, aged eight, whose mother is said to have had the same disease. And a man, aged twenty-six, whose portrait was given by Dr Wilks in the 'Guy's Hospital Reports' for 1870, had an uncle and a brother who were affected with ordinary goitre. The disease is never endemic, and appears to have no relation to cystic bronchocele.

Symptoms.—Of the three cardinal symptoms the first to be observed is commonly an *increased action of the heart.* This may for a time be only occasional, but afterwards it is constant. The number of beats is much

* *Synonyms.*—Maladie de Graves—Basedow'sche Krankheit—Cachexia exophthalmica.

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augmented, reaching 120, 140, or even (it is said) 200 in the minute. The cardiac impulse is exaggerated, the sounds are loud and ringing, and a blowing systolic murmur is occasionally audible at the base or, still less often, at the apex. The carotid arteries throb, and the hand feels the pulsation of the enlarged thyroid, often with a well-marked thrill. The beats of the radial arteries, however, are not unduly forcible. Trousseau, in opposition to Aran, affirms that there is no increase in the area of absolute cardiac dulness, and the author can confirm this statement from personal observations. This would not of itself prove that the heart is not enlarged. But in the fatal cases that have occurred at Guy's Hospital the organ has weighed only nine, ten, or once perhaps eleven ounces. It is true that each patient was much emaciated, so that a degree of relative hypertrophy may be said to have been present. Although no cardiac bruit is to be heard in uncomplicated cases, when anæmia is considerable a systolic murmur may be sometimes audible in the subclavian or the pulmonary artery. Atheroma of the aorta has been occasionally seen as an effect of the increased strain upon that vessel.

Swelling of the thyroid may sometimes be observed before the exophthalmos, but usually not until afterwards. It is sometimes symmetrical, sometimes more marked on one side, and usually on the right. In some cases it is but slight, so as hardly to deserve the name of goitre, and it never approaches in size to the huge tumours which sometimes occur in the endemic form of bronchocele. But it may cause a considerable projection of the throat, and it may press on the trachea, so as to alter the voice and to compel the patient to lie with the head thrown back upon a pillow. In Dr Wilks's case, already referred to, the lower part of the left lobe was found after death to extend down into the chest, altering the shape of the trachea, and perhaps compressing the thoracic duct. In an early stage of the disease the thyroid body sometimes undergoes rapid variations in size, becoming larger when the heart's action is more disturbed, or even under emotional excitement. This of itself shows that the state of the gland is partly one of vascular turgescence, and its vessels have been found increased in diameter, the arteries especially being tortuous. Its tissue may be perfectly normal in appearance, or it may contain "colloid cysts," sometimes of considerable size.

Prominence of the eyeballs varies greatly in degree. One patient merely seems to stare a little more than natural, another has a fierce expression, from a wide space left between the corneal margin and the edges of the eyelids. It is said that the points of insertion of the recti muscles into the sclerotic may be visible; and Trousseau alludes to a case in which one of the eyes "actually came out of the orbit and had to be pushed back by the fingers." Sometimes the protrusion is more marked or begins earlier on one side than on the other; and according to certain observers it may be permanently unilateral.

A symptom described by von Graefe is that, when the patient looks towards his feet, the upper eyelid fails to descend by an associated action, as it normally should do. He showed that this is by no means a necessary consequence of the exophthalmos; for when a tumour of the orbit causes the eye to project, the movements of the eyelid remain unimpaired. In one patient whom he saw, and whose complaint was of palpitation of the heart, the symptom in question constituted the sole ground for regarding the case as one of Basedow's disease. Eulenburg, however, has found "von

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Graefe's symptom" absent, or almost absent, when there was great protrusion of the eyeballs.

When the exophthalmos is extreme, the eyelids may fail to meet during sleep; the cornea is then apt to become inflamed, and even to slough. As a rule, patients affected with exophthalmic goitre see perfectly well, but sometimes they notice muscæ, or complain of fatigue in using the eyes. The only ophthalmoscopic appearance is said to be a dilated and tortuous state of the retinal veins. The pupils have been described as dilated, but Eulenburg says that von Graefe failed to observe this in an experience extending over nearly 200 cases, and he believes that when present it has been due to the presence of myopia.

There appears to be still a doubt as to whether the protrusion of the eyeballs is entirely due to turgescence of vessels in the orbit, or partly to an overgrowth or swelling of the fat. A third hypothesis is that it may in part be caused by contraction of Müller's non-striated orbital muscle. Exophthalmos has sometimes been noticed to increase or diminish as palpitation became more or less severe, and to vary with the menstrual periods. In some cases, but not always, the eyes are scarcely if at all prominent after death. The recti muscles have twice been found in a state of fatty degeneration, but this is attributed to disuse or to the stretching which they had undergone.

Other symptoms which have been noticed in patients suffering from exophthalmic goitre are irritability of temper, sleeplessness, headache, impairment of memory, and unfitness for employment. Muscular tremors were observed by Dr Hector Mackenzie in the majority of twenty-eight cases; this symptom was first recognised in France by Charcot and by Marie. Muscular cramp also is not an infrequent symptom. In some cases mental disturbance is associated with Graves's disease (see Dr Savage's paper in the 'Guy's Hospital Reports' for 1882); in others flatulence and constipation, anæmia, amenorrhœa, leucorrhœa, or epistaxis. Irregular febrile attacks sometimes occur, in which the temperature may rise 2° or 3° F. There is often extreme emaciation. Trousseau notes that he has obtained a *tache cérébrale*. The spleen may be felt swollen during life. Enlargement of the breasts has been mentioned, but Trousseau speaks of them as undergoing atrophy. It is perhaps worthy of note that in two fatal cases at Guy's Hospital, the patients being respectively twenty-nine and twenty-one years old, the thymus was persistent. In one it was four inches long, and had a maximum thickness of three quarters of an inch. Melanodermia and leucodermia have been observed by Trousseau, and since by many others (see Dr Drummond's lecture, 'British Medical Journal,' May 14th, 1887; and Dr H. Mackenzie's papers in the 'Lancet' for September 6th—27th, 1890).

Pathology.—We need not discuss at length the many speculative views that have been advanced to account for the phenomena of exophthalmic goitre. That no one of the three cardinal symptoms can be taken as the cause of the others is now generally admitted. Some of the earlier writers on the subject held that some cardiac affection constituted the starting-point of the disease. Stokes, for instance, believed that this was usually a persistent functional excitement of the heart; and he cited Dr Parry's cases as showing that organic lesions might occasionally be followed by similar effects. Moreover, in two fatal cases at Guy's Hospital there had been an antecedent attack of rheumatic fever, and in one of them pericarditis and

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endocarditis were found at the autopsy. But in the great majority of cases the heart is perfectly sound. On the other hand, Koeben imagined that the goitre gave rise to the rest of the symptoms by pressing upon the cervical sympathetic; but such a notion is altogether inconsistent with the fact that enlargement of the thyroid often follows the exophthalmos; moreover, much larger endemic bronchoceles produce no such consequences.

There seems to be no escape from the conclusion that the three classical phenomena of the disease are joint effects of some other cause; and a very obvious suggestion is that of Trousseau, according to which they are due to disturbance of the lower cervical ganglia of the sympathetic. Eulenburg cites eight autopsies, in each of which changes in their structure were demonstrated; and Dr Goodhart's case in the 'Path. Trans.' for 1874 may be added to them, as also Dr Shingleton Smith's ('Med. Times and Gazette,' 1878). The changes observed consisted generally in an overgrowth of the fibrous capsules of the ganglia, with or without an excess of the connective tissue in their interior, rendering them hard and tough. And it is perhaps worthy of notice that, in Dr Goodhart's case, the connective tissue of the neck and thorax also appeared to be in excess. In a few cases it is stated that there was atrophy of the nervous elements of the ganglia; but this may have been the result of reagents. On the other hand, there are two cases —one of them investigated with great care by Ranvier—in which the ganglia were found healthy. Moreover, the state of the superior cervical ganglion in Graves's disease has been recently investigated by Dr Hale White, who finds that the variations in the size and pigmentation of the corpuscles, and the amount and density of the interstitial tissue, do not vary more than in cases taken at random ('Guy's Hosp. Reports,' 1889).

In a case of Graves's disease, in a lunatic, Dr Savage, on careful microscopical examination of the cervical ganglia, found them perfectly healthy ('Insanity and Allied Neuroses,' p. 415).

Even if it were proved that there is any constant lesion of the cervical sympathetic ganglia, it would be difficult to explain how the symptoms of exophthalmic goitre could be caused thereby. Boddaert succeeded in producing enlargement of the thyroid body by ligaturing the jugular and thyroid veins in animals; and when he also divided the sympathetic nerves the eyeballs became prominent. Thus it may be that paralysis of the nerves in question, by dilating the blood-vessels, gives rise to like effects. But it is worthy of notice that the recognised effects of paralysis of the cervical sympathetic are absent (cf. 'Journ. of Phys.'). Moreover, the excited action of the heart corresponds, not with paralysis, but with irritation of sympathetic nerves. Some writers have indeed endeavoured to account for all the phenomena of the disease on a theory of "irritation." But it is a sufficient objection to such a view that a primary irritation of a nerve-centre, lasting for months or years unchanged, is as yet unknown in pathology. If the disease is of nervous origin at all—which certainly seems at present most likely—its seat is probably to be sought, not in the sympathetic ganglia, but in the spinal cord, or the bulb.

Not infrequently we find valvular disease of the heart, usually mitral but sometimes aortic, associated with Graves's disease; but this has always an independent origin.

After death, in uncomplicated cases, the heart is found normal, both in the valves and the muscular tissue, and the thyroid much reduced in size,

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while the eyes have usually returned to their normal position, so that a case might easily fail of recognition.

The spleen is sometimes found enlarged, as by Basedow, Sir Henry Marsh, and the elder Dr Begbie, who published a careful account of the then new disease of Graves and Stokes in 1849. His son, Dr Warburton Begbie, records two cases of the same condition, and quotes Hensinger to the same effect ('Edin. Med. Journ.,' September, 1863, and April, 1868).

Autopsies on cases of exophthalmic goitre are so rare that this deserves mention; but the size of the spleen varies greatly, and of three fatal cases of the writer's, the spleen was large in one and small in two.

With regard to the *diagnosis* of exophthalmic goitre little need be said. But it is important to note that one must be on the look-out for slight cases and for rudimentary forms, in which one or other of the cardinal symptoms may be absent. This is a point on which Trousseau insisted; and Dr Wilks has recorded several instances which might have been set down as examples of ordinary chlorosis but for the failure of ferruginous medicines to cure them.

Event.—Exophthalmic goitre is not often traced to a fatal termination in hospital experience. We have indeed made six or seven autopsies at Guy's Hospital since 1868; but the only case in which death appeared directly due to the disease was that of which Dr Wilks has recorded the details; and in this there was bronchitis with expectoration of mucus tinged with blood. Two (or perhaps three) patients died of pleurisy, with or without pneumonia; one of rheumatic pericarditis and endocarditis. One patient succumbed unexpectedly, after having been ailing for a day or two; the stomach and intestines were found to be affected with a remarkable form of follicular inflammation, the solitary glands and Peyer's patches being very prominent, and the whole mucous membrane intensely injected, swollen, ecchymosed, and lined with mucus. In the remaining case death was caused by the inhalation of an anæsthetic.

In all but two of these cases the disease was of recent origin, having lasted only a few months. One patient had suffered for four years. It does not appear that those affected with exophthalmic goitre, like so many with other chronic and incurable maladies, go about from hospital to hospital until they die. The only possible inference seems to be that most cases at length end in recovery; but it is difficult to obtain positive evidence in support of this conclusion. The duration of the complaint is too long, and the natural process of cure is too gradual, to allow of its being traced in a ward of which the inmates are constantly changing. Dr Hale White has, however, followed up twelve patients previously under treatment in Guy's Hospital, and found that four were well after illnesses of two, three, five, and eight years; and that one was much better. Seven were dead—two suddenly, and no cause was found *post mortem;* one from mitral regurgitation, one from abscess in the axilla and pyæmia, one from gastric ulcer, one from phthisis, and one from unknown causes ('Brit. Med. Journ.,' July 24th, 1886).

Treatment.—The treatment of exophthalmic goitre is unsatisfactory. Trousseau was strongly convinced that iodine was generally injurious, although he admitted that it was sometimes useful. He also believed that the tincture of iron did harm, and most observers will at least admit that it seldom if ever does good. Traube, however, is said to have given iron and quinine alternately, each for three weeks at a time, with great advan-

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tage. Digitalis, though recommended by Trousseau, seems to have no power whatever of slowing the pulse or tranquillising the heart. In two cases ergot was prescribed, but without marked benefit. Strophanthus is said to be somewhat more effectual.

Belladonna has been frequently given, and some cases of more or less benefit from its use have been recorded; but more often its effects are insignificant, or at best transitory. Dr Russell Reynolds believes that real benefit may be obtained by the exhibition of iodide, bromide, and iron in combination ('Lancet,' May 17th, 1890).

Some German physicians have applied galvanism to the neck with the object of "galvanising the sympathetic nerves." A battery of six or eight cells is used; the negative pole is placed upon the spine below the fifth cervical vertebra, the positive pole at different levels in front of the sterno-mastoid muscles. In this way the pulse was brought down from 130 to 70 or 80, and the general condition of the patient is said to have been much ameliorated. The application of a full current to the closed eyelids is also recommended, as tending to diminish the exophthalmos.

Unfortunately galvanic treatment has been adopted elsewhere with little advantage.

Other tumours of the thyroid are rather of surgical than medical interest. If innocent, they are either cystic, or solid and fibrous; if malignant, they are usually sarcoma.

The endemic form of bronchocele has already been referred to in connection with endemic cretinism (cf. *supra*, vol. i, p. 860).

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DISEASES OF THE ALIMENTARY TRACT

AFFECTIONS OF THE MOUTH AND SALIVARY GLANDS OF THE THROAT AND OF THE GULLET

Τοῖσι μὲν σμικροῖσι καὶ νεογνοῖσι παιδίοισιν, ἄφθαι.—HIPP., *Aph.*, iii, 24.

"In anginam ego me nunc velim verti,
Ut veneficæ illi fauces prehendam."
PLAUT., *Most.*, i, 3, 61.

STOMATITIS—*Vesicular ulcers of the tongue and lips—Single ulcer of the palate—Phagedænic ulcerative stomatitis—Gangrenous stomatitis or cancrum oris—Thrush—Aphthæ—Ptyalism—Xerostomia.*
MUMPS—*its pathology, symptoms, and complications—Metastatic parotitis.*
ANGINA—*Catarrhal, ulcerative, and herpetic forms—Subacute follicular tonsillitis—Acute tonsillitis or quinsy—Hypertrophy of the tonsils—Granular pharyngitis—Adenoid growths of the pharynx.*
STRICTURE OF THE ŒSOPHAGUS—*Minor disorders—Spasmodic stricture—Simple stricture—Cancerous stricture—Anatomy, course, and treatment.*

THE diseases of the alimentary canal vary more than those of the chest in their pathology. Some might be properly classed with the specific fevers, some are due to malaria or to parasitic animals, others to disorders of the nervous system, while others again are, as far as we know at present, purely functional. Nevertheless it is most convenient to consider them together, and the only classification attempted will be that of local distribution.

We will begin with the diseases of the alimentary canal above the diaphragm.

AFFECTIONS OF THE MOUTH.—*Simple or herpetic ulcers.**—Among the trivial affections to which children, and sometimes adults, are liable, is the formation of minute, shallow, round or oval ulcers in the mouth. They begin as raised white spots, looking like vesicles. In a few hours the roof is lost, apparently as a consequence of maceration in the buccal mucus and saliva. The ulcers which result have an ash-grey or yellow surface and a bright red border; they are as large as a pin's head or rather larger, painful, and very sensitive to the contact of particles of food, especially sugar and salt. A favourite seat is inside the lower lip, especially where it joins the gum; they also occur upon the lining of the cheek or upon the tongue. Some persons are more liable to these ulcers than others, and are long troubled with them at intervals of weeks

* *Synonyms.*—Aphthous ulceration of German and of some English writers—Follicular, vesicular, or herpetic ulcers—Ulcera mitia familiaria—Herpes oris.

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or months. Their presence is attributed by children to "telling stories," and by mothers to a "disordered stomach;" their true cause is unknown. In a day or two these ulcers heal of themselves without treatment, but touching them with a stick of lunar caustic removes the pain at once.

Single ulcer of the palate.—In marked contrast with this affection is one which is described by Vogel (in 'Ziemssen's Handbuch') as occurring in weakly infants, especially those brought up in lying-in or foundling institutions. It consists in the formation of a single flat ulcer at the back of the hard palate, just where the *velum* joins it. There is not usually any tendency to spread deeply, but neither is there any disposition to heal, and the ulcer remains until the child's death, which usually results from diarrhœa.

*Phagedænic ulcerative stomatitis.**—Another form of ulceration of the mouth is a disease of considerable gravity, attended with great fœtor of the breath. The ulceration chiefly affects the gums, the edges of which become reddened and swollen, are detached from the teeth, and finally seem to break down into a grey pulp. So complete may be the destruction that the alveoli are sometimes exposed, and the teeth become loose and fall out. The whole of the lining of the cheeks and lips becomes the seat of ash-coloured ulceration. The tongue is large and doughy-looking, marked at its edges by the teeth, thickly furred or ulcerated on the surface. A large quantity of acid fluid escapes constantly from the mouth, running out upon the pillow while the patient is asleep. All movements of the mouth are very painful, and food is taken with much difficulty.

The chief instances of this affection among the patients of Guy's Hospital have occurred in children between the second dentition and puberty; once, two sisters came with it at the same time. Among the soldiers of the French army it is stated to be frequent, occurring epidemically when they are overcrowded in close quarters. It probably depends on some local infective microbe, but this has not yet been ascertained. Bergeron, who described the disease in 1859, is said to have inoculated himself successfully with it in the lower lip.

In a milder form it is not very uncommon both in children and adults. Ulceration with spongy and bleeding gums without other signs of scurvy is ascribed by dentists to the fluids of the mouth being acid instead of alkaline, and the writer has found this to be verified by test-paper in some cases of the kind in children.

The remedy for ulcerative stomatitis is chlorate of potass, which may be given in ten-grain doses at frequent intervals, dissolved in water. Or lozenges containing the salt, or Wyeth's compressed tablets, may be used with the object of securing its local action upon the mucous membrane, although this appears after all not to be so essential as its absorption into the blood. It is surprising in how short a time the affection is brought to an end; within three or four days the diseased parts begin to show a clean, healing surface. An example of this action in an adult was given by the writer in the 50th volume of 'Virchow's Archiv' (1870), p. 462, where, under the title "Gingivitis," three cases of ulcerative stomatitis are related.

Gangrenous stomatitis† is a disease almost entirely confined to young children. It differs from the last-described affection in the fact that ulceration, or even phagedænic stomatitis, begins in the mucous membrane, whereas noma

* *Synonyms.*—Phagedænic gingivitis—Putrid sore-mouth—Stomacace, a French term, "mouth-ill," often applied to scurvy.

† *Synonyms.*—Noma—Cancrum oris.—*Germ.* Wasserkrebs—Wangenbrand.

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begins as a slough in the submucous tissues, commonly in the cheek or the lower lip. Its cause is unknown, but it often follows measles or other exanthems. Its treatment consists in destroying the diseased tissue by strong nitric acid or other surgical means, and in supplying stimulants freely.

*Thrush.**—In this country it has been usual to apply the term *aphthæ* to a condition of the mouth altogether different from that which general practice on the Continent has named *aphthous ulceration* (see last page). Confusion may be avoided by using the vernacular term "thrush" for the former affection, which we will now describe.

The earliest indication that thrush is setting in is a change in the mucous membrane lining the cheeks and other parts of the mouth; it becomes redder than natural, hot, and painful. Soon a number of minute milk-white spots appear upon its surface. These rapidly increase in size, and run together; and in a day or two the whole surface may be covered with a nearly uniform adherent layer. At first there is some difficulty in detaching the white material; but after a time it becomes loose, and can be peeled off in large flakes without any bleeding. Microscopically it consists of layers of squamous epithelium, mingled with the spores and mycelium of a fungus.

This fungus is commonly known as the *Oidium albicans*, although, according to Hallier, it is not really distinct from the *Oidium lactis*, which is the active agent in the souring of milk. It must not be supposed that the presence of the oidium in the human mouth is peculiar to cases of thrush. It has been found upon diphtheritic membranes, and in portions of fur taken from the tongue. Nevertheless the essential cause of thrush is a vigorous and rapid growth of this fungus, leading to inflammation of the mucous membrane and detachment of epithelium, just as the fungus of ringworm produces a red and scaly condition of a part of the skin upon which it grows. A necessary condition, however, before the oidium can germinate actively in the mouth appears to be an acid state of the buccal secretions; and according to Vogel the mouth always gives an acid reaction at the very commencement of the affection, before any white spots are visible. The preponderance in infants of mucus, which readily turns acid, over the alkaline salivary fluids is perhaps the reason why thrush is so much more common in them than in older children.

In many cases thrush comes and goes with little disturbance of the health, and without any danger to life. But in others it is associated with severe and even fatal diarrhœa. The popular notion is that the disease passes through the child and comes out at the anus. Although it is true that the œsophagus is sometimes affected in its entire length, there is no reason to believe that the *Oidium albicans* is capable of germinating on any surface which is not covered with squamous epithelium. Thus it never enters the nose or the larynx; but it does sometimes appear in the lowest portion of the rectum and upon the female genitals, and it may also be seen upon sore spots on the skin of the face and neck. The relation of thrush to diarrhœa in young infants probably is that they are both effects of a weakly state of health from bad feeding or from some other cause. In these cases there is usually superficial dermatitis (or "eczema") of the nates, which must be carefully distinguished from a syphilitic rash.

In adults, thrush rarely occurs except in those who are reduced to a state

* *Synonyms.*—Aphthæ (ἄφθαι), a good Greek word, and used by Hippocrates probably rather in the English than the German sense.—*Fr.* Muguet.—*Germ.* Soor.

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of extreme marasmus by a chronic malady (such as consumption or cancer), or who have passed through several weeks of pyrexia from enteric fever, or puerperal fever, or pyæmia. Thrush in adults is generally taken as warranting an unfavourable prognosis; but patients may, nevertheless, recover after having had thrush, as we have often observed.

In the treatment of thrush, all that is necessary is to wash out the mouth at frequent intervals with a weak solution of an alkaline carbonate or of borax, or to apply the *glycerinum boracis* freely to the surface of the mucous membrane.

Affections of the Salivary Glands.—*Salivation or ptyalism.*—In patients submitted to active mercurial treatment the mouth is very apt (unless the teeth are kept scrupulously clean and the gums well washed with alum, borax, or tincture of myrrh) to present an affection identical with that which was above described as "ulcerative stomatitis." But in addition there is also an extraordinarily profuse flow of saliva, the condition known as ptyalism or salivation. Salivation may occur independently of the administration of mercury, sometimes as a result of the action of other drugs (as iodide of potassium or pilocarpin), sometimes from irritation starting in distant organs (as the uterus or the stomach); sometimes it is apparently spontaneous or idiopathic. But such cases are few in comparison with those that are due to the medicinal use of mercury. The quantity of saliva that is poured out is sometimes astonishing. The usual daily amount is one or two quarts; but as much as five quarts are said to have been collected in extreme instances. The patient is incessantly spitting it out of his mouth, or allowing it to dribble into a spit-pot, which he keeps constantly by his side; at night it saturates his pillow. It is more or less viscid or glairy in consistence, and is said to have sometimes a specific gravity as high as 1059; but as the case goes on its specific gravity falls till it is scarcely above that of water. It contains little sulphocyanide of potassium, and less ptyalin.

Mercurial salivation is now seldom seen. In administering mercury one watches the patient's mouth carefully, and the medicine is at once stopped when any disagreeable odour is perceptible in his breath, or when his gums become in the least degree inflamed, or his teeth tender on pressure. Or, if there should be special reason to anticipate the onset of salivation from the fact that one is using mercurial inunction, or calomel vapour baths, or full doses of blue pill instead of minute doses of the bichloride, one may often obviate it by giving at the same time chlorate of potass. When once salivation has developed itself chlorate of potass appears to have little direct influence upon it, although it more or less quickly brings the gums and the mucous membrane of the mouth into a healthy state. The ordinary duration of salivation is from one to three weeks. While it lasts the patient generally becomes thin. His urine is scanty and his bowels are constipated. Relief may be given by washing out the mouth with astringent solutions, as of alum or gallic acid, to which some tincture of myrrh and tincture of opium may be added.

Xerostomia.—In the 21st volume of the 'Clinical Transactions' Mr Hutchinson and Dr Hadden described independently two cases in which there was "deficiency of the salivary and buccal secretion," causing a permanently "dry mouth." Both patients were widows about sixty years of age, and a third case recorded by Dr Rowlands ('Lancet,' Jan. 14th, 1888)

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also occurred in a woman of sixty. The condition had in two of these cases begun suddenly and after nervous emotion, and it seems probable that the secretory, without the vaso-motor, nerves are primarily affected. In Dr Hadden's case tincture of jaborandi was exhibited with relief.

MUMPS.*—The salivary glands, like the pancreas, are very little subject to the diseases which affect the liver and kidneys, or the mamma and testes. The most frequent affection is one which, although it looks like a mere local inflammation, is really the expression of a specific disease, transmitted by contagion, occurring epidemically, and possessing the power of protecting against its repetition in the same individual. For this disease the popular name of Mumps is the most distinctive.

It is endemic like measles and whooping-cough, but local epidemics may often be recognised. This was the case in the American war of 1862–3.

The earliest symptom is commonly an aching pain in the parotid region on one side, increased by every movement of the jaw, as in speaking, or in taking food. But sometimes malaise and pyrexia precede by a day or two all local signs of the affection. Swelling very soon sets in; the hollow between the mastoid process and the jaw is filled up, its place being taken by an ill-defined projection, which throws outwards the lobule of the ear, and extends over the cheek towards the angle of the mouth, and downwards some distance into the neck. The submaxillary glands usually follow, and a little later the opposite side participates in the disease; so that in the course of from three to six days the whole of the face becomes surrounded by an immense mass of firm doughy infiltration. There is an enormous double chin, and the natural contour of the throat is lost. The skin over the affected parts is either slightly reddened, or pale and waxy-looking. Internally the swelling extends to the tonsil and the pharynx. The movements of the jaw are greatly impeded. The teeth can with difficulty be separated so as to admit the end of a spoon between them; the patient is obliged to confine himself to a fluid diet, with eggs, custards, jellies, and other things that require no mastication.

Sometimes the saliva is deficient, sometimes it is secreted in excess; but as a rule its quantity and quality are unaffected. The head is kept fixed in one position, with the face directed straight forwards. If, as sometimes happens, the affection remains confined to one side, the head is turned rather towards that side. Pain and tenderness continue more or less severe. Sometimes there is hardness of hearing, or the patient complains of shooting pains in the ears or of a continuous ringing sound.

On about the fourth day the pyrexia ceases. It seldom runs high, but temperatures of 104° are now and then recorded, and typhoid symptoms have been occasionally observed.

Soon afterwards the swelling begins to subside, and absorption takes place so rapidly that within three or four days it entirely disappears. Thus the whole duration of the disease is usually a week or ten days, rarely a fortnight. There is sometimes desquamation of the cuticle over the affected parts.

Natural history.—Mumps, like other specific diseases, protects from itself. But sometimes, when one side has been affected, the other follows suit within a week or fortnight, or there may be a relapse affecting both

* *Synonyms.*—Parotides—Cynanche parotidæa (Cullen)—Parotitis epidemica.—*Scottice*, The Branks.—*Fr.* Les Oreillons.—*Germ.* Ziegenpeter.

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sides. Apart from these relapses, cases of a second attack are occasionally met with: in one known to the present writer, a boy had mumps three times during his school life.

Mumps is most apt to occur in children from ten to fifteen years old, but it is not very uncommon in adults. It is said to affect more boys than girls. In a Russian epidemic in 1885 there were 170 male and 90 female patients. The secondary orchitis is most often seen in boys about the age of puberty and in young men.

The contagion of the disease is supposed to be transmitted by the breath. The length of the period of *incubation* is variously stated by writers; it seems to be generally about a fortnight, rarely less than twelve or more than seventeen days; but it may range from six to twenty-two days.

Complications.—In some cases, as the inflammation of the face and neck passes off, or even after it has ceased, the patient is attacked with acute "metastatic" affection of one *testicle*, generally (it is said) the right. The gland becomes swollen and painful, and there is sometimes effusion into its serous sac, with œdema of the corresponding side of the scrotum. After a short interval the other testicle may be attacked in its turn. When this complication occurs there is usually a return of the pyrexia.

This secondary orchitis is now and then accompanied by symptoms of the most alarming character, though apparently without real danger to life. High fever and delirium are sometimes present. In one case related by Trousseau a condition of collapse was suddenly developed; in another the patient fell rapidly into a typhoid state. What rendered the diagnosis of the second case the more obscure was that the initial attack of mumps had been so slight and transient that no notice had been taken of it, and nothing could be learnt about it until consciousness returned.

Some years ago the writer saw a case of orchitis from mumps in which the pulse became extraordinarily slow and remained so for several days; the temperature also fell to a low point, and the breathing was much reduced in frequency.

The inflammation of the testicle usually lasts from three to six days, and then rapidly subsides. Occasionally, however, it leads to a permanent atrophy of the organ.

No explanation seems to be at present possible of the liability of mumps to set up orchitis; we can only refer it to that correlation of distant organs, in their proclivities to disease, of which we find many other examples in pathology. Mr Stephen Paget has drawn attention to other instances of relation between parotitis and disorders of the abdominal viscera—of which, we must not forget, the testis is one ('Lancet,' April 17th, 1886, and 'Brit. Med. Journ.,' March 19th, 1887).

In girls affected with mumps it is said that the *mammæ* or the vulva sometimes exhibit a like tendency to swelling and inflammation. This fact (if it be one) illustrates a case observed by Peter, in which a young woman who had amenorrhœa was several times attacked with parotitis at what should have been her catamenial periods, while on other occasions one of the labia became swollen and painful. According to some authors, the *ovaries* may be the seat of "metastatic" inflammation after mumps. But this seems to be very doubtful.

Other complications recorded are acute inflammation of the ear, ophthalmia (rare), acute bronchitis, bubo, and urethritis.

Anatomy.—With regard to the exact seat of the morbid process in

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mumps there is still some uncertainty. It is clear that the connective tissue outside the salivary glands is largely involved in the inflammatory œdema which produces the swelling in the disease; and within the glands themselves the structure affected seems to be rather the fibrous stroma which supports the glandular acini than the acini themselves. The presence of a microbe in the blood or saliva has not been discovered.

The *prognosis* is always favourable. Even when the fever runs high and is attended by orchitis with delirium, there appears to be no real danger. Neither the inflamed parotid nor the inflamed testes suppurate, though the latter may atrophy.

The only *sequela*, beside this last rare effect of the metastasis, is deafness; and this also is not common. According to Sir Wm. Dalby, it may be either "catarrhal"—when it passes quickly away, or "nervous"—when the condition is usually permanent.

Very little *treatment* is required in cases of mumps. The patient should be kept indoors and out of the way of draughts. As has already been remarked, he can take only fluid food. Fomentations may be applied to the swollen parts.

Metastatic parotitis.—In marked contrast with mumps is an affection of the parotid gland which, instead of being a manifestation of a specific contagious disease, arises as a complication of other maladies, such as fevers, dysentery, or even local affections, such as intestinal obstruction from cancer of the sigmoid flexure, as in a case of the author's. This form of parotitis is almost always unilateral. It sometimes subsides without suppuration, but it far more often leads to the formation of an abscess, which may either point behind the ramus of the jaw, or break into the external auditory passage, or burrow down into the neck or upwards in the pterygoid region towards the base of the skull.

It is possible that this "metastatic" parotitis is the result of dryness of the buccal mucous membrane leading to an obstructed state of Steno's duct, with decomposition of the retained salivary secretion. But there is little evidence for this explanation, and if true there should be more frequent parotid abscess after fevers.

The prognostic importance of parotitis occurring in the course of a fever depends very much on the period of the disease at which it is developed. At an early stage it is of very grave significance; at an advanced stage, or during convalescence, it is a comparatively trifling matter.

Gouty parotitis has been described. It must be excessively rare (see 'Med.-Chir. Trans.,' vol. lxx, p. 217, 1887).

Affections of the Fauces.*—Systematic writers recognise a great variety of affections of the throat, all of which are attended with difficulty or pain in deglutition, and with more or less marked inflammation of the pharynx, palate, and tonsils. Some of these affections need detailed description, but of others a cursory notice will suffice. We have already spoken of the special forms of angina which characterise scarlatina, diphtheria, and syphilis.

Ordinary *catarrh of the pharynx* is sufficiently characterised as redness and slight swelling of its posterior wall and of the palate, coming on after exposure to cold. In persons who have repeatedly had attacks of catarrh,

* Sore-throat.—*Fr.* Mal de gorge.—*Germ.* Halsweh. The terms *angina* and *cynanche* are often applied somewhat indiscriminately to all inflammatory affections of the fauces.

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a "relaxed sore-throat" is apt to be of frequent recurrence; the fauces feel dry and painful (especially in the morning on first waking), but the symptoms all pass off after breakfast, when nothing is to be seen beyond elongation of the uvula, a pendulous state of the palate, and perhaps some dilated veins in the mucous membrane.

What is called *ulcerated sore-throat* is particularly apt to occur in nurses and medical students who are in close attendance on the sick, or in other persons if weakened by unhealthy surroundings. In this affection are seen on the surface of the tonsils and other parts of the fauces small white superficial ulcers, which are vaguely called follicular or aphthous, and resemble in appearance the minute yellow ulcers of the mouth and tongue described above (p. 132). There is a considerable general disturbance, the breath is foul, and the tongue furred. Wine, quinine, and tincture of iron are indicated in the treatment, and generally bring the affection to an end in a few days.

It is important to distinguish from ulcers on the tonsils certain circumscribed whitish-yellow patches, which are really masses of inspissated and protruding secretion. Patients who exhibit these patches often come for advice, complaining of a recent sore-throat. Guaiacum is often effectual in relieving such cases.

Follicular tonsillitis is the name given to what may perhaps be regarded as a slighter and sporadic form of the epidemic if not infective sore-throat just described. It is marked by diffused redness and moderate swelling of the fauces, with numerous yellow points dotted over them. These are supposed to be the mouths of mucous crypts, but of this there is no evidence, and they certainly are not swollen lymph-follicles. Probably they are vesicular at first, but they do not ulcerate. This kind of angina is always bilateral, and is not attended by the severe pyrexia of true quinsy. It is often best treated by quinine, port wine, and fresh air.

Another faucial affection that has been carefully studied by foreign observers is *herpes of the pharynx*. This consists in an eruption of opaline vesicles, sometimes few in number, but sometimes so thickly crowded together that a diphtheritic membrane may easily be supposed to be present. Sometimes it recurs again and again in the same patient; and sometimes it alternates with an herpetic eruption on the skin or on the genitals. There is no satisfactory evidence that these vesicles are due to occluded mucus, like those of the skin called sudamina. More likely they are inflammatory like those of zona.

*Quinsy.**—Among the inflammatory diseases of the fauces set up by cold, there is one that mainly affects the tonsils, and is well known by the popular name of "quinsy." Sir M. Mackenzie gives figures, based upon an analysis of 1000 cases, showing that it is far more frequent in persons between fifteen and twenty-five years old than at any other age; it is comparatively seldom seen in children, or in adults beyond the age of forty. Some persons are exceedingly liable to this affection, being attacked by it whenever they get a chill, or sometimes as the result of slight stomach disorder, or (in the case of women) of menstrual disturbance. Acute tonsillitis sometimes precedes rheumatic fever.

* *Synonyms.*—Cynanche—Acute suppurative tonsillitis or amygdalitis—Angina tonsillaris—Angina phlegmonosa. Κυνάγχη or Συνάγχη refers to the choking sensation produced by this and similar inflammations of the throat. *Angina*, from *angio*, has a similar meaning. The French *Esquinancie* and English *Quinsy* are corruptions of Cynanche.

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Not infrequently the pyrexia precedes the local inflammation by a day or two, so that in young adults the possibility of quinsy coming on should always be remembered. In many persons there is habitually a chronic enlargement of the tonsils, and upon this acute inflammation from time to time supervenes.

Quinsy appears to be rather more common during autumn than at other seasons. It is attended with an enormous increase in size of the tonsil, which forms a red, shining, globular mass, projecting into the fauces, and also distinctly to be felt in the neck at the angle of the jaw. When both tonsils are involved, they may come into contact in the middle line, being flattened and even (as Bristowe remarks) ulcerated from mutual pressure. But as a rule one side is alone affected; and sometimes, just when the inflammation is subsiding in one tonsil, the other is attacked in its turn. The uvula, the soft palate, and the pillars of the fauces all partake more or less in the swelling. The patient complains of severe pain in the throat whenever he moves his jaw, and especially in attempting to swallow. The pain may radiate to the ears. The amount of pyrexia often seems disproportionate to the severity of the local affection. Even if there is no actual rigor, the patient experiences alternate chills and flushes of heat, and complains of headache, malaise, and pains in the limbs; the pulse ranges from 100 to 120 in the minute, and is full and bounding; the temperature rises to 102°, 103°, or even 105°. The tongue is thickly furred and the breath foul.

The author once made an autopsy in the case of a young child who had died in Guy's Hospital during the previous night of suffocation as the result of severe swelling of the tonsils from quinsy. Such an occurrence, however, is exceedingly rare. The disease almost always ends favourably, either subsiding more or less rapidly, or else advancing to the formation of an abscess in the tonsil, which breaks and discharges a thick fœtid pus that is generally swallowed. This brings immediate relief to all the symptoms, the temperature falls rapidly, and the patient soon feels perfectly well. The ordinary duration of the disease is three or four days; but if the two sides of the throat are attacked in succession, it may be prolonged over a week or ten days. In some cases it is said that suppuration starting from a tonsil extends down into the neck, and burrows until it may even reach the chest. Cases also have been published in which tonsillar abscesses have eroded the carotid artery and given rise to fatal hæmorrhage. Mackenzie reports one case of suffocation as the result of the abscess breaking and filling the upper air-passages with pus.*

In the *treatment* of quinsy, sucking ice or the application of cold to the throat often gives more relief than anything else, particularly at the onset. But when an abscess is in process of formation, the course of the disease may apparently be hastened by the use of fomentations and poultices externally and by steam inhalations. As soon as fluctuation can be felt an incision should be made with a guarded bistoury. Störk remarks that the best way of detecting a soft spot is to push the parts inward with one forefinger placed at the angle of the jaw, while the other forefinger in the mouth is carefully passed down over the inflamed structures from point

* Some French writers, especially Maingault and Gubler, have maintained that tonsillitis is now and then followed by paralysis of the soft palate, like that which is so often seen after diphtheria. But one would rather be disposed to think that in such cases the specific disease was really present, for marked swelling, and even suppuration of the tonsil is by no means rare in diphtheria.

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to point. In opening a tonsillar abscess the cutting edge of the knife must be directed inwards and not outwards, lest the internal carotid artery should be wounded. Mackenzie remarks that when the patient refuses surgical interference an emetic often leads to the immediate rupture of the abscess.*

In many cases, however, it would seem that by suitable treatment quinsy may be made to abort, and the occurrence of suppuration be prevented when it would otherwise have taken place. Aconite has been recommended for this purpose, but according to many experienced practitioners guaiacum is more efficacious. Mackenzie administers it in the form of lozenges, each of which contains three grains of the resin; one of these, taken every two hours, seldom (he says) fails to arrest the disease at its first onset.

Chronic enlargement of the tonsils.—A not uncommon affection of the tonsils is chronic overgrowth of their substance. They may be as large as chestnuts, or even larger; and they are very firm and fleshy, smooth on the surface, but sometimes with cheesy or calcareous masses projecting from cavities in their interior. There is true hypertrophy of the adenoid tissue of the tonsil, the follicles increasing both in size and number. It is therefore strictly analogous to the hypertrophy of lymph-glands in delicate children.

The tonsil becomes scarred by the caseous follicles falling out as they soften, and thus the surface looks worm-eaten, and may remain so long after the swelling has disappeared and the tonsil has atrophied by the natural process of age.

The affection may exist from early infancy, or develop itself during childhood or at puberty. In most cases it subsides when adult life is reached, and it seldom persists after the age of thirty.

In children it is attended with many inconveniences; the mouth has to be kept open during sleep, and rhonchus accompanies the breathing; even a pigeon-breast may result from the obstruction to the entrance of air into the lungs. This condition often goes with a narrow and high-arched palate, with crowded teeth and a straitened passage through the nostrils. Such children snore at night and breathe through the mouth during the day, and thus constant faucial irritation is kept up. The obstruction to the posterior nares leads to breathing through the mouth, and this again aggravates the faucial swelling. There is something about the physiognomy of such patients that enables one to see at a glance what is the matter with them; Mackenzie speaks of their "open mouth, drooping eyelids, and dull expression;" and in addition to these peculiarities the voice is thick and nasal, while the act of deglutition (as in swallowing the saliva that accumulates in the mouth) is performed clumsily and with obvious effort.

Another effect of chronic enlargement of the tonsils is deafness, which is attributed to a coincident swelling of the mucous membrane of the Eustachian tube. Mackenzie says, too, that the senses of smell and of taste are often impaired; but sometimes the affection exists to a very marked extent without giving rise to any symptoms whatever.

The *treatment* may at first consist in the administration of cod-liver oil,

* The reader will remember the absurd story told of Abernethy's eating plum porridge against a patient with quinsy till he made him laugh and burst the abscess; and the Ingoldsby legend of how another case was cured by a less hilarious movement.

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iron, and other tonics, while the tonsils are every day brushed over with a solution of perchloride of iron, or smeared with powdered alum or tannin. But if there is much overgrowth of solid tissue, excision by means of the guillotine is almost always necessary. If hæmorrhage follows the operation, it is generally easily checked by making the patient suck ice; if not, Mackenzie recommends that he should slowly sip half a teacupful of a strong solution of tannic and gallic acids, containing ʒvj of the former and ʒij of the latter to the ounce of water. The wounded parts remain sore for some days; during this time the food must be soft and bland, and the use of marsh-mallow lozenges is recommended as soothing.

Granular pharyngitis.—A common affection of the back of the throat is one in which the pharyngeal surface appears dotted over with small prominences of about the size of millet-seeds. They may be either scattered, or closely packed together, or confluent into ridges. The smaller blood-vessels of the mucous membrane are dilated and tortuous. As a rule the surface of the throat is dry, there being apparently a deficiency of the normal secretions. With regard to the nature of the granulations, there appears to be still room for further investigations. It is generally supposed that they consist of enlarged muciparous glands which have taken more than their share of a process of hypertrophy that likewise affects other structures, but not to so great an extent. Störk, however, says that the mucous membrane generally is in such cases often thinner than natural, and that what characterises the granulations microscopically is the absence of a superficial stratum of epithelium, large round swollen cells lying uncovered and exposed.

Mackenzie describes an "exudative form" of the affection in which viscid mucus is seen adhering in patches to the follicles, or in which their orifices are filled with a white material resembling cream cheese in appearance. This is identical with pharyngeal herpes, and closely related to the subacute follicular sore-throat described above (p. 139).

Dr Horace Green, of New York, was the first writer to give such an account of granular pharyngitis as fixed itself upon the attention of the profession; he called it "follicular disease" and "follicular inflammation" of the throat and air-passages. It is by no means limited to those regions which are directly visible at the back of the mouth. Sometimes it spreads upwards towards the vault of the pharynx, sometimes downwards towards the larynx. Michel has recently insisted on the importance of thoroughly exploring with the laryngeal mirror every part of the fauces; for example, he says that when the rest of the surface is quite healthy, there may be a small patch of disease just behind one or both of the posterior arches of the palate; this may have been overlooked by one physician after another, and yet it may be the cause of great suffering to the patient, as is shown by touching it with a probe.

The subjective symptoms of granular pharyngitis vary widely in different cases. Probably it very often causes no discomfort at all. Sometimes, however, it gives rise to a very troublesome feeling of stiffness or dryness in the throat, to a constant need for swallowing, to a pricking pain during deglutition (especially deglutition of the fluid secretions of the mouth), to a tickling sensation compelling a frequent and often painful cough, or to incessant hawking, in the hope of getting rid of mucus, until actual retching may occur.

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In other instances the chief complaints are associated with exertion of the voice. Speaking or singing may be attended with a sense of painful effort; the patient may be obliged to stop from time to time, in the middle of a sentence even, to swallow or to clear the throat. Such cases are very common in those who earn their living by means of the voice, as in preachers and public singers, as well as in street hawkers and costermongers (cf. vol. i, p. 891).

As a rule, exposure to cold or rather talking in the open air plays a conspicuous part in bringing out and in aggravating the symptoms of granular pharyngitis. According to some writers, irritation of the fauces by excessive smoking should be mentioned among the exciting causes of the affection.

An over-sensitive state of the nervous system appears in many cases to contribute largely towards increasing the severity of its subjective symptoms; some patients appear to be almost incapable of forgetting, even for an instant, the morbid feelings which they experience in the back of the throat; they are ever flying from one medical adviser to another, and they thoroughly deserve the name of hypochondriac.

Another element in the ætiology of granular pharyngitis appears to be an inherited predisposition, a vulnerability of the fauces and pharynx. Dr Green speaks of three brothers, all clergymen, who were compelled by it to give up their official duties, and whose mother was also affected with it; and he also alludes to a large number of cases, recorded in his notes, of two or three members of a single family having been treated for it.

The age at which it is most apt to occur is from twenty-five to thirty-five, and in men more often than in women. But Mackenzie has seen it in children who were eight, six, or only three years old.

In the *treatment* of granular pharyngitis, the essential point appears to be the destruction of the granulations.

So long as no local applications were used except gargles, inhalations, and brushing over the fauces with solutions of nitrate of silver, or even with the solid caustic, it was admitted by all candid observers to be an exceedingly intractable affection, and one that often ran on for years in spite of all that was done to cure it. In fact, as gargling with fluids is commonly practised, it seldom brings them into contact with any part of the fauces behind the anterior pillars of the palate. Guinier, of Cauterets, has clearly proved, in his brochure entitled 'Étude sur le Gargarisme laryngien,' that when a person with his head thrown backwards makes fluid bubble about in the back of the mouth, while he goes on inspiring at regular intervals through the nose, the fluid is supported by the base of the tongue, the uvula, and the anterior pillars of the palate; any portion of it which passes further backwards is instantly swallowed. He has, however, shown that it is possible to teach patients to gargle in quite a different manner, and that the fluid then passes not only into the pharynx, but even into the larynx itself, resting directly upon the upper surface of the vocal cords. The directions are that the head should be slightly raised, the mouth but little opened, the lower jaw thrown forwards, so as to lift the chin. Having taken a small quantity of fluid into his mouth, the patient is to draw a deep breath through the nose, and then to allow the fluid to fall back into the fauces, while he endeavours to emit the sound of the vowel *é* (or the English *a* long). This means that the cords are brought together, and that the

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act of expiration is begun; while at the same time the epiglottis is raised so as to throw widely open the upper part of the laryngeal cavity. A bubbling sound is produced by the thin stream of air which passes outwards between the cords; this sound, as Guinier says, is quite unlike that of ordinary gargling; it resembles rather the rattle in the throat which precedes death. The act of gargling within the larynx can be continued only so long as a slow expiration is being maintained. Before a fresh breath can be taken the fluid must be thrown up into the pharynx, whence it often passes out through the nostrils. The author saw Dr Guinier demonstrate with the laryngoscope the presence of a layer of fluid resting upon his own vocal cords; and he seems to have gained a considerable amount of success in the treatment of granular affections of the pharyngeal and laryngeal mucous membranes by making patients gargle in this way with the sulphurous water of Cauterets.

Of late, however, far better results than had ever before been attained in this troublesome disease have followed the adoption of much more active local treatment. Mackenzie applies a caustic paste to each granulation separately, touching on the same day only two or three, and sometimes only one of them. It is easy to imagine that this practice must be tedious, and must in many cases cover a long space of time. But in 1873 Dr Michel, of Cologne, drew attention in the 'Deutsche Ztschrft. f. Chirurgie' to the success which he had attained in about seventy cases by the use of the galvanic cautery. Since then many other observers have adopted this method. It consists in applying a heated platinum loop to the granulations, so as just to destroy their surface; and, as a rule, the operation has to be repeated only three or four times, inasmuch as the effect is not limited to the part immediately cauterised, but extends to some distance around. There is little or no pain at the time, except when the pillars of the fauces are the parts touched by the instrument; the inflammation which follows can be kept within bounds by making the patient during the first few hours suck ice at intervals; there is nothing to prevent his continuing his usual avocations. Dr Foulis, of Glasgow, employed a small gas cautery for the same purpose; but this has the disadvantage that it must be heated before being passed into the mouth, whereas the platinum loop of the galvanic cautery is cold at the times of its introduction and of its withdrawal.

Adenoid vegetations in the vault of the pharynx.—In 1869 Dr Wm. Meyer, of Copenhagen, drew the attention of the Royal Medical and Chirurgical Society (vol. liii) to this affection, which he was almost the first to recognise, but which has proved to be exceedingly common. It consists in the presence of masses of various shapes and sizes, growing most frequently from the posterior wall or from the roof of the pharynx, but sometimes also from the sides of that cavity, or even from the upper surface of the soft palate, but never from the back of the nasal septum. They are described as occurring in three forms, the *cristate*, the *cylindrical*, and the *flat*. They are sometimes soft and sometimes hard. They contain many vessels, especially veins; and they are otherwise made up of a scanty areolar network, having its meshes filled with lymph-cells. Their epithelium may either be ciliated or of pavement form, according to their exact seat. Their colour is generally the same as that of the more or less congested mucous membrane in their vicinity, or they may have a yellowish hue. In most cases the disease

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begins with hypertrophy of the third or pharyngeal tonsil. Pathologically it is allied to enlargement of the tonsils and lymph-glands in children, and to lymphadenoma.

It is often possible to tell by the way in which a person speaks, and even by the expression of his face, that he is affected with pharyngeal vegetations.

The peculiarity of the speech consists partly in an inability to utter the nasal sounds *m*, *n*, *ng*—so that instead of "common" the patient says "*cobbod*;" instead of "nose," "*doze*;" instead of "song," "*sogg*." This depends upon the occlusion of the posterior nares preventing the nasal cavity from acting as a resonator. Hence the explosive vocal sounds take the place of the continuous vocal or "nasal" sounds. The same effect is produced by closing the anterior nares, or by occlusion of the nares by the swelling of the mucous membrane produced by a cold in the head.

The peculiarity of the facial expression also depends upon the obstruction to the passage of air through the nostrils in breathing. This causes them to appear narrow and collapsed, and the nose itself looks thinned and flattened from side to side. It also compels the patient to keep the mouth more or less constantly open; and since the orbicularis oris no longer gives support to the other muscles of the face, the countenance acquires a vacant stupid aspect, often increased still further by an odd trick of twisting and pouting the lips.

Other symptoms are a feeling of fulness in the upper part of the fauces, as though there were a foreign body there, a secretion of thick greyish or greenish mucus which glides down the pharynx and compels the patient to be constantly clearing the throat, the presence of blood in the mouth, especially on first waking in the morning, and a more or less habitual headache. Children not only habitually breathe through the open mouth, but when asleep they snore, a peculiarity that should always lead to suspicion of the presence of these adenoid growths.

But what most commonly causes patients who have vegetations in the vault of the pharynx to seek medical advice is the impaired state of their hearing. Sometimes there is only occasional deafness with tinnitus, especially if they happen to take cold. This depends on occlusion of the Eustachian tubes by the adenoid growths.

In certain cases the aural symptoms are far more serious and lasting; there is chronic catarrh of the tympanic cavity, and the drum may even become perforated, giving exit to a purulent discharge. It is to be observed, too, that various affections of the fauces are apt to be associated with the presence of pharyngeal adenoid growths. The tonsils may be enlarged, there may be granular pharyngitis, the uvula and the soft palate may be thickened. There may also be catarrh of the anterior parts of the nasal cavities, though in the majority of cases the secretion of the Schneiderian membrane is rather deficient than excessive. Occasionally the lips are excoriated by the discharge, or there may be hæmorrhage or ozæna.

The easiest method of detecting with certainty the presence of vegetations in the vault of the pharynx is to explore the upper part of the pharynx with the forefinger. This is to be passed between the tongue and the roof of the mouth, and insinuated by the side of the uvula until it glides upwards behind the velum. It is then carried along the posterior

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edge of the septum of the nose, and turned in various directions until every part of the space has been thoroughly examined. If necessary a probe introduced through the nostril may be used to bring the individual vegetations in contact with the finger. The examination may cause some nausea, and may even be followed by pain in the back of the head; there is often a good deal of bleeding from the growths when they are touched. Rhinoscopy is seldom of much assistance in diagnosis. The vegetations can be most satisfactorily seen in cases of cleft palate, a deformity that appears to be not infrequently associated with their presence.

The systematic examination of children at schools in Denmark, England, and Holland has shown that from 1 to 5 per cent. of them are affected with pharyngeal adenoid growths. The disease is said to be more frequent when the climate is cool and damp. It is seen chiefly in persons under the age of twenty-five years, and it appears often to be congenital or to date from very early childhood. Out of 107 cases Mr Symonds noted that 90 were under fifteen years of age. It frequently affects several members of the same family, and is sometimes hereditary. Meyer thought that it is more common in boys than in girls, but in Mr Symonds' cases there were 65 female to 58 male patients.* It is sometimes a sequela of measles. According to Wiesener, of Bergen, it may lead to a scrofulous (*i.e.* tubercular) infiltration of the cervical glands.

When adult life is reached, adenoid vegetations in the vault of the pharynx appear commonly to shrink and to disappear spontaneously. Nevertheless it is important to remove them as soon as their presence is detected, especially on account of the damage which they do to the organs of hearing.

Sometimes cauterisation with solid nitrate of silver suffices to destroy them, but they generally have to be scraped off by suitable instruments, of which descriptions may be found in Meyer's several papers, and in the 'Transactions of the International Congress of 1881.' He employed a small oval ring, with a sharp though not cutting edge, mounted on a slender stem. This is passed backwards through the patient's nostril, and is guided to the bases of the vegetations by the operator's left forefinger introduced through the mouth. The operation causes little pain but profuse hæmorrhage, which is easily checked by the injection of cold water containing salt and carbolic acid. It is often followed by headache, and even by slight stupor for a few hours. It sometimes has to be repeated once or oftener, because all the vegetations are not completely got rid of on the first occasion. If any remains of them are left they are sure to grow again. Meyer therefore insists upon the importance of after-treatment, consisting in cauterisations with nitrate of silver and daily injections of a solution of bicarbonate of soda, or of chlorate of potass. So long as there is any soft tissue to be felt which bleeds when touched, these measures should not be discontinued.

Adenoid pharyngeal growths are frequently removed in our out-patient room, without danger and with excellent results. The instrument now most used is a forceps devised by Löwenberg, but some surgeons use the finger-nail by preference in ordinary cases. The effect of the complete removal of pharyngeal adenoid growths is quickly to restore the natural speech, to change in a surprising manner the expression of the patient's face, and in many cases to bring back the sense of hearing.

* See a lecture reported in the 'Guy's Hospital Gazette,' October 11th and 25th, 1890.

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DISEASES OF THE ŒSOPHAGUS.—This part of the alimentary canal is remarkably free from the slighter inflammatory affections which are so common both above and below it. Its thick layer of squamous epithelium seems to protect it from all but the most violent irritation; and it has neither the rich blood-supply, nor the active secreting functions, nor the abundant lymphatic tissue, which elsewhere in the alimentary tract become occasions of disease.

Chronic *inflammation* of the gullet with thickening of the mucous membrane is, however, seen either as the result of external pressure, *e. g.* from a thoracic tumour or an aneurysm, or in cases of habitual congestion from chronic valvular disease of the heart. In the former case the thickened and opaque mucous membrane is, in external appearance as in pathology, like the "corns" produced by friction on the pericardium, or the white patches of the tongue where it is touched by a tooth or other mechanical irritant. In the latter case extreme venous congestion may be discovered after death, with desquamation of the upper layers of epithelium, a condition comparable to the congestive catarrh of the stomach which is so important a result of disease of the heart.

Occasionally, when no source of pressure or irritation can be ascertained, the mucous membrane is found (at times over a considerable space) to be covered with minute papillæ, which may be large and circumscribed enough to deserve the name of a *papilloma.*

Functional stricture.—The above pathological conditions are without clinical significance: there are, on the other hand, functional affections of the gullet which are at present without an anatomical explanation. Of these the most important is what has been called "spasmodic stricture." The patient is usually young, most often a girl at the age between puberty and childbearing, when functional neuroses are most common. It also, however, occurs in male subjects, and one of the most marked and obstinate cases the writer has seen was in a boy of fourteen. In most cases the neurotic or "hysterical" character of the affection is sufficiently evident, and the easy passage of an œsophageal sound completes the evidence.

Regurgitation.—Another condition which is probably at first functional is rejection of food after it has passed the constrictors of the pharynx. This differs from the gastric regurgitation, or, as it may be termed, "rumination," which will be described under disorders of the stomach; for here the food never reaches the gastric cavity, but is detained in the gullet. Œsophageal regurgitation appears to begin rather as a bad habit than a disease; but whether or not there be any structural lesion as its original cause, there is frequently, or perhaps always in confirmed cases, a pouch formed in the lower part of the gullet, in which food collects before its regurgitation. *Dilatation of the œsophagus* is said to be more frequent in men than in women. It usually involves the whole thickness of the tube; but cases have been described in which the mucous coat alone has protruded between the muscular fibres, so as to form a hernial pouch. A classical case of this curious condition was published in the thirteenth volume of the 'Medico-Chirurgical Transactions' (1849) by Mr Worthington, of Lowestoft.

An œsophageal pouch is most frequently met with, not as a primary lesion, but as the result of a stricture immediately below it. It has

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occasionally proved to be the result of a mere narrowing of the gullet at its cardiac end. Such a case is described and figured by the author in the 'Guy's Hospital Reports' (third series, vol. xvii, p. 414), where at last a cancerous growth developed and ended in the patient's death at eighty-four, forty years after the appearance of dysphagia. In the same paper is figured a dilated œsophagus resulting from a simple, non-traumatic stricture of the cardia, which was described by Dr Wilks in the seventeenth volume of the 'Pathological Transactions' (p. 138). Regurgitation and dysphagia had existed all the life of the patient, a healthy farmer, who had once consulted Sir Astley Cooper for œsophageal stricture, and who died at seventy-four of acute pneumonia. Mr Durham has discovered two cases of dysphagia and simple stricture, recorded by Sir Everard Home ('Practical Observations on the Treatment of Stricture in the Urethra and in the Œsophagus,' 1821, vol. ii, p. 398). One of these showed, *post mortem*, a fold of mucous membrane, which narrowed the gullet just opposite the cricoid cartilage.

Dysphagia lusoria, as it has been called, demands a word of notice. The term was first applied by Dr Bayford, of Lewes, to a case (probably of spasmodic stricture of the gullet) in which the right subclavian artery arose from the third part of the aorta, and passed to its distribution between the œsophagus and trachea. The existence of this abnormality was probably a mere coincidence. The much more frequent irregular arrangement of the right subclavian arising from the third part of the arch, and passing behind the trachea and œsophagus, between the latter and the vertebræ, is found *post mortem*, in persons who have never experienced difficulty in swallowing.

Organic strictures of the œsophagus may be divided into the simple or non-malignant and the cancerous.

Simple stricture can in most cases be traced to a *traumatic* origin, most frequently to irritant poisons, such as the mineral acids. Constriction of the gullet by external cicatrices or pressure of diseased vertebræ, abscesses, aneurysms, cancerous mediastinal glands, or possibly a distended pericardium, will of course have the same results as true stricture or contraction of its walls, and will only be distinguishable during life by evidence of the presence of the external compressing cause.

Stricture is occasionally due to contraction of a *simple ulcer* of the œsophagus, which is, however, a very rare affection compared with the corresponding lesion in the stomach, or even the duodenum. There is no doubt that *syphilitic* ulcers, probably of a tertiary gummatous nature, may give rise to contraction of the œsophagus ('Guy's Hospital Reports,' vol. xvii, ser. 3, 1872, p. 413). Above a stricture there may frequently be seen hypertrophy, with or without dilatation of the muscular walls of the gullet.

Malignant stricture of the œsophagus is always primary, and is usually of the epithelial, keratoid, or flat-celled variety. Adenoma or glandular cancer has occasionally been observed, but true examples of the encephaloid or scirrhous variety are extremely rare.

The most frequent position is generally stated to be in the middle of the tube, opposite the bifurcation of the trachea. A less common seat is the upper portion, where cancer spreads so as to be described indifferently as pharyngeal or œsophageal. Lastly, a malignant stricture is occasionally met

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with at the entrance into the cardiac orifice of the stomach. It is here that its presence is most apt to be overlooked, the diseased portion being left in the diaphragm when the stomach and œsophagus have both been removed, as Virchow long ago observed. The foregoing statement is that of Wilks, Rindfleisch, and Klebs. Many text-books, however, follow Rokitansky's original assertion that the upper part is the most frequent seat and the lowest the rarest. Förster, Moxon, Payne, and Coats say that the commonest seat of cancer of the œsophagus is its lowest third; and this statement is confirmed by the careful analysis of Petri and Zenker. In 58 cases collected by these two writers 4 were in the upper, 14 in the middle, and 24 in the lower third. The remaining 16 cases spread over the middle and adjacent parts also. The latter group of cases is the disturbing element which has probably affected the classification of statistics. It still, however, remains true that the part which corresponds to the bifurcation of the trachea is a frequent seat of œsophageal cancer, and that cancer of the extreme cardiac end is rare. In 13 cases collected by Dr Moore, of St Bartholomew's Hospital, the lower third was affected in 7, the middle third in 5, and the upper in 1.

The malignant growth is sometimes a mere cartilaginous ring like an annular stricture of the colon, but more frequently it forms an ulcer which only partially encircles the tube, and infiltrates and spreads up and down for an inch, or even more. Hughes Bennett recorded a rare case of double cancerous stricture ('Princ. and Pract. of Medicine,' p. 453).

The growth of epithelioma of the gullet is slow, and it rarely affects more than the neighbouring lymph-glands of the mediastinum; but occasionally secondary nodules are found in the viscera,—least rarely, perhaps, in the liver or the lung. It may produce fatal hæmorrhage, or may perforate the œsophagus by sloughing, involve the vagus nerves, or open anteriorly into the trachea or lung.

Myomata, polypi, besides the warty growths above mentioned, and other non-malignant growths, have occasionally been observed in the œsophagus.

Malignant stricture is more common in men than in women, and is rare before middle life.

Its early *symptoms* are slight and its progress insidious. Difficulty in swallowing solid food is commonly the first complaint. Pain, though occasionally severe, is often long before it appears, and is sometimes almost absent. Gradually the patient finds it more and more difficult to swallow soft food, and at last even liquids, and increasing emaciation is the result. So latent may the symptoms be that the disease has sometimes been only discovered after death, though a tradition of a diagnosis once made by Sir Astley Cooper shows how the aspect and age of a person suffering from this disease may lead to its recognition by experienced observation, or by a shrewd guess.

In most cases, when a man of middle age, or older, comes complaining of inability to swallow food, he can indicate the exact spot where he feels it stop; and the negative result of examination of the other organs, with his progressive loss of flesh, makes the diagnosis easy. The way in which he eats, taking very small pieces of bread at a time, is very suggestive. Soft food, like bread and milk, can be eaten when ordinary diet is rejected, and it is often weeks or months later before difficulty is experienced with liquids. The regurgitation of a little blood with the food is very characteristic.

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The only possible diagnosis between simple and malignant stricture depends upon the age of the patient and the history of previous injury.

The aspect of a patient in the advanced stage of the disease is very characteristic—the extreme marasmus, without jaundice or cyanosis or dyspnœa, the look of starvation and of long-endured suffering from thirst, the hollow eyes, dilated pupils, sunken cheeks and temples, and, above all, the deeply concave abdomen, feeling empty when examined, and allowing the aorta, the vertebræ, and the kidneys to be plainly discerned by the fingers. The skin is dry and rough, the condition known as pityriasis tabescentium. The bowels are confined for days or weeks. The urine is scanty, high-coloured, and often offensive.

The cautious passage of a bougie is the only decisive proof of the nature of the disease, and also gives a criterion of its position and of the calibre of the tube. Hamburger's methods of diagnosis by auscultation deserve mention (Erlangen, 1871. See also Dr Allbutt's paper, 'Brit. Med. Journ.,' ii, 1875). Of less practical importance is the ingenious attempt to obtain a view of the gullet by Waldenburg; his instrument is figured in the 'Berlin. klin. Wochenschrift,' 1870, p. 580.

"*Tandem, post Tantali pœnas diu toleratas, lente marasmo contabescunt,*" as Boerhaave remarked. Death, however, often occurs from intercurrent pneumonia or pleurisy. In one case under the writer's care the primary stricture was latent, and the first symptoms were pain, and afterwards paraplegia, caused by secondary cancer of the vertebræ.

The *duration* of these cases is very variable; it is to be counted by months, and some patients have lived on for two years, or perhaps for longer. The most rapid cases may prove fatal by perforation within a few weeks of the first appearance of dysphagia.

The *treatment* of stricture of the œsophagus is purely mechanical. When free from ulceration the stricture will usually be benefited by the frequent passage of a bougie; in fact, by the same method of dilatation as is adopted for stricture of the urethra. In cases of simple traumatic constriction this is sometimes an effectual cure; but even in cancerous strictures, so long as there is no ulceration, the occasional passage of an olive-shaped bougie frequently affords great relief. The utmost care and gentleness is essential, or fatal perforation may ensue. As Mr Bryant well puts it, when a patient complains of difficulty in passing food onwards down the gullet after the act of swallowing, and of its return into the mouth, the practitioner should first think of thoracic aneurysm, then of cancer, and then of some other kind of ulceration. The dread of such a catastrophe as perforating an aneurysmal sac, or thrusting a bougie into the pleural cavity, will, however, be the best safeguard against its occurrence. No force which an intelligent hand could use will perforate an intact mucous membrane. When perforation, or even ulceration, has already taken place, no one would willingly risk the passage of a tube. But when there is no evidence of ulceration the practice is defensible and beneficial.

The *sonde œsophagienne à demeure* is an instrument which is passed through a stricture, and then left in it as a catheter is left in a stricture of the urethra. It was advocated by Mons. Krishaber at the International Medical Congress of 1881 ('Transactions,' vol. ii, p. 392). He advises its passage through one of the nostrils rather than the mouth, and in proof of the tolerance of the instrument relates four cases in which the tube remained continuously

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in situ for 46, 126, 167, and 305 days respectively. Feeding, of course, takes place entirely through the hollow œsophageal sound. A similar plan has been carried out by Mr Symonds at Guy's Hospital with good success.

When obstruction has become complete, life may be preserved for a time by nutrient enemata. For this purpose small quantities of peptonised food without salt or alcohol are best employed; and in many cases raw eggs, beef-tea, and pancreatised milk are well retained and absorbed. But often the rectum rejects the nutriment; after a time this result is almost sure to occur, and even in the most favourable cases the patient is insufficiently nourished. In acute cases of disease, or of injury or operation about the mouth and throat, or even while a gastric ulcer is given time and rest to heal, the treatment by rectal alimentation is most valuable. But when, as in stricture of the œsophagus, the disease is progressive, it is far better, so soon as occlusion occurs, and no liquids even can reach the stomach, for the operation of opening the stomach to be faced before the patient's strength and endurance have been exhausted.

The operation of gastrotomy, or *gastrostomy*,* was first performed by Sédillot in 1849, and was introduced into England by Mr Cooper Forster. It has since been amply justified by the long periods of life and comfort which it has afforded to patients who would otherwise have died by one of the most painful deaths—that from thirst. In a case brought before the International Congress in 1881 ('Trans.,' p. 456) the patient survived a year and a half; and in a second case (for malignant stricture), by the same surgeon, life was prolonged for 128 days. For similar cases see a paper by Dr Gross, jun., in the 'American Journal of Med. Sc.' for 1884.

The safety of the operation has been increased by the plan introduced by Mr Howse, of first cutting down to the stomach and fastening it by sutures to the abdominal walls, and a day or two afterwards, when adhesions have formed, opening it and introducing the cannula.

* Gastro-stomie (*i. e.* making a mouth in the stomach) was the French surgeon's original term. Mr Bryant follows Dr Pooley, of New York, in defining "gastrotomy" as opening the stomach for removal of a foreign body.

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DISEASES OF THE STOMACH

"Qui Stomachum Regem totius corporis esse
Contendunt niti verâ ratione videntur;
Hujus enim validus firmat tenor omnia membra,
Et contra ejusdem firmantur cuncta vigore."

QUINTUS SERENUS, from '*Ventriculi Querelæ et Opprobria*' *of Bernhard Swalve*, 1664.

ACUTE GASTRITIS—*Acute catarrhal gastritis—its symptoms, causes, histology, and treatment—Acute paralytic distension—Acute suppurative gastritis.*

CHRONIC DYSPEPSIA—*Atonic dyspepsia and chronic catarrhal gastritis—Symptoms—Anatomy—Causes—Diagnosis and prognosis—Treatment—clinical varieties—drugs and diet.*

Gastralgia—Anorexia—Eructation and Pyrosis—Vomiting, bulimia, pica—Hiccough—Flatulence—Hæmatemesis.

GASTRIC ULCER—*Anatomy—Pathology—Ætiology—Symptoms and diagnosis—Duodenal ulcer—Latency—Event and duration—Treatment.*

CANCER OF THE STOMACH—*Carcinoma of the pylorus and of the body of the stomach—Sarcoma—Colloid cancer—Symptoms—the tumour—consequent dilatation—Gastro-colic fistula—Duration—Diagnosis—Treatment.*

Fibroid Induration of the stomach—Gastric concretions.

IN health, as everyone knows (or has known) by experience, the process of digestion is unattended with any kind of sensation; we ought not to be conscious that we have stomachs. But often digestion is accompanied with unpleasant feelings, varying in degree from a slight sense of weight or discomfort up to agonising pain. These may be symptoms of severe disease of the stomach, or of some slight organic change, or of a mere functional derangement. Many of the less serious disorders of the stomach are commonly grouped together as dyspepsia, or indigestion.

1. ACUTE GASTRITIS.—It is not uncommon in persons who were before perfectly well, for the stomach to resent some particular meal, on account either of its quality or its quantity. Such cases are very common; the following may be taken as examples:—A schoolboy hastily swallows a large quantity of grapes, skins and all; in a few hours he feels ill, and presently rejects the contents of his stomach; next morning he is well. A man eats much more than he requires; he goes to bed with an uneasy feeling at the epigastrium, and in the night vomits in surprising quantity the food that he had swallowed, almost unchanged, but dry, without the liquid with which he had washed it down; his discomfort is at an end, and he falls asleep. A little bile constantly appears in the vomit after the stomach has emptied itself of its accumulated contents; and formerly this fact would have been regarded as a sufficient ground for calling the complaint a "bilious" attack. But we now know that, in consequence of the antiperi-

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staltic movements induced in the duodenum, bile enters the stomach whenever there is much vomiting, and is rejected in its turn.

To attacks like these the name of acute indigestion is applicable. But it is usual to describe under that name a somewhat different class of cases, which are of longer duration; and since these are believed to depend upon a catarrhal inflammation of the gastric mucous membrane, the terms "acute indigestion" and "acute catarrh of the stomach" are used as almost synonymous.

Symptoms.—In addition to a sense of weight and oppression at the epigastrium, which is common to all forms of dyspepsia, there is local pain; usually a dull aching, but sometimes of a burning, stabbing, or griping character. One patient complains of soreness at the left of the sternum and about the left scapula; another feels more pain in the back than in the abdomen. In some cases the epigastrium is tender, and it often feels tense and full. The patient is thirsty and eager for cold and effervescing drinks; but he has generally no appetite, and often feels a loathing for all kinds of food. The breath is offensive, the bowels confined, and the tongue foul and coated with thick yellowish fur. Nausea and retching are generally present, or patients may complain of the sour gases which come up by eructation. Partially digested food, with a very acid taste, is vomited; or a quantity of whitish mucus. Aching in the back and limbs, malaise, and depression of spirits are more or less marked; and in some cases there may be slight fever, but the skin is generally moist. In one case the attack began with shivering, and an herpetic eruption has been seen to break out on the lips and chin.

An attack of this kind may last from a day or two to a week, perhaps longer; and if injudiciously treated, it may pass into chronic gastritis.

Various *causes* of acute gastric catarrh are mentioned by writers. The cases most readily accounted for are those in which it follows some indiscretion in diet. In one patient a severe and protracted attack came on the day after he had dined on venison and champagne. Decomposing meat or vegetables, and shell-fish under certain conditions, are especially apt to give rise to acute catarrh of the stomach. The same effect is produced by the inhalation of arsenical dust or vapour given off from green wall-papers. The late Dr Wilson Fox mentions the case of a healthy child who, after sleeping in a room lined with such a paper, was seized with severe vomiting, and brought up blood. The direct ingestion of irritants like arsenic and antimony, causes violent inflammation of the stomach, but the effect is not only local. In infants improper food frequently causes acute gastric attacks, commonly, but not always, complicated with diarrhœa. In these cases prostration is a marked feature, and death sometimes results.

Exposure to changes of temperature is said to be another cause. Dr Fox quotes from Guipon the case of a workman, who on several occasions was seized with vomiting and pain in the stomach after being exposed to the heat of a furnace. He thinks that attacks of this kind are apt to occur in changeable weather, as in the spring and later autumn, when cold and high winds prevail, as well as during the severe heats of summer and early autumn.

Epidemic influences are also supposed to act. Dr Fox cites Chomel as having observed that gastritis was frequent when cholera was raging, and had himself noticed the same thing in 1866.

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Acute catarrh of the stomach is said to be a frequent complication of scarlatina, erysipelas, measles, diphtheria, smallpox, puerperal and other fevers. No doubt these diseases are often ushered in by repeated vomiting; but this only lasts till the characteristic rash appears.

Since acute gastric catarrh is very seldom fatal except in infants, one might expect that its *morbid anatomy* would be unknown. But, half a century ago, the remarkable case of Alexis St Martin occurred. This young Canadian had a fistulous opening into his stomach, as the result of a terrible gunshot wound, so that part of the mucous membrane was permanently exposed to view. The case was carefully investigated by Dr Beaumont, of Pittsburg, in Pennsylvania, and he relates that deep red pimples sometimes appeared, which afterwards became filled with purulent matter; and at other times irregular circumscribed red patches, small aphthous crusts, and abrasions of the lining membrane, leaving the papillæ bare. These diseased appearances, when considerable, were attended with dryness of the mouth, furring of the tongue, thirst, and acceleration of the pulse, and the secretion of gastric juice was suspended, so that food remained undigested for twenty-four or forty-eight hours or more, although liquids were absorbed as soon as they were swallowed. Mucus was also poured out by the surface of the stomach, and slight hæmorrhages sometimes occurred.

It is true that symptoms were by no means constantly present when the mucous membrane was inflamed; but the case is of value as showing that the stomach is susceptible of morbid changes which, if they could generally be seen, would arrest attention. For, in the deadhouse, such changes can seldom be observed satisfactorily. After death the lining membrane of the stomach is often acted upon by the gastric juice, so that it becomes softened and pulpy, or may be entirely dissolved over a more or less extensive area, and all the coats of the organ may in this way be perforated, so as to leave a large ragged aperture. As might be expected, this is generally at the back of the stomach, on which the contents rest while the body lies with its face upwards.

Moreover, in the stomach, as in all other parts, congestion is very apt to subside after death, so that it can no longer be detected. We have, however, seen that in chronic disease of the heart the gastric mucous membrane is found intensely reddened, ecchymosed, and lined with tenacious mucus (pp. 60 and 78); and great reddening is also seen in the bodies of those accustomed to drink spirits to excess. It is probable that the changes which Dr Beaumont described would no longer be visible at an autopsy.

Certain other changes, however, are described as characteristic of acute gastric catarrh—a milky opacity of the mucous membrane, which is soft, thick, and lacerable. Under the microscope, the secreting cells and the nuclei are swollen, irregularly distending the tubules; they are filled with granular matter, and in severe cases they often break down into a granular *débris*. Dr Cayley and Dr Fenwick have found in fluids from the stomach tube-casts comparable with those which are well known to occur in the urine in Bright's disease. A drawing of these is to be seen in the 47th volume of the 'Med.-Chir. Transactions.' Infiltration of leucocytes between the tubules has been also observed, and proves the existence of inflammation.

In addition to these changes, Dr Fox describes an increase in the size of the solitary lymph-follicles of the stomach, which appear as small white

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specks, scattered over its surface, and which sometimes ulcerate, forming little cup-shaped depressions. The interstitial tissue of the mucosa also becomes infiltrated with leucocytes.

It is important to note that the cases in which these appearances have been found have not been simply cases of gastric catarrh, for these do not terminate fatally. The observations in question were made in the bodies of those who had died of scarlatina, diphtheria, pneumonia, or some other fever. They undoubtedly prove that these diseases are attended with morbid changes in the stomach, but not, it would seem, that they are to be regarded as so many causes of acute gastric catarrh, in the clinical sense of that term. For, although in the instances in which the morbid changes were found after death, the patient may have been more or less sick and have had a furred tongue, yet it is probable that the catarrh of the stomach was only secondary and did not in any way modify the course of the disease.

Clinical varieties.—We may clinically recognise acute catarrhal gastritis under the following conditions. First the cases due to irritant *poisoning*, as by arsenic. The possibility of this as a cause must never be forgotten; it may be accidental, suicidal, or homicidal in origin, and it may be the result of a single dose or of gradual absorption suddenly producing its accumulated effect. Irritability of the eyes or actual ophthalmia, diarrhœa, and severe pain in addition to the vomiting are the most important symptoms. If no arsenic can be discovered in the vomited matters or digesta, Marsh's or Reinsch's process of identification will probably have already pointed to antimony, should that metal (as in the well-known Balham case) have been the cause of gastritis. Mercury would be almost certain to produce salivation at the same time. Of vegetable irritants poisonous fungi are the most frequent causes of acute gastritis in adults, various berries in children. The poisonous symptoms from eating high game, "tinned lobster," or mussels merge into those of ordinary acute dyspepsia.

Secondly, *gastritis ab ingestis*—poison, overeating, and drink—particularly that of infants fed on milk which does not agree with them, is the above mentioned gastric catarrh which accompanies all *febrile* states, but is strictly symptomatic and secondary. Variola is perhaps, of all fevers, that in which the gastric symptoms are most constant and severe, and next to it come scarlatina, pneumonia, and acute gout.

Thirdly come the cases of acute and subacute gastritis which constitute so marked a feature of the morbid anatomy of *cardiac* disease.

Lastly, there is a clinical group of cases in which somewhat severe epigastric pain is accompanied by vomiting, first of the contents of the stomach, then of a quantity of mucus, and lastly, of bile. There is a thickly coated tongue, complete anorexia, considerable thirst, and, as a rule, constipation. These symptoms occur in elderly rather than in young patients, and may come in the course of chronic Bright's disease (quite apart from uræmic vomiting) or other lingering malady.

But they are most often seen in combination with bronchitis and myalgia. In such cases we find the heart and the kidneys unaffected and no evidence of more than moderate catarrh of the large bronchial tubes. The temperature is not raised, but the pulse is quick and irritable, and the "muscular rheumatism" severe, particularly in one or other shoulder and in the back and loins; or there is decided pleurodynia but no pleuritic rub or effusion. The patients appear to be more ill than any physical signs discovered account

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for; and one is anxious lest some primary lesion has been overlooked. In most cases, however, with careful nursing, warmth, strict diet, and well-diluted brandy if the strength begins to fail, these cases do well.

The *diagnosis* of acute gastritis is generally easy. Both Bamberger and Dr Wilson Fox observe that enteric fever in its early stage is the disease most likely to be confounded with it. What is most important is to remember the possibility of poison, and particularly in children, of the advent of scarlatina, pneumonia, or some other acute disease, of which the gastric disturbance is only a symptom.

In the *treatment* of an attack, the first thing to be attended to is the diet. In mild cases, complete abstinence from everything but cold water for twenty-four hours often effects a cure. In severe and protracted cases, nutrient enemata may be used with advantage. If any nourishment is given by the mouth it should be milk in small quantities, diluted with soda-water or lime-water. Sometimes this is less grateful than warm milk and water with a little cinnamon, or barley-water, or rice-water. Persons who cannot take milk may have veal or chicken broth. As the symptoms subside, light farinaceous puddings may be allowed. Unless there be great prostration the patient should abstain altogether from alcohol; champagne is not so suitable in this as in other forms of irritability of the stomach. If a stimulant appears necessary, brandy well diluted is the best.

In sucking infants, the quality of the milk must be carefully inquired into. Often they are given more than they can digest, and pain and vomiting ensue. Sometimes it is advisable to keep the child from the breast for a few hours, giving it only a little rice-water or very diluted milk at intervals. In infants brought up by hand, the substitution of asses' milk for cows' milk is sometimes effectual; but too often one can save the child only by engaging a wet-nurse at once.

Hot fomentations or linseed poultices may be applied with advantage; or a hot-water bottle. Or spongiopiline may be used, soaked in hot water, squeezed out, and sprinkled with laudanum.

The administration of emetics and purgatives is an important question. Two cases of Sir Thomas Watson's may here be quoted. One was that of a person who had been taking large quantities of cream with his tea and coffee. After suffering for several days with severe gastric pain and disorder, he threw up a mass of hard curd like a small cream cheese, and he was at once completely relieved. In the other case a similar fit of indigestion terminated in the ejection of a mass of snuff. It is certain, therefore, that irritating matters may remain for a considerable time in the stomach. But, on the other hand, as Bamberger remarks, one must not trust too much to the circumstances that the patient continues to experience uneasy sensations, for these may continue long after their cause is removed, just as one goes on imagining that there is a foreign body in the eye long after it has been got rid of. Bamberger, indeed, gives a caution against the use of emetics or purgatives, which, he says, have caused catarrh of the stomach much more often than they have cured it. Dr Fox says that an emetic may be administered "when the presence of undigested food is indicated by cramp-like pain, nausea, ineffectual attempts to vomit, and faintness;" but adds that antimony and even mustard are to be avoided, and that ipecacuanha with large draughts of lukewarm water or of infusion of chamomile is the best emetic in these cases. He recommends, however, rather active purgatives; from three to eight grains of calomel followed by a black draught,

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or by castor oil; or a dose of blue pill and compound colocynth pill, with a seidlitz powder afterwards. Dr Beaumont certainly found in the case of Alexis St Martin, already referred to, that the administration of calomel was followed by subsidence of the morbid appearances in the gastric mucous membrane.*

But whatever may be said as to the propriety of beginning the treatment of acute gastritis with an emetic or with a purge, all writers are agreed that when once given it should not be repeated. Sedatives and antacids are then the remedies, and sucking small pieces of ice often gives much relief. For severe vomiting, dilute hydrocyanic acid is the best drug. In ordinary cases bismuth is particularly useful; ten grains of the subnitrate with as much carbonate of soda and a little morphia will often give immediate relief. Seltzer or Vichy water may be prescribed with much advantage, a pint or a pint and a half being given daily. Effervescing medicines are also useful. But the patient should not be allowed to take liquids in such quantities as to distend the stomach. During convalescence the remedies that will hereafter be recommended for chronic dyspepsia become applicable. But the use of the bitter tonics in protracted cases of gastric catarrh of the stomach is probably harmful.

Acute gastric distension.—Although in adults acute catarrh of the stomach is unattended with danger, it may be a question whether certain very rare cases, in which rapidly fatal collapse occurs after moderate gastric symptoms have lasted a few days or a week or two ought not to be regarded as due to the supervention of a further morbid state upon one of catarrhal inflammation. The author described two instances of the kind in the 'Guy's Hospital Reports' for 1872–3, under the name of "acute dilatation of the stomach;" but a better title would perhaps be "acute paralytic distension." One case occurred in a man, aged thirty, who had for some time been in the Hospital under Dr Owen Rees, and was supposed to have incipient phthisis. He was seized with persistent vomiting; he passed no urine, and he gradually became collapsed. On examining him on the third day I found that the abdomen was retracted, and that its walls were rigid. There was dulness above the pubes and half way up to the umbilicus. This might have been attributed to distension of the bladder, but a catheter had been passed, and no urine could be obtained. Moreover, a splashing sound was obtained by manipulation of this region or of the iliac fossæ. He died the same afternoon. At the autopsy the stomach was found to fill the whole abdomen, and to contain a large quantity of fluid. But when removed from the body, it shrank back to about its natural size, showing only a number

* We ought to be very careful in prescribing purgatives in cases of supposed acute gastric catarrh. I can never forget a case which I diagnosed as of this nature, and which proved to be one of acute suppurative peritonitis. A bank clerk felt poorly one day after having eaten some pears in the afternoon. In the night he woke up with epigastric pain and vomiting. A medical man was not sent for for two days, and when he came he gave a mild aperient. This operated, and a day or two later the sickness subsided. There was a little delirium about the third day. The pulse was at no period of the case over 100; the temperature ranged from 100° to 101°. I was asked to see him on the sixth day. He then appeared to be better; the sickness and pain had ceased, he had begun to take food again. The pulse was about 90, of fair volume; the temperature exactly 100°. Except that the countenance was sunken, and that the eyes were surrounded by deep brown rings, there appeared no reason for alarm, and I concluded that the attack had been one of acute indigestion, and that the patient was in a fair way to recover. Within twenty-four hours, however, he died, and it turned out that there was diffused peritonitis, set up by ulceration of the vermiform appendix.—C. H. F.

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of white striæ on its serous surface, apparently analogous to "lineæ gravidarum." The other case was a man aged twenty, who had for a fortnight been suffering from abdominal pains and repeated vomiting. For two days the sickness had ceased, but he was worse in all other respects. His countenance was sunken; his eyes glassy and surrounded by deep rings of pigment; his breath nauseously sweet. His abdomen was generally distended, but the right hypochondrium was flat, and passing downwards and to the right above the navel a line could be seen, which indicated the upper border of the stomach. On manipulation of the lower part of the abdomen there was fluctuation and a splashing sound. Dilatation of the stomach was evident, and the long tube of a stomach-pump was passed down the œsophagus. A greenish fluid was ejected through it and by its side with considerable force. The pump was then connected with it, and no less than *seven pints* of the same fluid were removed by its means. The abdomen became deeply hollow while this was being done. The patient said that he felt much relieved, but he died four hours afterwards. The autopsy showed, as we expected, that the stomach had returned to its natural size and form; but there was a sloughing abscess behind the duodenum, communicating with the bowel. This must doubtless have caused the patient's death under any circumstances. But it may reasonably be hoped that in an uncomplicated case like the former one the prompt use of the stomach-pump would afford a chance of saving the patient's life if an early diagnosis could be made. It seems clear that the cessation of vomiting is due to a paralytic state of the gastric muscular coat, comparable with that which occurs in the bladder in cases of retention of urine. It is not clear what is the origin of the large quantity of fluid which the stomach contains in these cases. Unless it is merely what has been swallowed it must indicate an irritated or inflamed condition of the lining of the organ. Unfortunately the mucous membrane was not examined microscopically in either case.

The physical characters which indicate acute paralytic distension of the stomach during life are—(1) A rapidly increasing distension of the abdomen, which is unsymmetrical, the left hypochondrium being full, while the right is comparatively flattened. (2) The presence of a surface-marking which descends obliquely from the left hypochondrium towards the umbilicus, and which corresponds with the lesser curvature of the stomach. This seems to move up and down each time the patient breathes. (3) Dulness and fluctuation in the pubic region with resonance over the front of the abdomen. (4) The production of a splashing sound on manipulation. It is to be observed, however, that in one of my cases the first two of these signs were absent.

Phlegmonous and other rare forms of gastritis.—Another rapidly fatal disease of the stomach is acute diffused suppurative inflammation: a still more rare affection than paralytic distension. Bamberger mentioned it in Virchow's series of handbooks (1855), but Rokitansky had previously described it as suppurative inflammation of the submucous connective tissue, and refers to older cases recorded by Monro, Lieutaud, and others, which were collected by Albers. Sometimes the process, instead of being diffused, is confined to one or two spots, and may then be defined as submucous abscess of the stomach; such abscesses, or "gastric carbuncles," as Virchow calls them, may burst into the cavity of the viscus.

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Ackermann collected thirty cases, mostly puerperal. It has been compared to phlegmonous erysipelas or "pseudo-erysipelas" of German pathologists. Wilks and Moxon mention hepatic abscess as a result. A typical case of this rare disease was recorded by the author in the 'Path. Trans.,' vol. xxvi, p. 81.

The rarest form of all the anatomical forms of gastritis is *membranous* or "*croupous.*" It is sometimes the result of extension of diphtheria down the œsophagus, as observed by Jenner, but has also been seen in Bright's disease by Wilks, in phthisis by Fox, and apparently as an idiopathic affection by Niemeyer and Delafield. It is sometimes found associated with a similar membranous inflammation of the colon.

Sloughing, or, as German writers call it, "diphtheritic" gastritis, is described by Billard and Bednar as occasionally seen in new-born children.

CHRONIC DYSPEPSIA.—Writers on gastric affections describe two forms of chronic dyspepsia; one they call "atonic," the other "chronic inflammatory dyspepsia," or "chronic gastric catarrh." It is admitted, however, that there is great difficulty in distinguishing between them.

To begin with the former,—a person who has *atonic dyspepsia* complains of a sense of weight and uneasiness after food, which may last for some hours, or up to the next meal. The seat of these unpleasant sensations is usually the upper part of the abdomen; but sometimes, particularly by ladies, they are referred to the chest behind the sternum, so that a feeling of dyspnœa is experienced, or to the back between the shoulders, so that an impression is produced that food sticks in the gullet. There is rarely actual pain, unless it be as the result of flatulence. There is no tenderness of the abdomen, and pressure rather gives relief. Eructations are not uncommon, and often cause an offensive or rancid taste, perhaps due to the presence of butyric acid. The appetite is generally deficient; there may be a distaste for food of all kinds, even though the want of it gives rise to a sense of exhaustion. Thirst is generally absent; indeed, the ingestion of fluids often seems to aggravate the symptoms. The tongue is broad, pale, and flabby; it is marked at its edges by the teeth, but is not thickly furred. The bowels are always constipated.

Beside these symptoms, referable directly to the digestive organs, the patient complains of a sense of languor and weariness of the limbs, especially after his meals. His spirits are depressed, morose, or irascible. His pulse is soft and compressible, slow when he is at rest, but quickened by any exertion. Palpitation is commonly complained of, and occasionally the heart's action becomes intermittent. The skin is moist and there is no fever. The complexion is often pallid, sallow, and muddy, but there is seldom marked anæmia or loss of flesh, except in very chronic cases, and even then it is remarkable how well nourished the dyspeptic patient may be. The urine is copious and clear.

The symptoms of *chronic gastric catarrh* are in most respects very similar. The patient complains after his meals of weight and discomfort rather than pain; and tenderness of the abdomen, if present, is but slight. Thirst is a prominent and distinctive symptom, especially in the intervals between meals; and the patient often experiences a sense of exhaustion or of internal heat, which is relieved by drinking. The appetite is capricious, the breath is often offensive, and a nasty taste in the mouth is often complained of, especially on first rising in the morning. The gums

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are spongy, red, and inclined to bleed. An extensive secretion of saliva is not uncommon, so that at night it may escape from the mouth and wet the pillow, particularly if the dyspepsia is complicated by excess in smoking. The tongue is often of a bright red colour, and raw-looking, the papillæ standing out as bright red points. Or this condition may exist only at its sides and tip, the rest of its surface being coated with a white or brownish fur of greater or less thickness. The mucous membrane of the pharynx may be granular and inflamed, and often secretes a tenacious mucus, which is a source of great annoyance to the patient.

Some loss of flesh is common from chronic catarrh of the stomach, but emaciation or decided anæmia should make one search carefully for evidence of phthisis, gastric ulcer, or some other organic disease. Slight febrile disturbance is not uncommon; sometimes it occurs at night, and is followed by perspiration. The urine is generally scanty and deposits lithates, sometimes oxalates; while sometimes it is alkaline and throws down phosphates. The expression is anxious and careworn.

Vomiting does not always occur in chronic catarrh of the stomach. In the dyspepsia of habitual drunkards, however, vomiting of mucus, especially in the morning, is one of the principal symptoms, and it is probable that this kind of vomiting is always an evidence of a catarrhal state of the gastric mucous membrane. Some caution appears to be required in concluding that vomited matters contain mucus from their naked-eye appearance only; Frerichs showed that starchy substances are sometimes converted in the stomach into a tenacious glutinous material which may closely resemble mucus.

In some dyspeptic patients in whom vomiting occurs, the ejecta are intensely sour. This is generally due, not to an over-secretion of acid by the stomach, but to the formation of lactic, butyric, and acetic acids by fermentation from the starchy and saccharine elements of the food. These acids are often developed with great rapidity, and in such large quantity, that when the patient vomits the throat burns, the teeth are set on edge, and the eyes smart, just as though strong acetic acid had been taken into the mouth. At the same time the sour smell of acids, volatile at a low temperature, is diffused through the air. A further evidence that fermentation is the cause of the formation of acid in such cases is the fact that gas is evolved, which has been found to consist of a mixture of carbonic dioxide and a volatile hydrocarbon. Sulphuretted hydrogen is also found when eggs have been eaten, or other articles of diet containing much sulphur.

The morbid appearances presented by the mucous membrane are described as distinguishing atonic dyspepsia from chronic gastric catarrh. In the former the lining of the stomach is thin and transparent. In the latter it is almost always thickened and indurated. It may be so tough that it can be stripped off the subjacent tissue in large pieces, or the submucous tissue may at the same time be white and fibrous, in which case there is increased rather than diminished difficulty in separating the coats. The mucous membrane near the pylorus is often mammillated in cases of chronic gastric catarrh; but it is important to note that this condition is not always the result of inflammation, for it may be found in a healthy stomach, owing to contraction of the muscular layer which exists round the bases of the secreting glands. The most characteristic change in the appearance of the stomach in cases of chronic catarrh is its ash-grey pigmentation. This, when closely examined, is seen to depend upon the

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presence of numerous minute specks scattered thickly over it. Under the microscope they are seen to consist of granules of pigment (doubtless originally derived from hæmatin), which are deposited in the connective tissue between the tubes, or in the epithelial cells.

The gastric glands appear to present morbid changes in both forms of chronic indigestion. At least it seems to be certain that such changes are often observed in cases which in all other respects would come under the head of atonic dyspepsia. The secreting tubes are then found to be shrunken and wasted, and to have undergone fatty degeneration. They are often irregular in form and calibre. Their epithelium may have almost entirely disappeared, being represented only by granules and fat globules. In chronic gastric catarrh the changes in the epithelium appear to be the same. Cysts are not uncommonly found, which are probably the result of distension of parts of the tubes that were constricted off from the rest. Dr Wilson Fox observes that fatty degeneration is especially apt to affect groups of the glands one or two lines in diameter, which are then visible as small dead-white spots in the mucous membrane. Dr Habershon, Dr Handfield Jones, and Dr Fenwick are the other observers who have worked most at this branch of morbid histology. Several of Dr Jones's cases were in persons advanced in life, so that the fatty changes in the tubes might be attributed to a process of senile degeneration. Dr Fenwick has demonstrated the fact that when the secreting tubes are atrophied the digestive power of the dead mucous membrane is much less than under normal conditions. He found changes in the glands especially frequent in those who died of cancer of the breast, and he is disposed to regard this as one cause of the rapidly increasing anæmia which occurs in such cases.*

Redness of the mucous membrane has often been mentioned as one of the appearances characteristic of catarrhal gastritis; but in many cases of this kind there is no redness, at least in the dead body. There are cases in which the most intense injection of the gastric mucous membrane is an almost constant appearance; but these are cases of chronic or subacute catarrh from *disease of the heart.* The stomach is then found lined with a thick layer of mucus; and after this is washed away, the surface is seen to be of the most vivid crimson colour, which may either affect a large part of its surface uniformly or occur in spots. Ecchymoses are often present at the same time, and still more frequently they are simulated by small patches, due to arborescent injection of the branches of some minute vessel. When effusion of blood occurs into the submucous tissue it would seem that the gastric juice sometimes dissolves off the corresponding part of the mucous membrane. A little ulcer is the consequence, the floor of which is occupied by a layer of black coagulum. This process is known by the name of *hæmorrhagic erosion.* The greater part of the redness and congestion in these cases is due not to inflammatory but to passive congestion.

Another form of gastric catarrh in which the stomach is often found intensely reddened is that which results from alcoholic *intemperance.* An unskilled pathologist may easily be led to suspect the presence of an irritant poison in cases of this kind. Some time ago the author made an autopsy

* It must be said that the fatty or granular degeneration of secreting epithelium is a condition which has often been supposed to exist from want of knowledge of the normal or the *post-mortem* changes of these delicate and changeable structures. All observations made with low powers on the tissues, not perfectly fresh and without the aid of osmic acid and the other modern histological appliances, need confirmation.

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in a case of a young man who had suddenly died in a railway train early in the morning. There was no cause for his death discovered, but the stomach was most intensely reddened and ecchymosed. It was clearly ascertained that there had been no foul play, and there appeared to be little doubt that the abuse of stimulants had been the cause of the gastric irritation. As Wilks and Moxon remark, no mere redness and injection of the gastric mucous membrane is enough to prove the presence of an irritant poison. There must either be chemical evidence of its presence or actual ulceration.

Ætiology.—Theoretically, the following are possible or probable causes of indigestion of wholesome food: want of secretion of hydrochloric acid or of pepsin, or over acidity of the gastric juice, or over secretion of mucus; want of active movements of the stomach-walls; fermentation of food, leading to development of carbonic dioxide, of acetic or butyric acids; irritability of the gastric mucous membrane from inflammation or from neurotic hyperæsthesia, leading to pain or to vomiting, or to inhibition of secretion or movement, by reflex influence.

Again, the digestive apparatus being healthy, indigestion may arise from the food put into the stomach being too bulky; or not cooked enough if starchy, or over-hardened by cooking; or imperfectly comminuted by mastication; or imperfectly insalivated.

Practically, faults in diet may be regarded as the most frequent causes of chronic dyspepsia by producing chronic catarrh of the stomach: want of secretion and want of movements are probably important causes of atonic dyspepsia; and direct inhibition of cerebral origin is also without doubt frequently operative. It is possible that a disposition to dyspepsia is sometimes inherited.

Hot seasons, relaxing climates, exhausting discharges, sedentary occupations, venereal excesses, prolonged anxiety of mind, long-continued depressing emotions, have been counted among causes of chronic dyspepsia. They probably act, if they act at all, by interfering with proper meals.

Undoubted causes of indigestion are: over-eating, the habit of waiting too long between the meals, *imperfect mastication* of the food (the state of the teeth should always be looked after), taking too much fluid (especially cold water) with the meals, the *abuse of stimulants*, of condiments or of tea, excessive smoking, and taking bodily exertion or making mental efforts while digestion is going on.

The quality of the food may cause dyspepsia. Dr Chambers cites the case of a poor needlewoman who had subsisted for a year on bread, potatoes, and tea, getting sometimes a little bacon but hardly ever other kinds of meat, and who suffered so much from dyspepsia that she dreaded to eat. For such a patient and for many hypochondriacs variety and attractiveness of food are essential. In other cases indigestion always follows some particular article of diet, such as fatty matters or soups. Dr Chambers attempted to describe "indigestion of vegetable food," "of albuminoid food," "of fatty food," and "of watery food" separately, but with no great success. He gives an instance of a lady who from childhood had never been able to take roast beef without afterwards having heartburn. This he attributes to the fat which lies between the muscular bundles in stall-fed bullock's flesh. With regard to digestion, however, many idiosyncrasies are met with, of which it is not possible to give explanations, but which the physician must not overlook. Strawberries disagree with some persons, eggs with others, melted butter with many. Twice-cooked meats, potatoes,

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new bread, cauliflower, and jams or candied fruits are some of the most frequent sources of indigestion.

Although atonic dyspepsia sometimes attends senile decay, the ordinary flatulent dyspepsia with constipation which comes on after a meal is by far most frequent in young adults. It is very rare in children, though they furnish some of the most striking cases of acute gastric catarrh (p. 151). Young men and unmarried girls are extremely liable to it, and it often continues through the first half of adult life. In women it is aggravated, or sometimes begins, about the menopause; but most men suffer less after forty or forty-five, and in old age, notwithstanding loss of teeth and sedentary habits, it usually disappears; so that, as Dr Michael Foster puts it, the old man who suffered martyrdom from dyspepsia throughout his active life, "now eats with the courage and the success of a boy." This undoubted fact seems to show that while indigestible food is certainly the occasion of dyspepsia, it is not its essential cause; and also that the histological changes described above do not exist or are not operative in the majority of cases.

The mental interest and anxiety which belong to active life, appear to spoil digestion by a kind of inhibition, either of the vaso-motor or of the secretory nerves, or possibly of the muscular walls of the stomach; and this quite apart from the irregular meals and hasty eating and over-use of stimulants which often accompany business life. The careless and unharassed periods of life—youth before the toil and strife are begun, and age when both have passed—are the periods of unconscious and therefore happy digestion.

The *diagnosis* between atonic dyspepsia and chronic gastric catarrh is often unsatisfactory. But, after all, it is of little consequence, for with practical experience one learns to adjust one's remedial measures to the necessities of the case without attempting to draw fine distinctions between maladies of which the true pathology is still obscure, and far less important for their treatment than their clinical characters.

The diagnosis between chronic dyspepsia and the serious organic diseases of the stomach is of far greater importance. We must remember that their early symptoms are very commonly attributed to mere indigestion, and that the possibility that one of them may develop itself must never be overlooked in any dyspeptic case that seems to be protracted or severe. Vomited matters should always be subjected to the closest scrutiny, and we must make it a rule to examine the patient in the recumbent posture, and with the abdomen exposed to view as well as to manipulation.*

The *prognosis* in dyspepsia depends chiefly on whether its causes are or are not capable of being completely removed, on the age and sex of the patient, and on the degree of severity and the duration of the symptoms. Proper treatment scarcely ever fails to give some relief; most patients are greatly benefited, but it seldom happens that a case of long standing is permanently and absolutely cured.

Treatment.—It will be convenient to classify the forms of indigestion clinically as follows:

(1) The so-called "bilious" dyspepsia of young adults, corresponding

* I can never forget the case of a gentleman in whom, as soon as his shirt was raised, the existence of obstruction at the pylorus was indicated by the obvious peristaltic movements of a dilated stomach, but who assured me that his abdomen had not hitherto been examined, although he had been under the care of more than one specialist.—C. H. F.

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to what is called chronic gastric catarrh, with constipation, furred tongue, and frontal headache.

The first prescription for this, perhaps the commonest kind of dyspepsia, is to eat less, to take stimulants sparingly at meals and none between meals, to eat slowly, and to cease before repletion is reached.

Next come injunctions to secure as much as possible of open air, to rise early, and bathe every morning.

Thirdly, the drugs most useful are alkalies and gentle laxatives. Sir William Roberts recommends a lozenge of nine grains of carbonate of soda with one grain of common salt, to promote the flow of saliva.

In the more definitely irritative cases bismuth is useful, particularly when there is vomiting without constipation. A drachm of Schacht's solution, or ten grains of the subnitrate, may be administered three times daily. At the same time small doses of some alkali, and of morphia may be given with great advantage; a very useful formula is the Mist. Bismuthi Sedativa of the Guy's Pharmacopœia.* Another valuable remedy in cases of this kind is magnesia; it may be prescribed with three-minim doses of dilute hydrocyanic acid, and equal parts of lime-water and cinnamon-water. In long-standing cases, Dr Wilson Fox recommended the oxide of silver (in doses of one grain to two grains), alum (in doses of two to five grains), tannin or decoction of oak-bark, and matico. He disapproved the use of purgatives in cases of chronic irritative dyspepsia, and advised that, when necessary, the action of the bowels should be solicited by the daily use of enemata of cold water, although he allows castor oil in some cases, and in others the decoction of aloes, or the useful dinner pill containing aloes and extract of nux vomica.

Among the natural mineral waters of Great Britain, the most useful in cases of chronic gastric catarrh are said by Dr Fox to be those of Harrogate, Bath, and Leamington. Trousseau recommends Plombières, Vichy, and Bagnères de Bigorre, in France. Carlsbad, Marienbad, Wiesbaden, and many others are advised by German writers. The water itself is one chief element of value, apart from its more or less aperient salts.

Where there are less inflammatory symptoms with more constipation and flatulence, a sallow complexion, and yellowish fur on the tongue, sodic carbonate is indicated, together with small doses of blue pill, euonymin, or iridin, to which taraxacum or ipecacuanha may be added. A brisk purge once a week is better in these cases than frequent aperients.

(2) The chronic catarrhal gastritis of drunkards. This is characterised by nausea, thirst, morning diarrhœa, a yellow furred tongue, and the general indications of intemperance in the eyes, the skin, and the nervous system. In addition to prompt and complete abstinence, it is best treated with free diluents, soda-water, "imperial drink," and any mild bitter infusion, as buchu, that the patient will take. A blue pill every other night, and alkalies with bismuth, are valuable aids.

(3) Gouty dyspepsia. This is also probably due to gastric catarrh. It is usually benefited by colchicum and alkalies, particularly potash salts and diuretics.

(4) The cases of chronic dyspepsia with constipation and flatulence, often with heartburn and acidity, but without a furred tongue and without nausea. These may be called atonic, and are more common in women

* It must be kept in mind that the preparations of bismuth, like those of iron, give a black colour to the fæces.

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than in men. They are also seen in early cases of tuberculosis. Probably the chief cause of the symptoms is want of active movements of the stomach and bowels; this leads to distension with gas, delay of food in the stomach, slow digestion, and slow peristalsis.

The exact pathology of gaseous distension of the stomach is not yet fully made out. Hysterical flatulence probably depends in part on swallowing air; other cases result from chemical decomposition in the stomach or intestines. But most often it is probable that there is no abnormal amount of gas; the muscular walls of the containing viscera yield, and the gas naturally present expands.

In these cases the diet must be somewhat scanty, but regular and varied. It should be dry in quality, soups and broths being apt to cause flatulence; and potatoes, cauliflowers, and even bread, are often best replaced by boiled rice, spinach, and toast or biscuits. Meals should be eaten slowly, and no water should be drunk with them, but either bitter ale or a little sound wine. Exercise out of doors should be followed by half an hour's rest before a meal, lying down on a bed or couch; for a tired body does not digest well.

The most useful drug in flatulent atonic dyspepsia is nux vomica, given after food in ten drops of the tincture with soda and peppermint, or before meals in half a grain of the extract with the compound rhubarb pill.

(5) The dyspepsia of anæmia in general and of phthisis in particular. This is of the "atonic" kind, and is best treated by mild preparations of steel with strychnia, and sometimes by small doses of arsenic. In cases of chlorosis, iron and aloes are the cure for dyspepsia and anæmia together.

In many cases of atonic dyspepsia, and particularly in the later stages of the affection, the dilute mineral acids are very useful, especially the hydrochloric. In doses of ten or fifteen minims, properly diluted and taken with or after the meals, it prevents the sense of weight and oppression which would otherwise be experienced by the patient, and relieves flatulence arising from fermentation of the food. Trousseau speaks very highly of this remedy, which appears to be little known in France. He mentions that he learnt its use by sitting at dinner next to a tourist, who said that he never travelled without a little bottle of the acid, of which he took a few drops after each meal.

Pepsine, again, is often useful in cases of atonic dyspepsia; but Dr Pavy has shown that care is required in obtaining it at the druggists, since much of what is sold in London is devoid of any active properties. The acid solution of pepsine in glycerine, taken in drachm or two drachm doses after a meal, is valuable in atonic flatulent dyspepsia.

Even the milder preparations of iron (although indicated when the patient is anæmic) often disagree with dyspeptic patients, and the same is the case with quinine. When there is much flatulence, creosote, thymol, or carbolic acid is useful in the form of pills.

Dr Fox recommends Brighton as the best place for persons affected with this form of dyspepsia; next in order he places Scarborough, Dover, Folkestone, Margate, Eastbourne, Malvern, Tunbridge Wells. He also speaks highly of Ilfracombe.

(6) Children are, as before noticed, comparatively little liable to dyspepsia. So common, however, is the complaint, that even in them cases frequently call for treatment. These depend almost always on one of two causes: either unsuitable food, or general anæmia, in which the stomach shares. The first we see in the discomfort of an infant fed too soon on

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starchy food, or given material fit only for an older child; and again in the acute gastritis of a schoolboy who has eaten too many apples or tarts, or other "trash." The immediate remedy is an emetic, if the stomach has not unloaded itself, and the subsequent treatment is better choice of food in quality and in amount.

The anæmic or atonic form of dyspepsia in children is seen in cases of rachitic, tubercular, or other kinds of marasmus, and is best treated by steel wine, fresh air, and sometimes by wine or malt liquor with meals.

(7) Senile dyspepsia, when not the direct expression of organic disease of the stomach, is usually atonic, and benefited by wine and some such dinner pills as rhubarb or aloes with capsicum. The quantity of food must generally be reduced.

Diet.—As a rule, a dyspeptic should take three meals daily, at one of which freshly cooked meat should be eaten. White fish, mutton, poultry and game (but not hare or rabbit), are recommended; pork and veal, and salted or preserved meats are usually forbidden. Eggs agree well with some dyspeptic patients, whereas others are unable to take them. Vegetables must not be omitted from the dietary. Potatoes should be taken sparingly, and only if well boiled, floury and not young; turnips, parsnips, and Jerusalem artichokes—in fact, all solid roots are better avoided; but green vegetables, as spinach and asparagus, and in some cases onions, may be taken in moderation. Peas and beans are famous for causing flatulence, and so are cauliflower and other Cruciferæ. When vegetables are found to disagree, their place may be supplied by rice or macaroni. New bread should never be eaten by persons who are subject to indigestion. Sometimes it is of great importance to substitute toast or biscuits for bread. Light farinaceous puddings generally agree well with dyspeptic patients. Everyone is agreed that lobsters and crabs, cheese, nuts, and pickles are to be strictly forbidden. A large amount of fluid should not be drunk at meals. Cocoa, or milk and water, may be used as substitutes for tea or coffee. Dr Fox advises that a moderate quantity of wine (sherry, claret, hock, or champagne) should be taken twice daily; but the present fashion is to limit the patient to weak brandy (or whisky) and water with his meals. Malt liquors are often injurious, but in certain cases a light, well-hopped, and not effervescing bitter ale is an excellent stomachic; and some young women can take porter without discomfort who bear no other form of stimulant. Whatever causes flushing of the face after the meal is bad. Food is to be taken slowly, time being allowed for mastication and the due admixture of saliva; and on this account it is advisable that the patient should have his meals in company with other persons.

There are certain effects of gastric disorder, which although often associated with the ordinary symptoms of dyspepsia, yet also occur by themselves, and may be so serious as to be regarded as independent affections.

Gastralgia.—Perhaps the most important of these is *pain*. This has been already mentioned as one of the symptoms of dyspepsia, particularly in its more acute and inflammatory forms; but it may also occur without any other indication of impairment of digestion. Several names are applied to pains situated in the stomach, but unfortunately different writers use them in different senses. Cullen employed *cardialgia* for the less severe varieties which would commonly be called "heartburn" or "acidity," while

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he described as *gastrodynia* a more violent but also more transient pain, such as would usually be spoken of as "cramp" or "spasm" of the stomach. Most English writers follow Cullen in the use of these names, but the Germans employ them with meanings exactly reversed. *Gastralgia* is a term used chiefly by French writers with a very wide range of application. The late Dr Anstie proposed to limit it to a particular kind of pain, namely, to that which comes on when the stomach is empty, half an hour or so before the time appointed for a meal.

This last kind of gastric pain *before food* is recognised by all writers on the subject. It is often quickly relieved by even a small quantity of food. Sir Thomas Watson mentions the case of a clergyman whom he knew, and who was much harassed by its recurrence several times daily until he found by accident, after having tried a round of drugs, that eating a small biscuit would at once appease it. This writer also says that a drachm of the aromatic spirits of ammonia, or half a drachm of magnesia, will sometimes remove the pain in a moment as if by magic. According to Dr Budd, the gastric pain which occurs when the stomach is empty is also accompanied by slowness of the pulse and by coldness of the surface of the body; the recumbent posture relieves it, and hydrocyanic acid is the medicine which he recommends for it.

Dr Anstie regarded it as a form of neuralgia, and he speaks of strychnia as the most valuable remedy. He prescribed five or ten minims of the tincture of nux vomica three times a day, or sometimes gave $\frac{1}{80}$ to $\frac{1}{50}$ of a grain of strychnia by subcutaneous injection. One case in which this remedy effected a cure was that of a patient who had attempted suicide on account of the agonizing pain which he endured.

When gastric pain comes on soon *after* food it may be due to a variety of causes. Its diagnosis always requires great care, for the pain arising from organic disease of the stomach is usually of this kind. The strongest indication of the presence of ulceration is the circumstance that the pain begins as soon as the food is taken, and lasts until digestion is completed or until vomiting occurs. But, as we shall see, these characters may be wanting in cases of gastric ulcer, whether simple or malignant; while, on the other hand, very severe and protracted pain is sometimes complained of after every meal by nervous or hysterical persons, in whom there is no serious disease.

Abercrombie described another form of gastric pain which begins from two to four hours after a meal, and lasts for several hours; he thought that its seat was in the duodenum. But Sir Thomas Watson remarks that we can generally remove it by giving an alkali, or by letting the patient swallow a cup of warm tea; and he therefore supposes that it is due to the continued secretion of gastric juice after the food has passed through the pylorus, and adds that the onset of the pain may often be prevented by a small quantity of alkali in some aromatic water taken immediately after dinner. Trousseau speaks of this kind of pain as being often attended with a sense of sinking at the stomach, a craving appetite, and a great feeling of weakness. Constipation usually accompanies it, but sometimes diarrhœa, which he attributes to the circumstance that in cases of this kind the food is propelled into the duodenum before the digestive action of the stomach is completed. The treatment which Abercrombie found most useful consisted in giving two grains of sulphate of iron, with one grain of aloes and five grains of aromatic powder, three times daily. The sedative bismuth mixture is useful in such cases, or an alkaline lozenge may be taken.

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Pain in the neighbourhood of the stomach sometimes bears no relation to the times at which the meals are taken, or to the stage which the process of digestion has reached. Pain in the left side, under the nipple and running round to the shoulder, has been repeatedly removed by bismuth and morphia, although not a single fact could be elicited to prove it due to gastric irritation. For this kind of pain—coming on at uncertain intervals in violent paroxysms—Cullen reserved the name of "gastrodynia." Sir Thomas Watson says that it is often accompanied by a sensation of distension, much anxiety, and extreme restlessness. In females hysterical symptoms are frequently present, and the stomach is sometimes distended with enormous quantities of gas.

Gastrodynia may recur at irregular intervals for a very long period without appreciably affecting the general state of the patient's health. It cannot in such cases be due to any active disease; but probably it is sometimes the indirect result of long past pathological changes. Bamberger speaks of the cicatrices of gastric ulcers as giving rise to paroxysmal attacks of pain, and it seems exceedingly probable that they may do so by irritating the filaments of nerves embedded in them. The author once made an autopsy in the case of a lady who had for years suffered from a pain in the back, which was supposed to be connected with an abscess near the sacrum she had when a child. All the parts in front of the spine—the aorta, the vena cava, and the nerves—were embedded in a dense mass of cicatricial fibrous tissue. This had doubtless been in some way the cause of the pain. In another case the destruction of a hydatid in the liver was followed by severe pain, probably due to pressure on nervous filaments during the contraction of the cyst.

But a person may suffer from gastrodynia for many years, and an autopsy show nothing to account for it. Bamberger gives a case of this kind. It occurred in a powerful man who for nine years had been subject to frequent attacks of the most violent pain in the stomach, lasting for days, or even weeks, and attended with great prostration and temporary loss of flesh. He died of acute phthisis. A slight dilatation of the stomach was the only morbid change in that organ.

Gastrodynia has to be distinguished from several other kinds of pain. In the first place pain arising in the colon may resemble it somewhat closely; this will be discussed when we deal with colic (*infra*, p. 203).

According to Briquet, the abdominal muscles are often the seat of pain, without any affection of the subjacent parts. He lays stress on the circumstances that superficial tenderness is present, that the left recti and obliqui abdominis are the muscles principally affected, that not only their fleshy parts but also their tendinous attachments are concerned, and that dorsal pain and tenderness in the vertebral groove often exist at the same time. But we shall hereafter see that rigidity of the upper part of the rectus with tenderness is a very common effect of organic disease of the stomach, and there seems to be no reason why it should not also occur when the pain is of functional origin. In one very striking instance, a pain in the left hypochondrium, which had long resisted other treatment, was again and again removed by quinine and iron, and in that case the pain was probably myalgic. True myalgia of the abdominal muscles would be recognised by its being increased with movement of the body.

Lastly, pain situated in the epigastrium, over the stomach, may be severe, continuous, and last for a long time; but of this kind of pain

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one may say that it is seldom caused by organic disease of the stomach, and perhaps never by functional gastric disorder. There are doubtless many conditions that may give rise to it; but two in particular must always be borne in mind,—aneurysm of the abdominal aorta and incipient disease of the dorsal vertebræ. Some very striking instances in which epigastric pain was due to spinal disease are related by Hilton. One was that of a boy who for two months had been complaining of severe pain just above the pit of the stomach, and who used to walk about with his hands placed over that region, and with the body a little inclined forwards. It seemed as though he were suffering from irritation of some of the abdominal organs, and he had been treated on that supposition, but without much benefit. The pain was relieved when the boy lay down. Its seat was not to one side of the body more than to the other. Disease was detected between the sixth and seventh dorsal vertebræ, and pressure on their spines excited the pain in front. He was kept in a recumbent posture for four or five months, at the end of which time he was completely cured. Another case was seen by Mr Hilton with Dr Addison. A Westminster boy had pain at the pit of the stomach and occasional vomiting. He was found to have disease between the same two vertebræ; he too was easy when lying in bed. He was made to lie down uninterruptedly for two or three months, and from that time he got well.

True continuous and severe gastrodynia without signs of gastric disorder is often very intractable. Watson recommends the application of a mustard poultice to the epigastrium, and the administration of a carminative (such as a few drops of cajeput oil suspended in mucilage) or of sedatives, among which he assigns the chief place to hydrocyanic acid. Opiates are often necessary, but the first thing is to ascertain, if possible, the cause of the pain.

Alterations of the appetite are often due to gastric disorder. Loss of appetite (or *anorexia*, as it is called*) may indeed be a symptom of almost any kind of disease. It doubtless depends upon the loss of digestive power which accompanies so many morbid conditions, particularly those attended with fever. Beaumont found that when Alexis St Martin was feverish, the secretion of the stomach was diminished or suppressed, and food remained undigested for twenty-four or even forty-eight hours. Accordingly, patients suffering from acute diseases do not have regular meals, and are allowed only fluid nourishment in small quantities and at intervals which are often extremely short. In other persons the appetite may be greatly improved by the administration of bitters and mineral acids before meals; a pill of capsicum, nux vomica, and gentian is very useful for this purpose; but such medicines should never be prescribed until it is ascertained that the anorexia is not due to gastric catarrh, for if present this must be first corrected.

Excessive appetite is called *bulimia*.* It is said to be produced by the presence of worms in the intestines, and is a symptom in diabetes and in some diseases of the mesenteric glands. It has already been mentioned as accompanying one of the forms of gastralgia.

Lastly *pica** is the name given to a perverted state of the appetite, in which substances without nutrient properties are swallowed greedily, or

* *Anorexia* (*ἀνορεξία*), want of desire of food.—*Bulimia* (*βουλιμία*), excessive hunger.—*Pica* is a modern translation of *κίσσα*, the jay (*Pica glandaria*), a word applied by the Greek physicians to the indiscriminate greediness of a morbid appetite.

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filthy matters, such as excrement, horse-dung, &c. It occurs either as a variety of the "longings" of pregnant women, or in hysterical, half demented girls, or in maniacs.

Regurgitation.—Other symptoms of gastric disorder are those which consist in the regurgitation of gaseous or fluid matters upwards through the œsophagus. Of these the simplest form is *eructation* or belching. Generally gas alone arises, but sometimes a small portion of imperfectly digested food as well, which, however, is instantly carried back into the stomach. In rare cases a large part of the food is habitually brought back into the mouth, to be deliberately remasticated and swallowed a second time. Some years ago there was a patient of Dr Pavy's in Guy's Hospital who possessed this habit of ruminating. Dr Copland collected a number of cases of this kind, some of which came under his own observation. In one patient the rumination began in from fifteen minutes to an hour after almost every meal. Each bolus of food came up during an act of expiration; it had the same taste and flavour as when first swallowed; there was neither nausea nor pain; he masticated it a second time with pleasure. No treatment seems to help persons who ruminate, beyond advising them to eat very slowly and with moderation.

When only a few drops of gastric juice are regurgitated the hot acid taste is unpleasant in the mouth, but there is no true vomiting. A little secretion is accidentally belched up, entangled in the bubbles of gas which form the eructation.

Pyrosis.—A somewhat analogous affection is what is called waterbrash, or *pyrosis.** This is not very common in England, but the people of Scotland are very liable to it, and still more so those of Norway, Sweden, and Lapland. It is supposed to be caused in the Scotch by the oatmeal of which they eat so largely. Cullen, who was very familar with pyrosis, described it as coming on usually in the morning when the stomach is empty. According to him, it begins with a severe pain at the pit of the stomach, which, after continuing for some time, brings on the eructation of a thin watery fluid in considerable quantity. The fluid is sometimes acid, but is often absolutely tasteless. The repetition of the eructation seems at length to give relief to the pain, and the attack is then at an end. It is apt to return more or less frequently for a considerable length of time. Cullen says that the complaint occurs chiefly among the lower classes, in women more often than in men, and between puberty and middle age rather than at any other period of life. He speaks of it as often unattended with any symptoms of dyspepsia.

In England pyrosis seldom appears as an independent malady, apart from other effects of gastric disorder. Sir Thomas Watson speaks of one remarkable case in which no less than three pints of a thin tasteless liquid were brought up every day.

There is difference of opinion as to the nature of this secretion, and possibly it is not always the same. If the liquid brought up is acid (as the term pyrosis might seem to imply) the case is really one of eructation of gastric juice above described. But "waterbrash" is ejection in often much larger quantity of a clear neutral or alkaline fluid, neither gastric juice, nor mucus, nor food. Dr Handfield Jones regards pyrosis as a

* Etymologically, pyrosis ($\pi\bar{\upsilon}\rho$ = fire) should mean the same as heartburn; but in England it is never used in this sense. In Greek, the word means inflammation.

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catarrhal affection of the gastric mucous membrane, analogous to bronchorrhœa. But the liquid is often ejected without any effort of vomiting; and, moreover, this sometimes occurs immediately after or even during a meal. Dr Chambers gives the case of a retired surgeon, who often had to leave the room at mealtimes, and would throw off as much as five or six ounces of frothy clear liquid. The contents of the gastric cavity never came up at the same time, although the ejection of the fluid sometimes made him retch. There seems, therefore, to be much probability in the opinion suggested by Dr Chambers that the fluid is really saliva, which trickles down the œsophagus, and, being arrested by spasm of the cardiac orifice, collects there until it gushes back into the mouth. Frerichs, indeed, is said to have detected sulpho-cyanide of potassium in liquid of this nature. One may perhaps object that Dr Pavy, who believes that the fluid is often secreted by the stomach, found it to have the power of digesting proteids, but this might have been due to the admixture of a little gastric juice. The last-named observer showed that water injected into the stomach of an animal quickly becomes charged with pepsine, so that the possession of digestive power might be explained by supposing that the saliva had passed down into the stomach before being ejected.

Sir Wm. Roberts has carefully observed this phenomenon, has analysed the liquid, and found it to possess diastatic powers, so that at least in his cases there can be no doubt that it was saliva, rapidly secreted, swallowed, and brought up again ('Lectures on Dietetics,' p. 81). He calls this disorder paroxysmal pyrosis with cramp of the stomach.

The preparations of bismuth are very useful in the treatment of pyrosis. Sir Thomas Watson recommends opium combined with an astringent, as, for instance, in the pulvis kino compositus. Dr Pavy is in the habit of prescribing the liquor opii sedativus in eight-minim doses, with an ounce of the compound infusion of gentian, three times a day.

*Vomiting.**—Another effect of gastric disorder is *emesis* or *vomitus.* This has already been mentioned as occurring in certain forms of dyspepsia, and we shall find that it is a principal symptom of the organic diseases of the stomach. But it may also be met with independently.

The act of vomiting is usually preceded by a peculiar feeling, which is termed *nausea* (literally "sea-sickness").† In persons unaccustomed to being sick, this is accompanied with sensations of giddiness or faintness, coldness of the surface, pallor of the lips and face, and a small and feeble pulse. After a pause, salivation, and then retching occurs, and is followed by the expulsion of the contents of the stomach. But vomiting is not always preceded by such painful sensations and efforts. Some persons, chiefly women, are subject for years to occasional sickness, attended with scarcely any discomfort, like that of infants. This kind of vomiting is especially apt to occur at night or in the early morning. So far it resembles the chronic vomiting of alcoholic dyspepsia, but it is a mistake to suppose that morning sickness is always an indication of intemperance.

Sometimes habitual vomiting, independently of gastric pain and of any indication of dyspepsia, may reach such a point as to be alarming, and even dangerous to life. At Guy's Hospital we have had several cases of this

* *Gr.* ἔμεσις.—*Lat.* Vomitus.—*Fr.* Vomissement.—*Germ.* Erbrechen.

† The vomiting of sea-sickness appears pretty certainly to be cerebral in pathology, and not connected with gastric catarrh.

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kind, and Sir William Gull used to describe them as the vomiting of women with "mad stomachs." Some striking instances are related by Dr Chambers. In one of them the affection had been of three years' duration, and it was stated that the food was always returned, unchanged in appearance, within ten minutes after being swallowed. Another patient was said for five years to have hardly ever kept down a whole meal. This kind of sickness is almost confined to the female sex, and occurs chiefly in young women. It is very frequently associated with disorder of the menstrual functions; in one of Dr Chambers's cases, just quoted, it was attributed to a chill, by which the catamenia had been suppressed for several months. These patients, too, are often hysterical. They sometimes reject the food before there has been time for it to be swallowed. Like other neuroses, this kind of vomiting is sometimes catching. But the most remarkable feature of many of these cases is that, although the vomiting is so constant, yet there is little or no loss of flesh. It is evident that a good deal of food must be retained by the stomach.

In cases of this kind the *diagnosis* cannot be carried further than "irritability of the stomach," or "hysterical vomiting." But it is of extreme importance to remember that there are several organic diseases—affecting parts far distant from the stomach—of which a similar form of vomiting may be one of the symptoms. There are, indeed, few serious diseases in the course of which the stomach does not occasionally reject its contents. But the present point is that in certain diseases irritability of the stomach is often the earliest, and, for a time, the only indication that the patient is ill.

The most important of these is perhaps incipient *phthisis*. The lungs should always be most carefully examined before one arrives at the conclusion that habitual vomiting is merely due to functional disturbance of the stomach; and, if there be any other ground for thinking that tubercular disease is likely to develop itself, a guarded opinion should be given, even though there may be no discoverable stethoscopical sign of its presence.

Another morbid state, of which vomiting is a principal symptom, is that due to *Addison's disease* of the suprarenal capsules.

Again, the possibility of the presence of *cerebral disease* must never be overlooked; in some cases of abscess of the brain there are few other symptoms than sickness. According to Romberg, the vomiting which accompanies affections of the brain is characterised by the absence of nausea and of retching, and by its occurring when the head is moved, as in swinging, shaking, or stooping, or in suddenly rising; it also occurs when the patient is erect rather than when he is recumbent. Affections of the cord seldom cause gastric disturbance.

In female patients the possibility of *pregnancy* must never be forgotten, particularly if the vomiting should be only of a few weeks' duration. All these varieties of sickness are commonly attended with constipation.

When chronic vomiting and diarrhœa occur in the same case, the presence of some form of *Bright's disease* should be suspected. In one instance the observance of this rule led to a correct diagnosis which might not otherwise have been reached, for the quantity of albumen in the urine was very small, and might have been overlooked.

Another possible cause for the existence of vomiting and diarrhœa in the same patient is chronic *poisoning* by small doses of arsenic or antimony, or other irritant poison.

The diagnosis of primary, neurotic "irritability of the stomach" having

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been reached, its *treatment* is often highly successful. In many cases the best plan is to give the stomach entire rest for two or three weeks, the patient being fed solely by enemata. Or minute quantities of milk may be administered by mouth, as in the well-known case related by Dr William Hunter. A boy was brought to him in a state of the most extreme emaciation, who vomited up almost everything that he swallowed, in spite of the treatment of three very eminent physicians. Dr Hunter recommended that only a single spoonful of milk should be given at a time. The boy was never sick afterwards; he gradually became able to take more and more nourishment, and he ultimately recovered entirely. A most graphic and interesting account of the case is given in the sixth volume of Dr Hunter's 'Medical Observations and Inquiries.'

In neurotic cases, "an enforced administration of more and better food is often the surest as well as the shortest road to deliverance from gastric hyperæsthesia."*

The application of a blister to the epigastrium is often highly serviceable in cases of this kind, particularly if it be kept open by savin ointment. Sedatives may often be prescribed with advantage, but particularly morphia or opium. Occasionally the oxalate of cerium (in doses of one or two grains) has succeeded when bismuth and hydrocyanic acid had failed. Creasote is sometimes serviceable. In the cases related by Dr Chambers a daily shower-bath appeared to do much towards the restoration of vigour of mind and body, and in some of them the preparations of valerian were useful. Lately two, three, or four drops of tincture of iodine in a teaspoonful of water has become a favourite remedy in our wards, and it certainly appears sometimes to be efficacious.

In the most obstinate hysterical cases the best treatment is that recommended in anorexia nervosa (vol. i, p. 822)—seclusion and shampooing.

Other symptoms.—Gastric disorder may manifest itself by producing reflected disturbance of other parts. Thus *palpitation* of the heart is one of the commonest effects of flatulence or of an overloaded stomach.

A *stomach-cough* is believed to be a frequent malady; but most cases are probably really early phthisis with tubercular dyspepsia.

Hiccough or *singultus* is due to a sudden spasmodic contraction of the diaphragm, repeated at more or less regular intervals, and attended with a clicking sound which is caused by the abrupt passage of air through the glottis. Its recurrence can often be stopped by holding the breath. Hiccough is not usually a matter of any consequence, and lasts only a few minutes, or at most an hour or two. But in dangerous illnesses it sometimes continues for days together, so as to exhaust the patient, and appear to be the immediate cause of death. Indeed, a persistent hiccough in chronic diseases has always been regarded as of bad omen. It is very difficult to cure.

Dr Edward Liveing had a man past middle age under his care in whom hiccough occurred in paroxysms of twelve hours' duration about twice a week for four years; and he quotes a case of a little girl of twelve, who for nearly three years was subject to fits of violent hiccough, even during sleep, which lasted from ten minutes to an hour, and returned three or four times during a day and night. She was cured after taking turpentine.

* Sir William Roberts, in his admirable "Address on Some Points in Dietetics" ('Brit. Med. Journ.,' Oct. 18, 1890.

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In all probability hiccough is due to the presence in the stomach of food which is incapable of being digested.

No one can make frequent autopsies without observing how often the stomach contains a pint or more of brandy and egg mixture, or of some similar substance, which must include all that had been given by the nurses for some hours before death. In one case of fever, as the end was approaching, the relatives began to flatter themselves with vain hopes that the patient would recover because he took all his nourishment; but it evidently was not absorbed, for a splashing sound could be constantly produced by manipulating the upper part of the abdomen. It probably embarrasses the heart, and hastens or at least disturbs the hour of death.

Hæmatemesis, or vomiting of blood, is very rare except as the effect of acute irritant poisoning or as the result of some organic lesion. This, therefore, appears to be the most fitting place to discuss it, before we pass on to describe the organic diseases of the stomach.

Diagnosis.—In investigating a case in which blood is said to have been vomited one must, in the first place, make sure that the blood was really ejected from the stomach. Strange as it may appear, there is often considerable difficulty in distinguishing between hæmoptysis and hæmatemesis, particularly when one has to rely upon the statements of the patient, and does not see the blood actually brought up. When blood escapes copiously into the air-passages it may issue from the mouth in gushes; while some of it may pass back into the pharynx, and there excite retching and vomiting. Indeed, a portion of the blood may run down the œsophagus: in an autopsy on a child who died of hæmoptysis, the author once found an ounce of coagulated blood in the stomach. On the other hand, when sudden and profuse hæmatemesis occurs, the blood may irritate the larynx in passing over it, and so provoke a paroxysm of cough. The patient's statements, therefore, may afford a very unsafe basis for distinguishing between vomiting of blood and hæmoptysis; the diagnosis must be founded first upon the character of the blood brought up, whether bright, frothy, and mixed with sputum, or dark, clotted, and partially digested; and secondly, upon a consideration of the conditions in which these two forms of hæmorrhage occur.

The stomach is a large cavity into which a great quantity of blood may ooze before it excites vomiting; hence hæmatemesis is commonly preceded by the characteristic symptoms of hæmorrhage—pallor of face, dimness of vision, giddiness, or fainting. On the other hand, hæmoptysis occurs suddenly, the patient finding the hot blood in his mouth or feeling a tickling in his throat, or a sensation as of bubbling in the chest, immediately before he begins to cough up the blood.

After hæmatemesis, any blood that may be left in the stomach passes downwards, and (if not completely digested) it is ultimately discharged from the bowels. But when any part of the air-passages is the seat of the hæmorrhage, the blood that is almost necessarily sucked into the smaller bronchial tubes is afterwards got rid of by expectoration, and the sputa remain discoloured for hours, or even for several days.

The appearance of the blood after its ejection is different in cases of hæmoptysis and of hæmatemesis. In the former it is usually frothy from admixture of air; it has an alkaline reaction, and it is of a bright red or "arterial" hue. In the latter it is acid and darker coloured, or brown,

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from being mixed with the gastric juice. Dr Chambers mentions a case in which the application of test-paper to a woman's clothes over which she had thrown up blood, showed that it had come from her stomach. When the hæmorrhage is very profuse—and particularly if a large artery be opened—blood rejected from the stomach may be fluid and of a scarlet colour; but whenever it is retained for any length of time in the cavity before being vomited it undergoes a peculiar change: it often coagulates, and sometimes solid clots are rejected which are so tough as almost to choke the patient; more frequently the blood, whether clotted or not, is acted on by the gastric juice, the acid of which decomposes the hæmoglobin and produces acid hæmatin. The presence of comparatively small quantities of blood in vomited matters thus gives them an appearance like that of coffee-grounds, while pure blood altered in this way has more or less the appearance of tar.

Blood which passes from the stomach into the intestines is found in the stools altered in exactly the same way. It is often perfectly black or, as it is called, "tarry." Evacuations having this character were supposed to consist of "black bile" by the older writers, who described them as characteristic of a special disease which they called *melæna*. But it has long been known that this affection is one of the results of hæmorrhage into the alimentary canal. Addison taught that when blood having this black colour was discharged from the bowels, the source of the bleeding was always the stomach. An exception to this rule ought perhaps to be made for the duodenum above the entry of the alkaline bile and pancreatic juice, although it is doubtful whether the blood discharged from an ulcer in that part of the bowel would be sufficiently long in contact with the gastric juice to undergo the peculiar change. But when the hæmorrhage occurs from the rest of the intestine the blood is always more or less distinctly red.

The dark appearance which the fæcal evacuations so constantly present when a patient has been taking a preparation of iron or bismuth medicinally must be distinguished from that due to blood. It is of a more slaty hue, and the fæces are as a rule remarkably dry. In a doubtful case one might apply the guaiacum test or that of the spectroscope.*

It is important to note that hæmorrhage into the stomach may, and often does, occur without any blood being vomited. Thus a case was observed at Guy's Hospital in which a patient (who had once before brought up a large quantity of blood) became blanched, called out that she was dying, and expired in twenty-five minutes, after a convulsive seizure. The stomach was full of clotted blood. Other instances of a similar kind have occurred at the hospital, and one such is mentioned by Sir Thomas Watson. Doubtless, therefore, it is a frequent occurrence for small quantities of blood to escape into the gastric cavity without exciting vomiting. One can, indeed, see no reason why small quantities should be rejected unless there be also some other condition making the stomach irritable. Up to a certain point, it is probable that blood undergoes digestion and is absorbed before it is passed down the whole length of the intestine; so that the stools may

* The detection of iron in an acid solution of the dark pigment by yellow prussiate of potash would distinguish melæna from the effect of bismuth. On one occasion a hysterical or malingering schoolboy, whose case is referred to in the first volume (p. 822), said that he had passed blood from the bowels, and produced very dark motions. There was no evidence of disease, and it was found that the scybala were of natural colour, except on the surface. The stools gave no iron reaction, and the colour was found to be due to a silver solution which had been poured into the nightstool from a bottle used in photography.

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themselves fail to afford evidence of the hæmorrhage. Still, whenever patient's symptoms can possibly be attributed to bleeding into the stomach the fæces should be most carefully examined.

A case in point is given by Dr Chambers. A woman aged thirty-three who had suffered from well-marked symptoms of gastric disease, grew rapidly weaker and paler, and her tongue became dry and furred. It was long suspected that she was passing blood, but she constantly denied it. She was made an in-patient, and then it was discovered that blood came from the bowels every time they acted.

On the other hand, true hæmatemesis may occur without there being in reality any hæmorrhage from the vessels of the stomach; so that in investigating a case in which blood is vomited, one must in the first place determine whether the blood comes from the gastric blood-vessels or from some other source. Attacks of hæmatemesis have sometimes been (so to speak) manufactured, the patients having first secretly drunk the blood which they afterwards vomited in the presence of others. Sir Thomas Watson mentions two cases of this kind. Again, when blood escapes into the back of the mouth or into the pharynx it is often swallowed by the patient unconsciously, particularly during sleep; and the first indication of the fact may be the occurrence of profuse hæmatemesis.

But in most cases of hæmatemesis the blood really comes from the blood-vessels of the stomach; and we have now to consider its causes.

Ætiology.—In the first place gastric hæmorrhage may occur in certain *general diseases*, as a consequence (it is supposed) of changes in the blood itself: for example, scurvy, purpura, malignant smallpox, yellow fever, acute yellow atrophy of the liver, and the grave forms of anæmia, leuchæmia, Hodgkin's disease, and Addison's idiopathic anæmia. Then, again, it may be a subordinate symptom of organic *disease of the heart*, or of the high arterial tension produced by chronic *Bright's disease;* but in the latter case the arteries are often morbidly brittle as well. It is said, too, that hæmatemesis may be caused by *disease of the spleen.* Sir Thomas Watson quotes from Latour the case of a man whose spleen was immensely enlarged, as the result of obstinate ague. Latour foretold that hæmorrhage from the stomach would occur, and his prognosis was justified by the result. The bleeding recurred several times; in a month the spleen could no longer be felt, and the patient had good health for twenty-five years afterwards. Sir Thomas Watson thinks that he has more than once seen the spleen diminish in size in proportion as blood was poured out of the stomach. We may doubt, however, whether in his cases the spleen was primarily diseased, and whether the enlargement of that organ and the hæmatemesis were not joint results of portal congestion, itself due to hepatic disorder.

The two principal organic diseases of the stomach—simple ulcer and cancer—are each frequently attended with hæmatemesis. In cases of *cancer*, however, it very rarely happens that any considerable quantity of blood is vomited until the disease has reached an advanced stage, or has, at any rate, declared itself by well-marked symptoms. On the other hand, a *simple ulcer* of the stomach is sometimes latent up to the time when a large hæmorrhage takes place; the patient having either had no symptoms previously, or only such slight indication of gastric disorder as appeared to be of no consequence. Perforation of a gastric ulcer is particularly common in young subjects; sudden large hæmorrhage often occurs in persons advanced in years. This kind of hæmatemesis is very seldom immediately fatal. In

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some cases it returns at intervals of a few hours, and the patient lives five or six days. Sometimes no fresh bleeding occurs for several weeks or even months. The ulcer generally presents certain special characters which will be fully described further on, but Dr Murchison has placed on record two cases, in each of which it was a mere pore-like aperture leading directly into a large branch of artery. A preparation of a similar kind is contained in Guy's Hospital museum. It is worthy of note that in these three instances the seat of the ulcer was not the lesser curvature, but the cardiac pouch of the stomach. A small ulcer of this kind might easily be overlooked, and it is possible that such may have been the explanation of some of those cases in which a *post-mortem* examination has failed to reveal the source of the hæmorrhage. Some time ago a carman, aged thirty-two, who was in the hospital for gout and albuminuria, died suddenly from hæmatemesis. The stomach was full of blood, but we could not discover from what part of the stomach it had come. The vessels of the stomach were not found much congested, for the hæmorrhage had doubtless emptied them; and the liver appeared to be quite healthy.

Vomiting of blood is very frequent in cases of *cirrhosis of the liver*, and it is often the earliest symptom. Many cases in which ascites has already appeared terminate by sudden and fatal hæmatemesis.

Other forms of chronic hepatic disease causing portal congestion may have the same effect, adhesive pylephlebitis, perihepatitis, syphilitic gummata, &c.; but all these causes are comparatively rare.

Hæmatemesis from rupture of an aneurysm into the stomach has been recorded.

Systematic writers mention among the varieties of hæmatemesis one in which the effusion of blood is *vicarious* to the menstrual flow. Sir Thomas Watson relates a case of this kind which came within the knowledge of the late Dr Latham. A girl about the age of fourteen became the subject of hæmatemesis, recurring at monthly periods. She married without ever having menstruated, and became pregnant; the hæmatemesis then ceased, and did not return until she had been confined, and had suckled and weaned her infant.* All modern clinical observers are agreed that vicarious hæmatemesis is, to say the least, exceedingly rare. And probably many cases which were formerly supposed to be of this kind would now be differently explained.

Another cause for hæmatemesis, independent of visceral disease, is atheroma of the blood-vessels. In one case under the writer's observation it followed profuse epistaxis, and was itself the precursor of cerebral hæmorrhage.

The occurrence of profuse hæmatemesis in a person who presents no other well-marked symptoms of disease is, in most cases, the result either of a latent ulcer, or of congestion of the stomach from early cirrhosis of the liver. The next question is whether these two conditions can be distinguished from one another. And this must be answered in the negative. Both Dr Murchison's cases, above mentioned, occurred in persons who had been intemperate; one of them was a plethoric woman, aged fifty, the other a soldier aged twenty-eight, whose liver was cirrhosed. It might well have been thought that in both instances the hæmorrhage was due to mere congestion.

* Sir Thomas Watson also quotes Mr North as having met with two instances in which suppressed menstruation was followed by repeated and at length fatal hæmorrhage. I have searched in vain for the original record of these two cases, so that I cannot tell whether the presence of a gastric ulcer was disproved by an autopsy.—C. H. F.

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There remain certain unexplained cases of large and repeated hæmatemesis occurring in persons otherwise healthy. The writer has seen more than one case of the kind, in young and temperate men, without the pain and vomiting which usually accompany gastric ulcers.

Treatment.—The question of diagnosis is one of no little importance, for the practice recommended in cases of hæmatemesis from gastric congestion is hard purging. Sir Thomas Watson directs that five grains of calomel should be given every night and a black draught every morning, till the stools lose their pitchy colour; and he says that he has pursued this plan with perfect success, even when the patient had been blanched by previous hæmorrhages, and when the pulse was feeble and irregular. But it is evident that such treatment must do harm if the blood has come from an ulcerated artery; and, if the case is one of mere venous congestion, the occurrence of hæmorrhage shows that the vessels are on the way to relieve themselves, even if they have not already done so. The administration of astringents at this stage might be injurious. The best course, therefore, is to wait a few hours, or even for a day or two, until we see whether the hæmorrhage returns. Although the patient should pass several tarry evacuations in succession, this is not a proof that bleeding has occurred more than once, for they may all have arisen from a single hæmorrhage.

When, however, repeated attacks of hæmorrhage occur in a person already blanched by loss of blood, it is clear that they depend on something more than portal congestion; astringents must then be used, and acetate of lead may be prescribed in doses of three or four grains, with a quarter of a grain of opium, every two or three hours. Some writers have spoken highly of oil of turpentine, twenty or thirty minims of which are given every four or six hours. Other valuable styptics are gallic acid and dilute sulphuric acid. Of the former ten grains may be administered every two or three hours, or even oftener; of the latter ten or twenty minims. Dr Chambers records a case in which it appeared certain that a gradual oozing of blood, causing continued melæna, was arrested by dilute sulphuric acid, with Battley's solution of opium.*

The patient may suck small pieces of ice, but he should be allowed to take scarcely anything into his stomach. Starvation is indeed the cardinal point in the treatment of hæmatemesis, nutrient enemata being given if support appears necessary. If the hæmorrhage is profuse the patient's head should be kept low. The application of a bladder of ice to the epigastrium is often serviceable. Brinton recommends that it should be removed when it has been kept on for a few minutes, and that it should be reapplied from time to time; but no harm has arisen when it has been left in its place for several hours continuously.

GASTRIC ULCER.†—We have seen that vomiting, gastric pain, and hæmatemesis may each or all be symptoms of ulcer of the stomach.

Minute hæmorrhagic erosions are often seen in cases of advanced cardiac disease, and both in enteric fever and in diphtheria the surface of the

* It is doubtful whether sulphuric acid can act as an astringent in cases of diarrhœa, of hæmoptysis, or of profuse sweating; for in medicinal doses it must always be neutralised by the bile and pancreatic juice, or, if absorbed from the stomach, by the soda of the blood. It may be of service when applied directly to the mucous membrane of the stomach; even in cases of hæmatemesis, however, it is rarely prescribed without opium, catechu, or some other astringent, so that it is difficult to judge of its efficacy.

† *Synonyms.*—Abercrombie's ulcer—Round ulcer—Simple chronic ulcer of Cruveilhier—Perforating ulcer of Rokitansky.

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stomach sometimes, though very rarely, presents a number of small ulcers.

In these cases the ulceration of the stomach is due to an acute process, but we occasionally meet with a large number of chronic ulcers. A striking instance of this kind occurred some years ago at Guy's Hospital in a man of colour aged thirty, who had recently come from the Southern States of America. He died of thoracic aneurysm, after an illness of twelve months' duration, his principal symptom having been vomiting of his food about an hour after its ingestion. Dr Moxon found that almost the whole of the stomach was diseased. There were numerous recent ulcers with raised irregular edges, and there were also many thick, puckered cicatrices.

Anatomy.—The affection now to be described differs altogether from these secondary and multiple ulcers. It is limited to a small part of the surface of the stomach. Most frequently there is only a single ulcer; sometimes there are two; very rarely more. Rokitansky found the ulcer solitary in sixty-two out of seventy-nine cases, and in twelve of the rest there were two. When there are more than one, they differ in size and in other characters, so as to prove that they began at different times.

One of the most remarkable circumstances connected with gastric ulcers is that their seat is, in the great majority of cases, along the lesser curvature of the stomach. Sometimes an ulcer lies across the curvature itself; more often it is situated either in the anterior or posterior wall, but almost always close to that line. According to Brinton, ulcers are found on the posterior surface of the stomach eight times as often as on its anterior surface. But our *post-mortem* records at Guy's Hospital by no means bear out this statement. Not infrequently two ulcers are found in the same stomach exactly opposite one another, one on each surface. Since they generally appear to be of different dates, it has been supposed that one of them has been set up secondarily to the other, as the result of its coming into contact with the opposed surface of mucous membrane. Sometimes the pylorus is the seat of the ulcer, sometimes the cardiac pouch. Sometimes, lastly, an affection exactly similar occurs in the first portion of the duodenum; and it is convenient to take it with ulcer of the stomach.

A gastric ulcer has a sharply-defined edge, at first entirely free from thickening. It has the form of a flattened cone, the base corresponding with the mucous surface of the stomach. It is often described as "punched-out," on account of its regular circular form, and the evenness of its margin. Its floor may be formed by the muscular coat, or the ulceration may extend through this, forming a pit, which is always considerably smaller than in the mucous membrane, and at the bottom of which the peritoneum is visible. Very frequently, unless a different process should be started, the serous coat in its turn becomes attacked; a minute yellow slough forms; and the detachment of this allows the contents of the stomach to escape into the lesser bag of the peritoneum or into the general abdominal cavity, setting up fatal diffused peritonitis. The acute perforating ulcer is seldom larger than a sixpenny-piece. Not infrequently, instead of eating its way through the coats of the stomach, it erodes some large vessel, and thus gives rise to hæmatemesis.

So far there is nothing in the character of a gastric ulcer different from those of a similar affection occurring in other parts of the alimentary canal, as is sometimes (though rarely) the case. Thus Mr Flower has recorded an instance in which a small round ulcer developed itself in the œsophagus, and

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passed straight through into the descending aorta. In the duodenum similar ulcers occasionally follow severe burns of the skin, as the late Mr Curling first pointed out; their occurrence is, however, extremely rare, and isolated cases have been published of similar ulcers in the jejunum, the ileum, and the descending colon.

If a gastric ulcer becomes chronic, it acquires further characters which are almost peculiar to it. Its edge, although still perfectly even and regular, becomes thickened; and, for a little distance beyond, all the coats of the organ are matted together. These changes evidently depend on the occurrence of a chronic inflammatory process. The peritoneum covering the floor of the ulcer also becomes thickened and opaque, and adherent to whatever part may be opposed to it.

From the locality affected by the chronic gastric ulcer its floor almost always becomes attached either to the under surface of the left lobe of the liver, if it be in the anterior wall of the stomach, or to the pancreas and the adjacent connective tissue and vessels, if it be on the posterior wall. Thus perforation of the serous cavity is for the time prevented, while the ulcer gradually increases in size. Its growth in different directions is not always uniform, and thus it often loses its circular shape, and becomes oval or irregular in form. This last, however, is frequently due rather to the circumstance that when two or more ulcers are present they come into contact as they grow larger, and finally coalesce. As before mentioned, two ulcers are often found just opposite to one another, one on each side of the lesser curvature; these, when they run together, give rise to a single sore of dumb-bell shape. The size to which an ulcer of the stomach attains is sometimes very considerable; the 'Pathological Transactions' contain a record of one which measured five and a half by three inches. While thus expanding in circumference gastric ulcers also increase in depth. The peritoneum is gradually worn through where it is adherent, and the surface of the pancreas or of the liver comes to form part of the wall of the stomach. This at first takes place only at a small spot, but the area of adhesion and that of destruction gradually become more extensive. Thus the greater part of the pancreas may in time become exposed in the floor of the ulcer; it is covered only by a thin film of connective tissue, through which its lobulated character can be plainly identified.

In comparatively rare cases the floor of an ulcer in the anterior wall of the stomach becomes adherent, not to the liver, but to the abdominal walls, and these may in time be perforated, so that a *gastro-cutaneous fistula* is formed. Murchison collected twenty-five cases of this kind ('Med.-Chir. Trans.,' vol. xli), of which, however, only twelve were originally instances of simple gastric ulcer; six of them were cases of cancer, and in seven the penetration of the walls of the stomach was due to wounds or injuries of the corresponding part of the surface of the abdomen.* A gastro-cutaneous fistula may remain open for several years. It sometimes closes of its own accord.

In the only case which has come under the writer's care the nature of the epigastric ulcer was proved by its discharging a clear liquid of strong acid reaction. Although firm adhesions had taken place, death ensued as the result of profuse hæmatemesis, possibly from a second ulcer. It was

* Of these last, the most remarkable of all is perhaps one recorded by Dr Murchison himself of a woman who for three years kept a penny pressed into the sore left by a seton, until an opening into the stomach was formed.

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remarkable that two of this patient's children subsequently died of gastric ulcer at about the age of twenty.

But, as might be expected, this process of adhesion of the floor of the ulcer to different parts is by no means unattended with risk. In the first place the protective process of adhesion may at any time fail to keep pace with the spread of the ulceration, and perforation into the peritoneal cavity may take place, or the adhesions may be broken through by some muscular effort made by the patient, when the same result follows. For obvious reasons perforation is more apt to occur when the ulcer is in the anterior than in the posterior wall of the stomach; indeed, in the former position ulcers seldom attain any considerable size.

In other cases danger arises from the penetration of blood-vessels. We have seen that the recent "punched-out" ulcer often erodes an artery of some size; but in the chronic cases now described it is no uncommon thing to see a large artery, or even more than one, with its coats abruptly cut across, lying in the floor of the ulcer, and plugged with a little cylinder of clot that can be pushed out with little difficulty. In other cases, in which death has been directly due to hæmorrhage, the vessel is patent. The artery is sometimes a branch of the coronary artery of the stomach, or the trunk of that vessel, or a pancreatic branch of the splenic artery. Even the trunk of the large splenic artery itself is not infrequently penetrated by a gastric ulcer.

Happily, another change to which a gastric ulcer is liable is cicatrisation. This occurs not infrequently. Indeed, one seldom sees a large ulcer which has not healed over in some parts of its surface. Brinton speaks of cases in which the whole extent of the ulcer has been found cicatrised, with the single exception of a point in the centre occupied by an eroded artery, hæmorrhage from which had caused death. But in most instances, when a gastric ulcer heals, the patient has good health afterwards. Should he die from some other disease, the cicatrix varies in appearance according as the coats of the stomach were more or less deeply and widely destroyed; it may show merely a little thickening of the submucous tissue, or it may form a hard, puckered mass, with radiating processes extending into the surrounding mucous membrane.

When an ulcer is seated at the pylorus its cicatrisation may give rise to narrowing of that orifice, and obstruct the passage of food through it. The result is that the stomach becomes dilated and hypertrophied, exactly as when the pylorus is narrowed by scirrhous growth.

Again, the cicatrix of a large ulcer occupying the middle of the stomach may constrict it, and so cause what is termed an hour-glass contraction.

Pathology and origin.—The credit of having originally described gastric ulcer is commonly assigned to Cruveilhier, who published his account of it in 1830; or to Rokitansky, whose work appeared in 1839. But Dr Abercrombie had previously pointed out all its distinctive characters in 1828. It must, however, be admitted that Rokitansky laid the foundation of the most modern view in regard to this affection, by suggesting that it arose out of a hæmorrhagic erosion. Virchow, in 1853, adopted this hypothesis and developed it. He attributed the destruction of the coats of the stomach to the corrosive action of the gastric juice. This, he said, cannot dissolve the mucous membrane so long as the circulation is maintained, for the alkaline blood will neutralise the acid as it penetrates the tissues. He therefore supposed that the starting-point of the affection was some morbid change in the blood-vessels of that part of the stomach, whether obliteration

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of an artery or obstruction of a vein. He also traced the conical form of the ulcer to the distribution of the tuft of vessels arising from a single arterial rootlet. And he discovered a further argument in the fact that when perforation occurs, the aperture in the serous coat is always to be found in a particular direction, away from the centre of the ulcer. This he attributed to the circumstance that the apex of the vascular cone is likewise excentric, being always directed towards whichever is the nearer of the two curvatures of the stomach, along which the main arterial trunks run. Virchow's hypothesis has since been accepted by many other writers. Panum endeavoured to support it by injecting little globules of wax into the branches of the abdominal aorta in dogs. He found that when they made their way into the arteries of the stomach the mucous membrane presented ulcers which resembled an early stage of idiopathic gastric ulcer.

It is evident that Virchow's theory consists of two distinct parts. First, there is the question whether ulcer of the stomach at its commencement is caused by arrest of circulation in the corresponding part of the gastric mucous membrane, and begins in a hæmorrhagic erosion. This must be denied, on the ground that if such were its origin it ought to be frequent in those who suffer from obstruction of the portal circulation, as a result of disease of the heart or liver. Hæmorrhagic erosions are in fact common in cases of this kind, but not gastric ulcer; so that the commencement of the latter affection remains unexplained.

But a second question is whether the corrosive action of the gastric juice has anything to do with the further development of the affection. That it is not concerned in its commencement is clear, not only for the reason given by Virchow, but also because in its early stage it is exactly like ulcers which may occur in other parts of the alimentary canal. But a *chronic* ulcer of the stomach presents characters which are seen nowhere else, with the single exception of the first part of the duodenum; and this is exposed to the influence of the gastric juice. The only affection at all like it is a thick-walled chronic ulcer on the leg. It is quite conceivable that the frequent contact of an acid secretion with the surface of an ulcer of the stomach may not only retard its cicatrisation, but also set up a process of chronic inflammation in its edge and floor that may give it its special characters. A further argument in favour of this view may be found in the fact that truncated blood-vessels are often exposed in the floor of a gastric ulcer, whereas in all other parts of the body the walls of arteries show a remarkable power of resisting the ulcerative process.

Another point requiring explanation is that the great majority of ulcers occupy the lesser curvature of the stomach, or its close neighbourhood; probably, as Sir William Gull suggested, it in some way depends upon the fact that this part is more fixed than the rest of the organ.

Sex.—All writers are agreed that gastric ulcer occurs much more often in females than in males; according to Brinton and Wilson Fox, two or even three times as often. In von Ziemssen's *post-mortem* cases the proportion was 35 to 15.

But of 101 fatal cases extracted from the *post-mortem* records of Guy's Hospital for the second edition of this work (1888), there were 59 in men and only 42 in women.

Of 100 other cases which were not fatal (viz. 93 collected from our clinical records, and 7 others added from notes of private cases to make up the 100), there were 33 in men and 67 in women.

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Age.—It is commonly believed that gastric ulcer is a disease of young women, and rare in either sex before puberty or after forty. This, however, was not the opinion of the late Dr Brinton, who maintained that it occurred at all ages from sixteen to sixty and upwards. The following table, drawn up by Mr Keiffenheim from the clinical and the *post-mortem* records of Guy's Hospital, may be of service in determining this point. Ninety-three of the cases are those of patients who left the hospital more or less benefited; the other 101 are those of patients who died, and in whom the diagnosis of gastric ulcer was verified by an autopsy.

	Clinical Cases.						*Fatal Cases.*					
Age.	Male.		Female.		Totals.		Male.		Female.		Totals.
Under 9	0	+	0	=	0		2	+	2	=	4
10 to 19	2	+	9	=	11		3	+	4	=	7
20 „ 29	6	+	24	=	30		7	+	5	=	12
30 „ 39	9	+	14	=	23		7	+	8	=	15
40 „ 49	12	+	10	=	22		15	+	7	=	22
50 „ 59	4	+	3	=	7		15	+	15	=	30
60 „ 66					0		9	+	0	=	9
70 and 72					0		1	+	1	=	2
	33	+	60	=	93		59	+	42	=	101

The striking difference in the two sets of cases seems to show that the prevalent belief that gastric ulcer is a disease of young women must have determined the diagnosis during life. Of the writer's own cases the slight majority have been in men. He has seen it in a lady of seventy-two, verified by an autopsy; and in a gentleman of seventy-eight, who recovered after suffering from hæmatemesis and other symptoms, referable only to gastric ulcer or cancer, and lived for eight years afterwards.

There is one class of cases of gastric ulcer particularly prone to attack girls at or soon after the age of puberty; namely, those cases in which the affection remains latent until fatal perforation occurs. Dr Buzzard has recorded an instance of this kind in a girl nine years old. But it is a great mistake to suppose that the risk of perforation is limited to such cases, and ulcers that have already been recognised by the characteristic symptoms of the disease appear to be equally apt to destroy the patient's life in this way at all ages and in both sexes.

Symptoms.—Of these, the most significant, and generally the earliest, is *pain*. This may be of every possible degree of intensity, from a mere feeling of weight or tightness in the epigastrium, up to the most severe sensations of burning or gnawing or boring, attended with feelings of sickening depression. According to Brinton, it is rarely or never described as stabbing or lancinating. In the great majority of cases it comes on in from two to ten minutes after the ingestion of food, and remains an hour or two, subsiding when digestion is accomplished. If vomiting occurs, this almost always brings relief. Sometimes the pain does not begin until half an hour or an hour after a meal. It is often distinctly increased by the ingestion of hard or indigestible matters, by food which is hot, and (according to Brinton) sometimes particularly by tea and by beer. In very rare cases it comes on chiefly when the stomach is empty; and it is relieved by food, by hot water, or by brandy.

The pain of gastric ulcer is not always intermittent. It may be continuous, lasting for days or weeks together. These are generally cases in which the ulcer is already of long standing.

The seat of the pain is most frequently the epigastrium, immediately

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below the ensiform cartilage; but sometimes it is described as being behind the cartilage, and sometimes it is three or four inches lower down. Occasionally it is outside the median line, in one or other hypochondriac region. It is usually limited to a very small area, which, according to Brinton, is rarely more than two inches in diameter, and is sometimes a mere spot, less than half that size.

Scarcely less important than the pain already described is a pain in the back, to which Cruveilhier first drew attention as a symptom of gastric ulcer. This is of a gnawing character, and is generally referred to a single spot between the eighth or ninth dorsal vertebra and the first or second lumbar.

Dr Brinton devoted great pains to the elucidation of the question whether variations in the seat of the pain in different cases could be traced to differences in the position of the ulcer. He collected some twenty-five cases in which, the pain having been referred to one or other hypochondrium, the ulcer was afterwards found to occupy the corresponding extremity of the stomach. He also ascertained that in some cases the position in which the patient chose to lie, as affording the greatest ease to the pain, was a guide to the seat of the ulcer; the prone position indicating that this was on the posterior, and the supine that it was on the anterior wall of the stomach; whereas, again, when the patient found relief by lying on the right side, the ulcer was at the cardiac end, and when on the left side it was at the pylorus. But he was obliged to admit that in most cases, whatever the seat of the ulcer, the recumbent posture gave ease, and no information could be elicited as to the effects of changes of position.

Pressure on the spot to which the pain is referred almost always aggravates it, and in many cases there is the most extreme tenderness, so that not the slightest contact with the clothes, nor the gentlest touch from the physician's hand, can be endured. Sometimes pressure on the epigastrium increases the pain in the back. A very few instances have been recorded in which pressure has given relief to the epigastric pain.

A second symptom of gastric ulcer is *vomiting*. In the most marked cases this does not occur for some weeks after the patient has begun to suffer pain. It takes place when the paroxysm of pain induced by food has reached a certain height; and as soon as the stomach is emptied the patient is free from discomfort, or at most experiences a slight burning sensation for two or three minutes longer. The expulsion of the gastric contents is seldom attended with violent retching. In rare cases sickness occurs independently of the ingestion of food, but it would appear that this is generally due to some other cause than the ulcer; such, for instance, as alcoholic intemperance.

The vomited matters do not necessarily present any characteristic appearance. But whenever the presence of gastric ulcer is suspected they should always be very carefully examined, and on several different occasions; for proper search may reveal the presence of blood. As above stated, *hæmatemesis* is frequently caused by ulcer of the stomach—it was present in four fifths of Lebert's 252 cases observed at Breslau.

Sometimes the blood is present in such small quantities in the vomited matters that it fails to attract the patient's notice. Specimens of vomit selected for examination should contain as little as possible of food. If this precaution be taken, a fluid which had seemed comparatively clear will often throw down a sediment containing blood-corpuscles.

In other cases the amount of blood effused into the stomach is

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larger. Being altered by the gastric juice, it gives to the vomited matters a brown colour, or (to use the common expression) the appearance of "coffee-grounds." This is due to the presence of acid hæmatin.*

It must not be supposed that traces of blood in the vomited matters, or even the occurrence of obvious hæmatemesis, is a proof that an artery has been exposed in the floor of the ulcer. The blood often comes either from the minute vessels which supply its surface, or from those which go to the adjacent part of the mucous membrane.

Other symptoms of gastric disorder frequently occur when an ulcer is present, which are not characteristic of this disease. Such are dyspepsia, flatulence, pyrosis, and, almost always, constipation. The appetite is generally defective, but in some cases excessive and ravenous, particularly when there is habitual vomiting of all that is taken; sometimes, although there is great desire for food, the patient is afraid to eat on account of the pain which follows every meal.

In young women amenorrhœa attends ulcer of the stomach so frequently that some writers have endeavoured to trace a causal relation between them. The fact was pointed out long ago by the late Dr Crisp. In older women menstruation often goes on regularly, although they may continue for several years to be affected with gastric ulcer; but if there should be anæmia from profuse hæmatemesis the catamenia may be suppressed for a time.

In long-standing cases there is often extreme wasting; by the constant pain and the deprivation of food sharp lines are worn in the patient's face, and cause a peculiar physiognomy. Dr Brinton says that he was often able to recognise the disease at a glance in a crowded out-patient room.

When a gastro-cutaneous fistula has developed itself the patient usually ceases to vomit. Food almost always escapes from the orifice as soon as swallowed; to prevent this the patient has to wear a plug of lint or gutta-percha unless the mucous membrane should happen to protrude, so as to form a kind of natural valve. The general health, however, often improves, so that the patient may go about almost as well as usual.

Diagnosis.—The characteristic symptoms of gastric ulcer, then, are the occurrence, soon after the ingestion of food, of a peculiar kind of pain, which is relieved by vomiting, or subsides when digestion is completed, and the presence of blood in vomited matters or as melæna in the fæces.

When these symptoms are present in a person under forty years of age, the recognition of gastric ulcer is not difficult; but often they are absent or obscured, and in older persons may be due to cancerous ulcer of the stomach or to cirrhosis of the liver. In young women the symptoms of gastric ulcer are often simulated by anæmia from other causes, and by phthisis.

The gastric juice obtained from the stomach in cases of simple ulcer is stated by Ewald, Reigel, and other observers to be constantly, or almost constantly, more acid than in health. The chemical diagnosis which has been proposed on this basis between ulcer and cancer of the stomach will be mentioned under the latter head (p. 199).

When an ulcer is seated in the first part of the *duodenum*, the pain is said to come on between half an hour and two or three hours after meals, or even later still; vomiting is not very common, but (according to Krauss, who has published a monograph on this disease) hæmorrhage has occurred

* It ought perhaps to be noted that the administration, as medicine, of a preparation of iron may give a blackish colour to the contents of the stomach, if the patient should about the same time take tea, or anything else containing tannic acid.

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in one third of all recorded cases. Krauss collected fifty-eight cases of duodenal ulcer in men and only six in women.

Gastric ulcer is not infrequently altogether *latent* until it erodes a large artery; and the same is true of perforation into the serous cavity. It may indeed sometimes be a question whether the ulcer was absolutely unattended with symptoms during any part of its course, even when a patient who had seemed in perfect health is suddenly attacked with an illness which proves fatal in a few hours. In such cases the ulcer often presents no sign of inflammatory reaction in its walls; and there appears to be no reason why all the coats of the stomach should not be destroyed in a few days, or even in a few hours. Niemeyer relates the case of a young medical man who died rapidly of perforation, and who declared positively that for just a week previously there had been some trifling symptoms which had seemed to him to indicate a slight catarrhal affection of the stomach; and this writer appears to conclude that the time during which the ulcer had been forming must also have been a week. But this conclusion is very doubtful, for often when perforation occurs in persons apparently in good health, the ulcer has smooth rounded edges, and must certainly have existed for a considerable time; and it not infrequently happens that this affection is found accidentally in the body of a person who has had no gastric symptoms, and has died of some other disease.

But in other instances, ulcer of the stomach is overlooked, not because the patient has shown no signs of gastric disorder, for such signs may have been observed for several years, but because they have been of so slight a character that the case has been regarded as one of mere dyspepsia. The absence of vomiting is particularly apt to lead to this mistake. Brinton mentions one case in which there was no vomiting for four years during which an ulcer remained active; and he speaks of other cases in which it was represented by slight nausea only, or was limited to a single attack, or occurred only at the very close of the disease. No absolute rule can be laid down for the diagnosis of gastric ulcer in cases of this kind. Much depends upon the acumen and judgment of the physician.

Gastric ulcer has been mistaken for chronic poisoning. The symptoms of the fatal illness of the Duchess of Orleans in 1670 were commonly attributed to poison, until Littré in a masterly clinical and historical commentary on the case showed conclusively that she died from perforation of the stomach by an ulcer.

Events.—A gastric ulcer may have several different terminations. Cicatrisation sometimes occurs, and the patient regains his former state of health. Several years ago the state of the stomach was noted with great care in a large number of autopsies at Prague, and both cicatrices and unhealed ulcers were found very frequently, in the proportion of 147 of the former to 156 of the latter. Scars in the stomach have sometimes been discovered in the *post-mortem* theatre at Guy's Hospital, but far more rarely.

Few would endorse Cruveilhier's statement that "simple ulcer of the stomach tends essentially to a cure;" but many cases are on record of symptoms followed by spontaneous cicatrisation. One is that of the anatomist Béclard, who suffered from pain in the stomach and vomiting, from which he gradually recovered; when he died, many years afterwards, the scar of an ulcer was found in the lesser curvature of the stomach.

The healing of a gastric ulcer is not infrequently partial. Cicatrisation

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may take place on one side, while in the opposite direction it goes on spreading. The pylorus may become narrowed, or the stomach may acquire an "hour-glass contraction," while part of the surface still remains unhealed. Thus symptoms of obstruction may arise, in addition to those which indicate the presence of the ulcer. Or the sore may heal for a time and afterwards again break out. Probably this is one cause of the remarkable fact that there may be a complete intermission of all the symptoms of gastric ulcer for many months, after which they return. In other cases, however, an apparent intermission is really due to the fact that, after the first ulcer has finally healed, a fresh one develops. Lastly, it appears probable that the inclusion of nervous filaments in a cicatrix is sometimes the cause of the continuance of pain after the subsidence of the other symptoms, and, indeed, after the cure of the disease.

The *duration* of this disease is often exceedingly protracted. Cases have been recorded in which symptoms were present uninterruptedly for twenty or thirty years, or even longer.

The proportion of cases of gastric ulcer which recover under suitable treatment is certainly large. In 100 cases in our wards only fourteen deaths occurred while in the hospital.

There are several different ways in which gastric ulcer may prove fatal. Sometimes the patient dies by gradual exhaustion. This, however, seems to be rare; it is recorded only three times at Guy's Hospital out of thirty-four cases in which the immediate cause of death is noted. Brinton speaks of having seen three or four cases of this kind within a few months.

In eleven of our thirty-four cases death was traceable directly to hæmorrhage, and in seventeen to perforation. Other results of gastric ulcer are much more rare. In some cases the destruction of the gastric coats is followed, not by general peritonitis, but by a circumscribed abscess, which generally occupies the left hypochondrium, and which may in its turn perforate the diaphragm and set up fatal pleurisy. In others, after perforation, an abscess is formed in the posterior mediastinum. Still more rarely, the ulcer sets up pylephlebitis and abscesses in the liver.

It is an interesting question whether cancer ever develops itself secondarily in the floor or edge of a simple ulcer of the stomach. Trousseau speaks of the two diseases as antagonistic, but Brinton is disposed to think that the one may pass into the other. We have had at Guy's Hospital more than one case in which the stomach presented part of the circumference of what was apparently a simple ulcer, of which the remainder had been replaced by a malignant growth. If this view is correct, the occurrence is probably more frequent than might appear from the absence of direct observations of it; for in many cases the extensive development of cancer would doubtless obliterate all traces of the previous ulcer. Dr Shaw reports that, of fifty autopsies, in six there was found a cicatrix surrounded by or near the cancer which was believed to be of older date than the latter, but on comparing the clinical histories of these cases no confirmation of the previous existence of a simple ulcer could be found.

Treatment.—There are some cases of ulcer of the stomach in which all that can possibly be hoped for is the palliation and relief of its symptoms. The medicines that are then to be prescribed are those which have already been mentioned under the heads of gastric pain, of vomiting, and of hæmatemesis respectively. Brinton found in certain cases a preparation of bismuth, or the kino powder with opium, as effectual in arresting hæmorrhage from

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the stomach as the more powerful astringents. He expressed a very strong opinion as to the absolute uselessness of the oxide or nitrate of silver in this disease; but although it is no doubt impossible that these medicines can act upon the surface of the ulcer as lunar caustic does upon a sore to which it is directly applied, it is certain that they sometimes give relief to the pain. Brinton wrote highly of the value of opium, attributing to it, beyond its influence in relieving pain and sickness, a direct power of buoying up the nervous system and supporting the patient's strength. In fact, he believed it to be as essential to the cure of an ulcer of the stomach as some surgeons have found it to be in cases of chronic ulcer of the leg. His way of using it was to give a small pill of the watery extract, or a few grains of the compound soap pill, two or three times a day. He prescribed it especially for patients of advanced age, or of broken-down constitutions, in whom the disease was of long standing and the ulcer probably of large size.

The use of purgatives requires much caution in cases of ulcer of the stomach, and there is no doubt that they should as far as possible be avoided. But Brinton more than once noticed a definite and repeated coincidence between the occurrence of a paroxysm of pain and vomiting and an accumulation of fæces in the colon; and for such cases he recommends the use of castor-oil. In common with other writers, he speaks very strongly against the use of mercury in any form, saying that he is certain that he has witnessed relapses which could only be attributed to its administration.

A blister often relieves the symptoms of gastric ulcer, but it is said that in some patients it causes increased pain; Brinton speaks of having observed this in cases in which there were old adhesions between the stomach and the abdominal walls. This writer thought that leeches should never be used, but Dr Wilson Fox says that the application of two or three of them sometimes gives marked relief to pain. A blister applied to the back has been found to relieve the pain in that region.

In most cases, however, something more must be aimed at than the mere relief of symptoms. If there is any truth in the hypothesis that cicatrisation of gastric ulcers is prevented by the action of the gastric juice, the rational treatment is evidently to keep the stomach quite empty for a time, supporting the patient by enemata or nutrient suppositories. In cases of obstruction of the œsophagus life may be maintained in this way for at least two or three weeks, a period which is probably long enough to enable a gastric ulcer to take on a healing action, even if it is not sufficient for its complete cicatrisation. Whenever the diagnosis is clear, and the patient can be induced to submit to this method of treatment, it would be reasonable to carry it out for a certain period at once. However slight the symptoms may be, one never can tell how near the peritoneum or some large artery may be to the floor of the ulcer; and every week's or month's delay must needs add something to its size, and so far diminish the prospect of benefit if the patient should afterwards submit to this plan of treatment.

Moreover, in many cases, the very urgency of the symptoms affords an argument for giving complete rest to the stomach, and feeding the patient by nutritive injections: for instance, when all that is swallowed is shortly afterwards rejected. Vomiting, as Brinton remarks, is much less amenable to treatment than any other symptom of gastric ulcer, and often resists the action of every reputed remedy. It is also by far the most dangerous and important of these symptoms, on account of the risk that it entails of the supervention of perforation from rupture of the protective

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adhesions. One must therefore forbid any food to be swallowed while there is obstinate vomiting.

In the well-known case of Dr William Hunter, before quoted (p. 173), vomiting that had been uncontrollable was checked by the limitation of the food to milk, given a spoonful at a time. A similar plan was suggested by Cruveilhier, for cases of gastric ulcer. Brinton says that sometimes the milk is better borne when it has previously been boiled, or when it is mixed with lime-water; in some cases he has found a little fresh curd, mixed with a thin pulp of arrowroot boiled in water, better than anything else. As convalescence advances, ground rice may be substituted for the arrowroot, and afterwards biscuit powder. Sugar was specially objected to by Cruveilhier, and subsequent writers have endorsed his opinion; it is thought to produce flatulence.

Some persons seem unable to digest milk, and according to Dr Wilson Fox there are some elderly people whom it fails to nourish. Animal broths must then be given in its place, or peptonised milk may be tried. But many patients suppose themselves to have digestive idiosyncrasies, who are afterwards found to do just as well as anyone else upon the most rigidly restricted diet.

During convalescence from ulcer of stomach, the most extreme care should be exercised as the patient gradually extends his range of diet. The quantity of food taken at one time must be such as will not distend the stomach; and all hot food or drink must be avoided. Complete abstinence from alcoholic stimulants is probably desirable.

Lastly, it must not be forgotten that pressure upon the epigastrium may do harm when there is a gastric ulcer. A woman should not be allowed to wear her stays, nor a shoemaker to press the last into the pit of his stomach. Care must be taken, in manipulating the abdomen, to use only very gentle pressure. For similar reasons all violent exercise and sudden efforts must be most carefully avoided.

CANCER OF THE STOMACH.—This is a most important disease, not only from its fatality, but also from its frequency. Of all forms of cancer in men, that of the stomach is probably the most common, more so than cancer of the rectum; and of all forms of cancer in women, that of the stomach is probably also the most common, more so than cancer of the uterus or of the breast. Brinton estimated that it caused 1 per cent. of the total mortality in London hospital practice; and at Guy's Hospital the proportion is even higher, namely, 79 in 5990 autopsies, or more than 1·3 per cent. According to Brinton it is less common than simple ulcer; but the records at Guy's Hospital during twenty years show that there were rather more than twice as many fatal cases of cancer as of ulcer of the stomach.

Anatomy.—It has been stated that there are three local varieties of this disease, as it affects the cardiac orifice, the body of the stomach, and the pylorus.

As regards the first of these, most of the cases recorded as examples of cancer affecting the cardia have really been instances of cancer of the end of the œsophagus, extending into the adjacent part of the stomach. The 'Pathological Transactions' seem not to contain a single example of cancer beginning in the stomach at its œsophageal end; nor does the museum of Guy's Hospital show any specimen in which the lower end of the œsophagus is not also affected. Moreover, in every one of the four or

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five cases that have at different times been recorded under the name of cancer of the cardia, in the reports of our *post-mortem* examinations during twenty years, there is room for supposing that the disease began either in the œsophagus or in the lesser curvature of the stomach. Apart from these facts, we might expect that a part at which the digestive tube is opening out into a large cavity would have little tendency to be affected with cancer, in comparison with the narrow passages above and below it.

Cancer of the body of the stomach, however, and cancer of the pylorus must be carefully distinguished from one another. The latter is the more uniform and more frequent affection of the two. Of fifty cases at Guy's Hospital, collected by Dr Shaw (1889), thirty-six occupied the pylorus, three the cardia, and eleven the body of the stomach, both orifices being free.

The *pylorus*, when affected with cancer, becomes greatly thickened, so that it forms a rounded swelling, often somewhat lobulated, which is almost always sharply defined towards the duodenum, while it passes gradually into the wall of the stomach, or extends for some distance along its lesser curvature. The disease generally involves the whole circumference of the orifice, which is consequently much narrowed. It grasps the finger tightly, or may be too narrow to admit it; but cases are very rare in which a large catheter cannot be passed into the duodenum. The mucous surface is generally much reddened; it may either be smooth or present nodular excrescences, and sometimes distinct villous growths. Most frequently it is more or less extensively ulcerated.

On making a longitudinal section, one finds that the several coats of the stomach are still plainly to be recognised. The thickest part of the mass is that which corresponds with the submucous connective tissue; this generally makes up two thirds of the whole. Next comes the muscular layer, which is likewise much thickened. This appears as a row of pinkish-grey translucent striæ, regularly arranged with opaque bands between them, which bands often consist of cancerous tissue. Still further outwards the subserous tissue is also thickened, but to a less extent; and it likewise is generally infiltrated with the new growth. The peritoneal surface may either be unaffected, or it may be the seat of inflammatory adhesions to the adjacent parts, or it may present more or less numerous cancerous nodules.

Cancer of the *body of the stomach* is much more variable in its characters. In many cases it begins along the lesser curvature, and then it may either remain limited to that part, or spread to one or both surfaces. In one case of this kind only a narrow border along the greater curvature was left untouched by the invading growth. In some instances it forms a more or less broad ring completely surrounding the middle of the organ. Often it appears as a large patch, with or without ulceration.

Whatever part of the stomach may be affected with cancer is very liable to become adherent to any other structure with which it happens to be in contact. Most commonly this is the under surface of the liver, and the growth may then extend into the glandular tissue, and undergo ulceration; thus a large cavity may be produced, in which food (such as grape skins) may lodge. Or the diseased portion of the stomach may become fixed to the surface of the abdomen. In one instance the anterior part of the abdominal wall had altogether disappeared, being fused in a mass of cancer two or three inches thick. A gastro-cutaneous fistula may result, but this is even more rare than with non-malignant gastric ulcer. Or, again, the growth may become continuous with a mass of diseased glands

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near the pancreas, and with that structure itself. Or, perhaps more frequently, the first portion of the duodenum is drawn into adhesion with the back of the diseased pylorus; and sometimes an ulcerated opening forms between them, behind the proper orifice. This depends upon the fact, pointed out by Luschka, that the normal direction of the first part of the duodenum is from before backwards. In other cases the diseased part of the stomach becomes adherent to the colon, and a fistulous communication between them may afterwards develop itself.

Histology.—Cancer or malignant disease of the stomach is with few exceptions true *carcinoma.* In ten years at Guy's Hospital (1880—1889) there were reported from the deadhouse forty-five cases of carcinoma, one of colloid, and three of sarcoma of the stomach.

Virchow mentions only two instances of primary *sarcoma* of the stomach, one of them being a case recorded by Dr Wilks in the tenth volume of the 'Pathological Transactions.' In each of these cases, and also in a third case of Dr Wickham Legg's (in the twenty-third volume of the same 'Transactions'), the patient was a young girl; and both the ovaries were at the same time affected with sarcoma. The disease seemed to have begun in the lesser curvature. Virchow remarks on the comparatively slight tendency to ulceration, as a distinction between this form of disease and carcinoma of the stomach. But in a fourth case of Dr Cayley's, recorded in the twentieth volume of the 'Pathological Transactions,' there were large nodules of the growth projecting into the cavity of the stomach, and these were extensively ulcerated. The patient was a man aged fifty-seven.

Virchow does not mention any instance in which disease occupying the pylorus was of a sarcomatous nature; but three or four cases of this kind have been observed at Guy's Hospital. One was a woman aged forty-seven; the submucous tissue of the pylorus was three quarters of an inch thick, and Dr Moxon described it as thick but flabby, of a milk-white colour, yielding a clear fluid when scraped, consisting mainly of a well-developed fibrous tissue, but also containing some delicate spindle-cells with very large tails. In another case, which occurred in a man aged sixty-six, the pylorus was the seat of a new growth of yellowish look and of firm consistence, which proved to be a round-celled sarcoma. A third case came under the author's observation in June, 1876. The patient was a man aged sixty-seven. The pylorus, through which the finger could readily be passed, presented a large ulcer seven inches in circumference, the base and sides of which were formed by a homogeneous pinkish-white substance, which yielded no juice, and consisted of round and oval cells and spindle-cells, embedded in an intercellular substance containing mucin. Perhaps the cases which Sir Thomas Watson and others have described under the name of simple hypertrophy of the pylorus were examples of sarcoma.

Dr Wilks used formerly to lay stress upon the fact that in some cases of so-called scirrhous pylorus the disease was really only a local thickening of the submucous tissue, with consequent hypertrophy of the muscular coat. He based this opinion partly upon the dry, juiceless character of the growth, consisting mainly of fibrous tissue, partly upon the absence of secondary cancerous nodules. This last fact, however, is not by itself conclusive; for, out of forty-one cases of true carcinoma of the pylorus in succession at Guy's Hospital, there were at least five in which no cancer existed elsewhere in the body. On the other hand, in one case, which would otherwise have been designated simple fibrous thickening or hyper-

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trophy, there were secondary nodules in the liver, the exact nature of which, however, is not specified in the report. One must bear in mind that carcinoma of the stomach (unlike carcinoma of the breast) is situated in a vital organ. Secondary growths would doubtless be found much more constantly if death did not so quickly ensue.

As above stated, in cancer of the stomach the submucous tissue is more thickened than any of the other layers. The older pathologists therefore supposed that the disease began there, but this appears to be a mistake. As Waldeyer first showed, it always begins in an overgrowth of the glands in the mucous membrane. These become elongated and dip down into the subjacent connective tissue; when they have reached it they proliferate actively, and so give rise to a cancerous nodule, which spreads out horizontally and may reach a large size, but which is nowhere connected with the superficial glandular layer, except at its starting-point.

Carcinoma of the stomach, as of other parts, presents in different cases histological characters which differ widely. These in part depend upon the proportion between the amount of fibrous stroma forming the alveoli, and that of the nests of cells contained in them. If the stroma be abundant and the alveoli small, the growth has a tough fibrous appearance, and yields but very little juice. If the stroma be scanty and the alveoli large, the growth is soft and of a milk-white colour, and yields much juice when scraped. But between the former ("scirrhous cancer") and the latter ("encephaloid or medullary") all gradations exist, and in some cases it may be difficult to say under which head the disease should be placed. As a rule, however, carcinoma of the stomach belongs rather to *scirrhus*. According to Brinton, three cases out of four belong to this form of the disease, and among cases affecting the pylorus the proportion would be still higher. As already stated, the tumour grows towards the serous surface between the bundles of the hypertrophied muscular coat. On the other hand, as Moxon pointed out, *medullary carcinoma* often destroys the muscular coat over a considerable area; so that the whole thickness of the wall is converted into a uniform mass of disease presenting elevations on each surface, but especially towards the cavity of the stomach. Sometimes branching processes sprout from the mucous membrane, which form beautiful microscopical objects, being each made up of a central blood-vessel, clothed with thick layers of well-formed cells. These constitute what has been termed "villous cancer," which, however, has no claim to be regarded as a separate variety. Moxon has described a case of this kind, in which the floor of the growth was formed by a large mass of soft carcinoma, growing directly into the substance of the liver. It is doubtful whether a simple papilloma occurs in the stomach, analogous to the well-known villous growth which affects the urinary bladder.

In a few cases a cancerous growth in the stomach presents the characters of a *cylinder-epithelioma*. In sixteen cases microscopically examined at Guy's Hospital, there were five of this columnar-celled carcinoma.

The surface of a carcinomatous growth in the stomach is generally ulcerated. Sometimes there are only a few superficial erosions, but very often a deep sore is formed with hard, raised, ragged edges and a sloughing base. It is possible that the digestive action of the gastric juice may be concerned in bringing about the detachment of large masses of the cancer tissue, which sometimes leads to the erosion of blood-vessels of considerable size. In such cases the ulcer is horribly foul and offensive.

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Cancerous growths in the stomach, as in other parts, are liable to undergo *fatty degeneration.* Sometimes scarcely a trace of active growth is discernible; it might be said that the primary affection had undergone spontaneous cure; but the patient has, nevertheless, died of an extension of the disease to other parts.

Another kind of new growth to which the stomach is particularly subject is that known as *colloid cancer.** Sometimes the whole thickness of the organ is infiltrated with a jelly-like material, there being nothing to suggest the presence of ordinary carcinoma. But, more frequently, while some parts of the growth have the character of colloid, others have those of scirrhus or of a form intermediate between it and soft cancer; and under the microscope it is not uncommon to find that more or less colloid change is present in cases in which it may not be observed by the naked eye. It is in such cases that the real nature of the affection is most apparent. For it is found that the alveoli are no longer filled with the characteristic epithelioid cells, but that towards their periphery they contain a greater or less quantity of a structureless translucent substance. As this increases, the cells become less and less marked and finally disappear. It is said by Rindfleisch that they individually swell out into colloid globes which ultimately blend with the rest of the structureless material that now distends the greatly enlarged alveoli; but he also thinks it probable that a part of this is the result of a chemical change in an albuminous substance secreted from the blood. As the alveoli increase in size, they become spherical and the septa between them break down; and thus large translucent globes are formed which under the microscope appear almost structureless. Colloid cancer is less apt to infect distant parts than other carcinomata; but it often spreads over the peritoneal surface, and produces masses of enormous size. It is a remarkable fact that secondary nodules of colloid cancer reproduce, not the histological structure of the primary growth, but its degenerative features. Thus the lungs may be found studded with translucent gelatinous nodules of typical colloid character.

Anatomical results.—Certain consecutive changes in the form and situation of the stomach arise when the pylorus is the seat of cancer. The obstruction thus produced at the outlet of the cavity causes enormous dilatation, so that the stomach may become large enough to hold six or seven pints, and may fill the whole abdomen, while its greater curvature sweeps round just above the pubes. Its walls may at the same time be greatly thickened by diffused hypertrophy of the muscular coat, but sometimes they are exceedingly thin. In some cases, however, cancerous disease of the pylorus fails to obstruct the outflow of the gastric contents; or vomiting is so frequent that no accumulation in the stomach takes place; or the patient's appetite is so bad that scarcely any food is swallowed; while in other cases the extension of the morbid growth along the lesser curvature and into the surfaces of the organ tethers it and prevents its dilatation.

When the lesser curvature is the seat of the disease, the cardiac and pyloric orifices may be approximated by the contraction of the growth;

* For a long time pathologists supposed that there was a special form of cancer characterised by its gelatinous appearance; this they termed colloid cancer. But such a view was attended with great difficulties; and not the least satisfactory result of the microscopical researches of late years has been the solution of the doubts which prevailed as to the remarkable affection in question by showing it to be a form of cancerous degeneration.—C. H. F.

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and the anterior and posterior walls of the stomach may be so flattened against one another that scarcely any cavity is left.

Ætiology.—Of the causes of cancer of the stomach very little is known. It occurs chiefly in persons over forty years of age. Of forty-six cases at Guy's Hospital eleven patients only were under the age of 40; of the remaining thirty-five, there were sixteen between 41 and 50, eleven between 51 and 60, and eight between 61 and 70. In forty-seven additional cases the youngest patient was 31, the oldest 77, and the average age was somewhat over 50—numbers which correspond generally with those given by Brinton.

Among ninety of our cases in which the sex was noted, sixty-three occurred in men, and twenty-seven in women. This also accords with Brinton's estimate; but other writers have said that the disease is more common in women than in men.

Hereditary predisposition is said to be well marked in some cases, and the case of the Napoleon family is cited in proof of the fact. In forty-six patients of Guy's Hospital whose family history was recorded with sufficient detail, Dr Shaw found that cancer had been present in one of the sisters twice, and in one of the parents of the patient five times: in four of these seven cases the cancer was gastric in locality.

Taking the hereditary cases only, one in nine, if not an accidentally large proportion, is certainly more than mere chance would produce.

In some cases cancer of the stomach has followed a blow upon the epigastrium. Andral related an instance in which it occurred in a patient who had taken nitric acid, and Dittrich one in which arsenic had been swallowed. Some writers admit the influence of depressing emotions in producing cancer of the stomach as of other organs.

Symptoms.—These differ so widely in different cases that it is difficult to describe them generally. At first they are indefinite; the patient, without apparent cause, begins to complain of discomfort in the epigastrium after his meals, he is troubled with acid eructations, and attributes these symptoms to dyspepsia. His tongue remains clean, yet he has no appetite, and finds that he is losing flesh. The uneasiness at the pit of the stomach passes into pain; this may be a dull aching referred to the epigastrium or to the back; or it may be exceedingly severe and of a burning or lancinating character. It is generally more or less increased by meals, but it is by no means limited to the periods at which the stomach contains food. Vomiting is the next symptom. When the seat of the disease is the middle of the stomach, vomiting may come on soon after meals; when it is the pylorus, the food is usually retained for three or four hours, *i. e.* until it should be passed on into the duodenum. The matters rejected consist at first of partially digested food or mucus, but soon these are streaked with altered blood, which is of a brown or black colour; or they may contain sufficient blood to resemble coffee-grounds. Constipation and flatulence are also complained of. The aspect of the patient is altered; he acquires a pale, sallow, or earthy complexion, and becomes depressed, irritable, and morose.

In some instances most of these symptoms are absent. Cancer of the lower end of the œsophagus extending into the adjacent part of the stomach is often latent, and most of the cases in which gastric carcinoma has run its course without producing any marked symptoms have been instances of this kind, generally known as cancer of the cardia. But Sir

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Thomas Watson relates a similar case, in which the disease occupied the greater curvature. A gentleman, between forty and fifty years of age, was on his way home from Scotland (where he had been deerstalking and shooting grouse) when he was seized one night in a London hotel with a deadly faintness, rapid breathing, and severe pain referred to the sternum. He had before been gradually losing flesh and strength, but the only definite symptoms of which he had complained were sour eructations, loss of appetite, and repugnance to solid food. Sir Thomas Watson could detect no physical sign of disease. The next night the patient had a similar paroxysm and died. The greater curvature of the stomach presented throughout its whole extent a mass of scirrhus, while the cardiac and the pyloric orifices were free. In a patient under the writer's care in Guy's Hospital with colloid cancer of the stomach, there was scarcely any vomiting, and no complaint of local pain during the three months before his death. It was supposed during life to be cancer of the peritoneum, and this was the case, but it was secondary.

Physical signs.—Examination of the abdomen, when cancer of the stomach is suspected, should never be omitted. The patient must lie down in an easy posture, with the shoulders low and the knees bent, and he should breathe deeply. After the abdomen is exposed, its shape must be observed, and particularly whether there is any fulness of the epigastrium or the reverse. Sometimes a tumour may be seen through the parietes, but most commonly it is to be detected only by manipulation. In some cases it can be felt as soon as the hand is laid upon the surface, but more frequently much care is required. The abdominal muscles are often very rigid, particularly those parts of the recti which overlie a deep-seated swelling, and careless handling may throw them into contraction, so that nothing can be felt. If the patient's attention can be concentrated on his breathing, or can be diverted by conversation, his abdominal muscles will often relax. The palm of the physician's hand (which must not be cold) is to be laid gently upon the abdomen, and allowed to rise and fall as the patient breathes; gradually slight pressure is made, which may be increased until the abdomen has been thoroughly explored. During all this time the palm of the hand as well as the fingers should be kept evenly applied to the surface; and all sudden movements of the fingers' ends which might excite contraction in the muscles of the abdomen must be avoided. This was Sir William Gull's method. There is sometimes extreme tenderness when the stomach is affected with cancer; but even then it is generally possible to make out the form and relations of any tumour that may be present, if only due pains be taken.

The position and form of the *tumour* produced by cancer of the stomach vary greatly; they are determined by the seat of the growth. If it occupy the middle of the organ, the epigastric region, a little to the left, will contain any mass that can be felt on manipulation. In one case two nodular ridges could be clearly made out, corresponding one with each curvature; while between them, and further back, lay an irregular mass, which seemed to occupy the posterior wall. In other cases a more or less rounded prominent mass is felt, which is the thickened anterior surface of the stomach. Dr Cayley has related a case in the 'Pathological Transactions' in which the left hypochondrium contained a firm slightly moveable tumour which reached below the umbilicus, and was supposed to be the spleen, but it proved to be the stomach indurated by carcinoma.

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When the pylorus is the seat of the cancer, the tumour is usually much more definite. In some cases its character can be made out almost as plainly during life as in the deadhouse. It forms a rounded mass, often somewhat lobulated, perfectly circumscribed on all sides except towards the left, where it can sometimes be felt to pass gradually into the wall of the stomach; it may vary in size from that of a walnut to that of a Tangerine orange. Its seat is usually a little above and to the right of the umbilicus; considerably lower than the position of the normal pylorus, which lies so completely under cover of the liver as to be inaccessible to palpation. The fact that a cancerous pylorus is sometimes felt in the umbilical region seems to have been recognised by Brinton only in the case of female patients, and he attributed it to the alteration in the position of the viscera caused by the use of stays; but this explanation is insufficient, since the same thing is frequently observed in men, and in men who do not wear a tight belt. It probably results from the traction exerted upon the lesser omentum by the weight of the tumour; for when that fold of peritoneum is thickened and involved in the growth, or when the pylorus is retained in its normal position by adhesions, no tumour can be discovered. On the other hand, it sometimes descends much lower. Wilson Fox says that it may be found in the right iliac fossa, or even in the pelvis, adhering to the intestine, uterus, ovary, or bladder. In one case a tumour in the left hypochondrium, of which the exact situation varied at different times, according as the stomach was more or less distended, proved to be the pylorus, which had been dragged over to the left side, and was firmly adherent to the parietes and to the edge of the liver. A scirrhous pylorus usually seems to move slightly downwards when the patient draws a deep breath, being pushed down by the liver. Some observers have thought that the movement is rather apparent than real, depending on the expansion of the ribs carrying the abdominal walls upwards over the tumour.

The tumour caused by cancer of the pylorus often receives an impulse from the abdominal aorta, which may simulate a cœliac aneurysm.

It is said sometimes to disappear entirely for days together as the result either of twisting of the stomach on its axis, or of the tumour itself being overridden by a distended colon.

To percussion it should yield a dull note, but when it is of small size this is masked by the resonance of the adjacent intestine.

The results of manipulation of the abdomen in cases of cancer of the pylorus are by no means limited to the discovery of a tumour; one has also to ascertain, if possible, the position and size of the stomach itself. This organ is often greatly dilated, and descends much lower in the abdomen than under normal conditions; the greater curvature may be below the umbilicus, and may even reach to the pubes. The epigastric and left hypochondriac regions are then deeply hollowed, while the lower part of the abdomen is protuberant. This, indeed, is not in itself proof of dilatation of the stomach, for it is common enough in persons whose small intestines are distended with flatus, if their abdominal walls are also loose and flaccid. What is conclusive is the detection of the peristaltic movements of its thickened walls. If the surface of the abdomen be attentively watched, a wave of contraction may often be seen to start from the left hypochondrium, descend below the umbilicus, and pass on to the right side, and then a little upwards towards the cartilages of the right ribs. Or a rounded protuberance, as large as an orange, may rise up on the left side

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and travel round to the right, in the same way as the wave. In one or two cases we have observed distinct antiperistaltic movements (from right to left) in a hypertrophied stomach. Another indication of enlargement of the stomach is the production of a splashing sound by manipulation of the lower part of the abdomen; but this is not a sign of much value by itself, for similar sounds may be produced from the presence of gas and fluid together in coils of intestine.

If there should be occasion to pass an œsophageal tube down into the stomach the end of it may sometimes be felt through the abdominal walls. According to Leube it may, in health, reach as low as the umbilicus; but if it descends below that level the stomach must be dilated (cf. p. 199).

It will be observed that the physical signs of this form of chronic dilatation of the stomach differ in some respects from those of the acute paralytic distension of the organ described above (p. 157).

The presence of dilatation of the stomach modifies some of the other symptoms. In ordinary cases of cancer of the pylorus the patient vomits about three or four hours after each meal, at the time when digestion is completed, and when the food ought to be passing on into the duodenum. But when the cavity of the stomach is enlarged three or four meals may be retained in succession; and the patient, when he does vomit, may bring up surprising quantities of fluid, several pints at a time. In one instance vomiting never occurred except at night; and sometimes the stomach rejects its contents only at intervals of some days.

The vomited matter is peculiar. It generally consists of a thin, highly acid fluid, of a dirty grey, brownish, or greenish colour, which, on standing, becomes covered with a thick, frothy, yeast-like scum, while it deposits at the bottom of the vessel a more or less abundant sediment. In the scum, as well as in the fluid, oval spores and beaded threads of the yeast plant (*Torula cerevisiæ*) are often found in large quantity; and also in enormous numbers certain rectangular bodies, which belong to another microscopic fungus (cf. vol. i, p. 14). They are divided by cross-lines into smaller rectangles, some of them into four, others into sixteen, and some into sixty-four, according to their size. They thus resemble packages tied across again and again by cords; and Goodsir, who in 1842 was the first to observe them, gave them the appropriate name of *Sarcinæ ventriculi* (*sarcina* = woolpack). The late Dr Chambers pointed out that they may be found in the dead body in stringy mucus adherent to the interior of the stomach, and therefore he regards this as their proper seat, and not the liquid which the organ may happen to contain.

Another circumstance that may considerably modify the symptoms of cancer of the stomach is the formation of a gastro-colic fistula. The best account of this affection is that which Dr Murchison gave in the 'Edinburgh Medical Journal' for 1857. In almost all such cases there is stercoraceous vomiting; it has been pointed out by Dr Gairdner that the only exceptions to this rule are cases in which there is at the same time narrowing of the pylorus, so that the stomach is constantly kept overloaded with its proper contents. The patient's breath often has a foul odour, or he has eructations of intolerable fœtor, or a horrible taste in his mouth. The fistula sometimes allows matters to pass the other way, from the stomach into the colon; but this appears to be much less frequent, for there are only seven out of the twenty-three cases collected by Dr Murchison in which undigested matters were recognised in the fæces. This con-

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stitutes what is termed *lientery*.* It must not be supposed to be of itself a proof that a fistula exists. The attempt has been made to increase its significance by giving food coloured with cochineal to patients who pass undigested matters from the bowels, and by observing what length of time elapses before the colouring matter appears in the evacuations. In a case of Schönlein's this occurred only at the end of twelve hours, in the last of seven evacuations that took place during that period. He inferred that the case was not one of gastro-colic fistula; and (with less reason) that the lientery was due to widening of the pylorus. In patients who have an opening between the stomach and colon, the appetite is generally very bad, but in one instance there was craving for food. Pain is not invariably present. Indeed, the formation of the fistula sometimes leads to the relief of pain that had before existed.

Sometimes a gastric cancer opens into the third part of the duodenum. In a case where this occurred at Guy's Hospital it gave rise to no symptoms.

Returning to the symptoms of cancer of the stomach in general, it is a remarkable fact that towards the fatal termination of the disease they often subside. In one case vomiting did not occur once during the last month of the patient's illness.

In the latter stages of gastric carcinoma, either with or without the presence of a tumour, the liver is often found to be enlarged and painful. Jaundice may appear from secondary hepatic carcinoma, or from pressure on the common bile-duct; ascites, from extension of the cancerous growth to the peritoneum, or from compression of the portal vein; œdema of one or both of the lower limbs, from thrombosis of the corresponding femoral or external iliac vein or veins. In some cases the patient lies for several days before his death with cold blue extremities, and with a scarcely perceptible pulse, but suffering no pain.

Diagnosis.—That the detection of cancer of the stomach is often a very difficult matter must be sufficiently evident from what has been already stated. There are, indeed, some cases in which the most acute observer cannot do more than suspect the presence of the disease. The only rule is that whenever gastric pain comes on without apparent cause in a patient over forty or fifty years of age, and is accompanied with rapid loss of strength and of flesh, the possibility that cancer of the stomach is developing must always be borne in mind.

The most important sign of cancer of the pylorus, when the tumour cannot itself be felt, is the existence of passive dilatation of the stomach. This may be recognised by the increased resonance, by the altered form of the abdomen, and by splashing on succussion. But it is often desirable to map out the extent of dilatation and the form of the stomach more accurately, and for this purpose the plan introduced by Frerichs of filling it with carbonic dioxide is ingenious and useful. The patient takes first a tea-

* The term λειεντερία (λεῖος, *levis*, smooth; ἔντερον, intestine) was applied in Greek medicine to cases of diarrhœa in which food or drink, as soon as taken, seems "to run through the body" without being digested. In ordinary cases the stimulus of food in the stomach provokes peristalsis, but the stools consist of mucus or other intestinal contents.—Ἐπὶ διαῤῥοίῃ, δυσεντερία, ἐπὶ δυσεντερίῃ λειεντερίη ἐπιγίνεται (Hipp., 'Aphor.,' vii, 76).—Intestinorum levitas, quâ continere nihil possunt, et quidquid assumptum est imperfectum protinus reddunt (Cels., lib. iv, cap. xvi).—Levitas intestinorum, Græce λειεντερία, est velox exitus eorum quæ comeduntur atque bibuntur, quæ talia dejiciuntur qualia fuerunt devorata (Stephani, 'Vocab. med. expos.,' 1564).

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spoonful of tartaric acid in solution, and then rather more than half as much bicarbonate of soda also dissolved in water. Carbonic acid gas is at once set free, and the limits of the stomach are seen. If the greater curvature reach below the umbilicus, the stomach is either enlarged or displaced. This plan is often useful in other ways, as in determining the relation to the stomach of a tumour suspected to be the pylorus. It is safer than pumping in gas, or than the plan of introducing a Nélaton's bougie and measuring the distance to which it can be passed (p. 197). But distension of the stomach with gas must never be used as a means of diagnosis when we have reason to fear ulceration of its walls, for it might easily cause fatal perforation.

Even when the symptoms point clearly to the existence of serious organic disease of the stomach, there always remains the question whether this disease is simple chronic ulcer or cancer. Between these affections the diagnosis is often perfectly easy. In very young persons, malignant disease of the stomach may be dismissed from consideration. It scarcely ever occurs except in the form of a sarcomatous growth, attended with ascites, while it produces comparatively slight gastric symptoms. Even in older patients there are many points of distinction.

In cases of ulcer, the pain and sickness are much closer to the time at which food is taken than in those of cancer. Vomiting of blood in considerable quantity is much more apt to occur at an early stage of the disease, whereas coffee-ground vomiting and occasional scanty hæmatemesis is much more frequent in cases of cancer. The duration of life differs greatly; so that any case in which well-marked symptoms have existed eighteen months or more may generally be pronounced one of simple ulcer and not of malignant disease.

Cancer of the stomach may generally be diagnosed whenever a tumour is discovered having the characteristics above described. Cases of simple ulcer affecting the pylorus have, however, been placed on record in which this part has been so thickened and indurated that the presence of a scirrhous mass has been simulated; and when an ulcer occurs at this part of the stomach, it tends, when it heals, to narrow the orifice, and so may give rise to further symptoms resembling those of pyloric cancer.

In considering the diagnostic value of pyloric tumour, it must be remembered that it is sometimes present when there are no other symptoms of gastric disease. Sir Thomas Watson relates a case in point. A young woman had a pulsating tumour in the epigastrium which was at first supposed to be an aneurysm, and afterwards a mass of fæces in the colon. She had no sickness nor any gastric symptom. The tumour proved to be a cancerous growth in the stomach; it lay in front of the abdominal aorta.

On the other hand, it must not be forgotten that in many cases of cancer of the stomach, and even in some cases of cancer of the pylorus, no tumour is at any time to be discovered.

The asserted fact that free hydrochloric acid is absent from the fluid removed by a siphon in cases of cancer of the stomach has been lately much discussed in Germany, and Dr Van der Welden, of Strassburg, introduced an alcoholic solution of tropæolin as a test with this object.*

* See the seventh number of the 'Berliner klinische Wochenschrift' for 1887, and Nos. 6 and 7 of the same periodical for 1888. Also a judicious paper on modern methods of diagnosis in diseases of the stomach, by Dr F. C. Shattuck, of Boston ('Trans. Assoc. Amer. Phys.,' May, 1890).

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But authentic cases are on record of hydrochloric acid being present in the contents of the stomach where cancer was afterwards proved to be present, and of its absence in cases of chronic gastric catarrh and even in persons presumably healthy. Moreover, the tests employed, tropæolin, gentian-violet, and Congo-red, to distinguish between an organic and hydrochloric acid, are none of them quite trustworthy.

Brinton says that he has met with one or two cases in which during the whole progress of the disease there was nothing to justify a positive diagnosis. The supervention of jaundice or ascites or enlargement of the liver would decide in favour of the disease being malignant.

The diagnosis from cirrhosis will be best considered in the chapter on diseases of the liver.

Prognosis.—The duration of cancer of the stomach cannot be stated with precision, because we have no means of fixing the date of its commencement. But it seldom fails to destroy life within a short time from the appearance of well-marked symptoms. Brinton estimated this period as amounting on an average to twelve and a half months. Niemeyer says that the disease generally proves fatal in from five to fifteen months. Wilson Fox says that the most rapid case he can find recorded is one by Valleix, in which death occurred in four months. But at Guy's Hospital four cases have occurred, in which the duration of the symptoms was stated at four weeks, five weeks, nine weeks, and three months. The longest case mentioned by Dr Fox was one in which the patient lived three and a half years after the appearance of the first distinctive symptom; but he cites from Abercrombie the case of Napoleon, who had paroxysms of severe pain for nine years before his death in St Helena. One remarkable case is recorded at Guy's Hospital in which the patient had suffered for seven years from symptoms of disease of the stomach.

Treatment.—With regard to the treatment of cancer of the stomach there is very little to be said. The patient's strength must of course be saved as much as possible; on this account the range of food should not be too strictly limited, and he must not be subjected to the starvation plan of treatment recommended for cases of simple gastric ulcer. Alcoholic stimulants may be allowed in moderation.

Medicines are required only for the relief of particular symptoms. The remedies available in the treatment of gastric pain, of vomiting, and of hæmorrhage have already been fully discussed (*v.* pp. 167, 173, 178).

For the treatment of sarcinous vomiting, Sir Thomas Watson recommends common salt, creosote, and the sulphites or hyposulphites. The last-named remedies were first suggested by Sir William Jenner; the dose is fifteen or twenty grains, and they should be taken soon after meals.

But the presence of sarcinæ in the vomited fluids is in most cases an indication that the stomach is in a state of chronic dilatation, and of this condition there is still something to be said. We have seen that it is one of the effects of cancerous disease of the pylorus, and that it may also follow the cicatrisation of a simple ulcer affecting the same region. Those writers who admit that the pylorus is liable to simple hypertrophy, regard this as another occasional cause of dilatation of the stomach. Paralysis of the muscular coat limited to the pyloric portion is also mentioned by Dr Wilson Fox, who quotes two cases from Andral in which the stomach was greatly enlarged without there being any actual narrowing of the pyloric orifice. But in one of these instances there was extensive ulceration of the

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pylorus; and in the other its walls were indurated, although the muscular coat was atrophied. And even in cases of cancer affecting the pyloric orifice, it sometimes happens that, after the ordinary symptoms have developed themselves and advanced to a fatal termination, it is found on *post-mortem* examination that the finger can still be readily passed into duodeum. Evidently, then, the outflow of the contents of the stomach may be arrested, without there being an actual mechanical closure of the channel.

Nevertheless, it is doubtful whether this kind of chronic dilatation of the stomach ever occurs independently of obstruction of some kind at the pyloric orifice. Bamberger says that it may be due to dragging down of the stomach by an omentum adherent to a hernial sac.

Great success in cases of chronic dilatation of the stomach has been attained by the systematic use of the stomach-pump, or rather the stomach-siphon. This treatment was first proposed by Professor Kussmaul, of Freiburg (now of Strassburg), in 1868. A long flexible tube filled with water is introduced into the stomach every day. On lowering the longer half, the water flows out and the contents of the stomach follow it. As much of the contents as will come away readily should first be withdrawn. Some tepid water is then to be injected and afterwards withdrawn again, and the process should be repeated two or three times until what returns is almost clear. Weak solutions of carbonate of soda, or of permanganate of potass, or even of creosote, may afterwards be injected. Dr Schliep introduced the practice into England in 1872 (Clinical Society's 'Transactions,' vol. vi, p. 41). It has now been tried in numerous cases, both in this country and abroad, and with decided success. The first introduction of the tube is exceedingly disagreeable to the patient; but before long he becomes accustomed to it, and he is even glad to pass it himself, so great is the relief which he experiences from its use. The vomiting often ceases entirely; there is usually great diminution of pain; the appetite improves considerably; the patient becomes more cheerful; he regains much of the flesh and strength that he had lost, and he is no longer troubled with constipation.

Such striking results are not, however, to be looked for in those cases in which dilatation of the stomach is the result of cancerous disease of the pylorus.

In recent years surgeons have ventured to open the abdomen and excise a cancerous pylorus. The possibility of the operation was proved by Heidenhain in the course of his experiments on the gastric and pyloric secretion in dogs. It was first practised on a human being by Billroth, and was not followed by immediate death. Mr Maylard performed excision of the pylorus on a patient of Dr Coats at Glasgow ('Brit. Med. Journ.,' July 24th, 1886), and Sir Wm. Stokes in 1890 (*ibid.*, i, 997).

Other surgeons, following Prof. Loreta (1882), of Milan, have opened the stomach and forcibly dilated the pylorus with the fingers, and others have avoided the obstruction by making a communication between the stomach and the third part of the duodenum.

Such cases of excision or of dilatation of the pylorus, and of "short-circuiting" by gastro-jejunal union are still rare.

Pylorectomy has been performed by Péan in Paris, by Billroth in Vienna, and by Senn in America, and more recently in this country also. Loreta's operation has been in some cases unexpectedly successful. Gastro-enterostomy has been followed by partial success in a few cases in Germany. Mr. Golding-Bird published a case of "jejunostomy" for pyloric cancer in the 'Clinical Transactions' for 1886 (vol. xix, p. 70).

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Gastric induration.—Another organic disease of the stomach is one in which its walls are uniformly thickened, without the development of any morbid growth, its cavity being at the same time greatly reduced in size. This affection is spoken of by systematic writers as "fibroid induration" or "cirrhosis" of the stomach. We shall see hereafter that it is occasionally the starting-point of a general chronic peritonitis. The coats of the organ may be from half an inch to an inch and a half thick, and only capable of containing four or five ounces of fluid.

The symptoms of this affection are exceedingly obscure. A tumour may be discoverable, and this may be more or less resonant on percussion. Probably it would be impossible to distinguish cases of this kind from those of diffused cancer of the stomach.

This fibroid thickening of the coats of the stomach has probably its first stage in the thick, pigmented, almost warty condition (*état mamelonné*) of chronic gastritis. It chiefly affects the pyloric region, and occasionally forms what may be termed a non-malignant fibrous tumour of the pylorus. Sometimes, however, it extends to the whole of the stomach, which, to use Dr Bristowe's words, "retains its form like an india-rubber bottle."

Gastric concretions.—Brief mention must be made of certain rare cases in which immense masses of hair and string, matted together and moulded to the shape of the stomach, have been found in its cavity, and in that of the upper part of the intestine. Sir William Gull brought a case of this kind before the notice of the Clinical Society in 1871, and another was related at a meeting of the Pathological Society by Mr Pollock. In the former case the mass, when dried, weighed five and three quarter ounces; it was composed of string, thread, cotton, wool, and hair of three colours, that of the patient herself (a woman aged thirty-two) and of her children. She had never been noticed to eat hair; but the person from whom Mr Pollock's specimen was taken, and who was a delicate girl aged eighteen, had been observed to put hairs into her mouth when only three or four years of age. In that case a projecting tumour, the size of a large orange, was felt in the epigastric region during life; it was apparently solid and slightly moveable. A tumour was also felt in a third case, referred to by Sir William Gull. It occurred in a woman aged thirty, who for fifteen years had indulged in the habit of eating her hair, and who had suffered all the time from pain in the stomach, but had worked as a servant until six years before her death. In that case the mass weighed thirty ounces.

None of these patients were of unsound mind; in lunatics a similar condition is not infrequent. A fatal termination appears generally to occur sooner or later from perforation of the stomach, with consequent acute peritonitis.

In a case of the kind, Mr Knowsley Thornton removed from the stomach of a girl of eighteen by abdominal section a mass of hair weighing two pounds, and the patient made a good recovery. He refers to a similar case successfully operated on by Dr Schönborn, of Königsberg, and quotes several instances only discovered after death. In one of these the mass of hair when removed from the stomach was found to weigh four pounds and seven ounces.

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FUNCTIONAL AND INFLAMMATORY DISEASES OF THE INTESTINES

"When my Lord went to Bed, he fell very sick of the [bloody Flux], which caused him to go to Stool from time to time all that Night, insomuch that from that time till Morning he had fifty Stools; and the Matter that he voided was very black, which the Physicians call'd *adustine*, whose Opinions were that he had not above four or five Days to live."—
CAVENDISH: *Life of Cardinal Wolsey.*

COLIC—*Onset—Causes—Diagnosis—Prognosis and treatment—Lead-colic—History of its recognition—Modes of infection—Diagnostic characters—The "blue line"—Pathology and treatment—Other effects of lead.*
CONSTIPATION—*Origin—Effects—Treatment—Clinical varieties.*
DIARRHŒA—*Acute and epidemic form—Chronic form—Tubercular enteritis—Diarrhœa from lardaceous and malignant disease—Symptoms—Treatment.*
TYPHLITIS—*Relation of inflammation of the cœcum to perityphlitis and disease of the appendix—Origin and course—Diagnosis—Treatment.*
DYSENTERY—*Anatomy—Catarrhal and diphtheritic forms—Sporadic and epidemic dysentery—Ætiology—Symptoms, diagnosis, course, and event—Prophylaxis—Treatment of acute and of chronic dysentery.*
Acute catarrhal colitis—Ulcerative colitis—Intestinal casts.

THAT part of the alimentary canal which lies below the stomach is liable, like almost all other organs of the body, both to disorders of function and to diseases of structure. As usual, we will begin with the former.

COLIC.*—Of this malady the main symptom is pain, of a twisting or dragging or wringing character, generally referred to the umbilicus or to some spot in the upper part of the abdomen. It comes on in paroxysms which are often of extreme severity; but during the intervals the patient may be perfectly easy, and there is frequently no tenderness on pressure. The patient rolls about in the hope of finding relief, or lies on his stomach with his hands clasped together beneath him, or leans with the whole weight of his body across the back of a chair. In exceptional cases, however, pressure increases the pain, especially when parts of the intestines are distended with gas.

An attack of colic is often attended with nausea and vomiting. Writers say that the skin is cool, and that the pulse is often slower than natural; but in a case of severe abdominal pain, admitted into Guy's Hospital one night, the fact that the temperature was two or three degrees higher than normal led to some doubt as to its real nature; yet next morning the patient was well, and a review of the symptoms seemed to prove that the attack had been one of colic. In another case of colic, which the writer watched for some hours with not a little uneasiness, the skin was covered with a profuse cold sweat, and the pulse was much quickened. The

* *Synonyms.*—Passio colica—Colum (Pliny)—Enteralgia—The gripes.—*Fr.* La colique.

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expression was anxious, but there was not the peculiar sunken look of the features which belongs to the more dangerous forms of abdominal disease.

The *immediate cause* of colic appears to be a spasmodic contraction of some part of the large (or perhaps of the small) intestine. Associated with this there may be an accumulation of gas in adjacent parts of the bowel; and the attacks of spasm are then attended with rumbling noises, "borborygmi," which are audible to the patient and those about him. In such cases there may be partial distension of the abdomen; but in most cases of colic it is hollow, the walls are hard, and the muscles feel drawn up into knots.

A cause of colic which must never be forgotten is plumbism; this form will be described separately.

In most other cases colic is due to the presence of indigestible food. Eating unripe fruit is one of the most frequent causes of colic. It also may follow the ingestion of meat, sausages, or game that is tainted or too "high." Ices may produce the same effect, or mushrooms or hard potatoes or turnips. Even if the food is wholesome and properly cooked it may excite colic by being swallowed too quickly without due mastication and insalivation. Most purgative medicines give rise to intestinal pains, which are essentially the same as those of colic.

Under some of these conditions the complaint is associated with diarrhœa, or the patient's sufferings may go on increasing in severity until one or more loose evacuations are passed, with more or less lasting relief. But in the more typical cases of colic constipation is a prominent symptom, the attack has no tendency to terminate of its own accord by the bowels acting, and the administration of medicine is necessary. The exciting cause is generally the presence of hard scybalous masses in some part of the large intestine; and they can often be plainly felt through the abdominal walls.

From what has been said in the previous paragraph it is evident that colic cannot be regarded as a true neuralgia. It is local affection of the muscles—a kind of intestinal cramp.

The *diagnosis* of colic is often easy, but always needs caution. For some of the most dangerous forms of inflammation to which the abdominal viscera are liable may for the first few hours present very similar symptoms, and a mistake may be attended with fatal consequences. Certain writers, indeed, define colic as a painful affection, dependent on spasm of the bowels, and regard it as present in cases of strangulation of the bowels, though only as a minor feature of the disease. But it is more convenient to limit the use of the term to those instances in which the pain is, so far as our knowledge carries us, the substantive complaint—as we do with such terms as epilepsy, neuralgia or dyspepsia. The rule must then be that no case should be set down as one of colic, and treated so, unless its characters conclusively prove that no organic disease is present. The most important of these characters are a retracted, hard, knotted state of the abdomen, the fact that pressure relieves the pain, and the absence of pyrexia and other indications of general disturbance. Sometimes important light is thrown on a case by the statement that the patient has had former attacks of the same kind, which have passed off in a few hours; or that he has not long before eaten something which he knows by previous experience may cause severe griping pain.

Colic has also to be distinguished from other functional disorders. One

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is a form of gastric pain with distended stomach which has already been described (p. 168). The epigastrium is prominent in both affections, but they can generally be discriminated by gentle percussion; the note being (as Dr Wilson Fox observes) less prolonged and higher pitched over the colon than over the stomach. Moreover, the pain has seldom exactly the same position in colic and in gastrodynia; in the former it often extends into the right hypochondrium or downwards into the left iliac fossa in the direction of the sigmoid flexure, whereas in the latter it is absent from these regions of the abdomen. In doubtful cases inflation of the stomach with carbonic acid gas as described above (p. 199) would decide the question.

The so-called "biliary colic" and "renal colic" have also to be distinguished from true intestinal colic.

Treatment.—An attack of colic always terminates in the recovery of the patient, and that within a few hours, or a day or two at the outside. It is therefore not of the greatest importance to adopt active treatment, particularly at first. And since some of the drugs by which this complaint would be shortened are precisely those which would do the greatest possible harm if the disease should be obstruction of the bowels or peritonitis, one cannot be too careful to avoid interfering in any doubtful case.

Colic, in fact, is the only exception to the rule which Dr Wilks used to lay down, that whenever a pain in the abdomen is so severe as to cause the patient to send for a medical man, this *ipso facto* proves that the administration of a purgative is unjustifiable. The rule itself is of great value, and should be kept constantly before one's mind; but there are a few cases in which it ought to be infringed. The patient may clearly owe his attack to something which he has eaten, and which has disagreed with him; he may have suffered in the same way before, and have quickly got well after taking a purgative; his abdomen may be retracted and hard, the pain may be relieved by pressure, and it may be entirely paroxysmal, with complete intermissions. Under such circumstances one is fully justified in giving him at once an ounce of castor-oil with twenty or thirty minims of tincture of opium, and in directing that half as much should be taken again at the end of three hours if the bowels should not have acted in the meantime. Enemata of turpentine or assafœtida may be prescribed if there should be accumulation of gas in the intestine. The abdomen may be rubbed with a stimulating embrocation, or an india-rubber bottle filled with hot water may be laid across it, or a hot bran poultice. A hot hip-bath sometimes gives great relief.

LEAD-COLIC.—Of all the causes of colic the most remarkable is absorption of lead into the blood.

Long before this fact was ascertained the complaint itself was well known as of endemic occurrence in certain parts of England and of the Continent. Thus it prevailed in Poitou, and was hence called *Colica pictonum;* in Devonshire, so that within the five years ending in 1767 two hundred and eighty-five cases were admitted into the Devon and Exeter Hospital; and in the West Indies, where it received the appropriate name of the "dry belly-ache." In each instance it was formerly attributed to some local beverage; in Poitou to wine; in Devonshire to cider; in the West Indies to rum. In the first half of the eighteenth century Huxham endeavoured to refer it more definitely to the "tartar" contained in all of these drinks.

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The discovery that this form of colic is due to the action of lead was first made by Sir George Baker, whose paper on the subject, read at the College of Physicians in 1767, is still quoted as a masterpiece of inductive logic. He showed first that in the counties of Hereford, Gloucester, and Worcester persons who drank cider did not suffer from colic; and next that Devonshire cider contained lead, from which Hereford cider was free. Next he traced the presence of the metal in the former to the facts that lead was used in the construction of the cider-presses, and that leaden weights were sometimes put into the casks to prevent its turning sour. Not long afterwards it was shown that preparations of lead were added to the wines made in Poitou with the same object of neutralising acidity, and that in the West Indies the stills in which rum was made had leaden worms. An important link in the chain of evidence was the circumstance that both in Poitou and in Devonshire a peculiar form of paralysis affecting the upper limbs was commonly associated with the colic, and this also was traced to the poison; it was described in the first volume of this work (p. 519).

The recognition of the cause of the complaint soon led to the disappearance of endemic lead-colic from drinking cider, wine, or spirits. But as a sporadic affection it is still often met with.

As might be supposed, those who work in the manufacture of white-lead are very apt to be attacked by colic. This is believed to be mainly due to the diffusion of the plumbic carbonate as a powder throughout the workshops, so that, besides being inhaled in respiration, it collects upon the hands and is carried into the mouth with the food. It is doubtful whether lead is ever absorbed directly through the skin.

Painters and plumbers often suffer from the disease. It is sometimes observed also in glassmakers, enamellers, shot-makers, printers and type-founders, but not so commonly as was at one time supposed. In all these occupations it is said to occur more seldom than formerly; and if the men were but sufficiently careful, it is probable that only those employed in making white-lead, or sugar of lead, would be attacked with lead-colic.

Lead-poisoning has been sometimes known to arise from the use of snuff with which the red oxide, or the yellow chromate, had been mixed, or which had been fastened up in lead-foil.

As an example of the production of colic by lead contained in food Sir Thomas Watson quotes the case of the troops at a station in Ceylon in 1832. More than seven tenths of those who made up the force were attacked, and the cause was found to be the presence of lead in some coarse sugar which had been distributed among the soldiers from one particular estate.

A more widely spread cause of plumbism is the impregnation of drinking-water with the poison, and this source of the disease is the more important because it is so liable to be overlooked. A well-known instance is that of the family of King Louis Philippe when living in exile at Claremont: several persons were attacked at the same time. The amount of lead in the water which they drank was seven tenths of a grain per gallon. Now, it is well known that water containing carbonic acid and certain salts of lime has less action on metallic lead than water which contains such ingredients only in minute proportions or from which they are altogether absent. Thus the distilled water sometimes used for drinking purposes on board ship is particularly liable to be impregnated with the metal; and even zinc vessels may contain enough lead to make distilled water which has stood in them injurious to health. It must be added, however, that the

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cause of colic occurring on board ship, especially in the French navy, has been a matter of much discussion; certain writers are still of opinion that it may be due to some climatic condition or unknown dietetic cause.

An inquiry held at Sheffield in 1889 showed that the water supplied to the town was capable (owing to a very slight acid reaction, and the absence of carbonates) of dissolving lead out of the pipes and taps through which it flowed; and numerous cases of colic, constipation, anæmia, and wrist-drop were observed in persons who showed by the characteristic blue line on the gums that they were the subjects of poisoning by lead.

There are marked individual differences in susceptibility to the influence of lead. Sir Thomas Watson mentions persons in whom the colic was caused by their sleeping for a night or two in a freshly painted room; and he contrasts with such cases that of a painter, whose first attack occurred when he had followed his occupation for nineteen years.

There is no satisfactory explanation of the fact that the introduction of lead into the body causes colic. The metal is deposited in the tissues; but, according to the analyses of Dr George Wilson, of Edinburgh, there is less of it in the intestines than in several of the other organs. In the case of a woman under the writer's care, who died from plumbic eclampsia, Dr Stevenson discovered 3·5 grains of lead in the liver, which weighed 45 oz.; ·465 grain in the spleen, weighing 4 oz.; ·246 grain in a deeply pigmented part of the colon, weighing 6 oz.; ·054 grain in the heart, weighing 10 oz.; and none at all in 8 oz. of cerebral matter.

Diagnosis.—Ordinary colic and the affection caused by lead do not differ in their symptoms. There is, however, this peculiarity: that whereas mild cases of the former scarcely ever occur, at least so as to come under medical observation, there are not a few instances of the latter in which the pain is of trifling severity and the abdomen soft and supple. Thus the patient may continue to suffer from the complaint for a considerable period, and yet go on with his work, absorbing all the time more and more of the poison.

What alone enables one to speak positively as to the real nature of such cases is the fact that the presence of lead in the body is revealed by a peculiar discoloration of the gums. This was noticed by Dr Burton of St Thomas's Hospital, in 1840, and it is commonly spoken of as the "blue line." The name however, is unfortunate, and has no doubt often led to mistakes; for under conditions of irritation the margin of the gums is very apt to present a purplish border, which has nothing to do with the presence of lead. The line which is really characteristic presents peculiarities which were accurately noticed by Sir William Gull. It consists at first of a single row of black dots, corresponding with the vascular papillæ of the normal mucous membrane. It is in the tissue of the gums, not between them and the teeth; and when complete it has a wavy course, easily detected by a lens if not by the naked eye. Mr Tomes several years ago proved that it was caused by a chemical action between the lead and the "tartar," or deposit of calcareous salts from the saliva which forms upon the teeth. At the same time he pointed out that in all probability the constituents of the tartar itself are not concerned in its production, but rather animal matters which had penetrated into the pores of the tartar, and of which the decomposition set free sulphuretted hydrogen. He showed that where there is a gap between the teeth, so that tartar is absent, no "blue line" is formed. Further evidence of the same fact is afforded by cases (of which more than one were noticed by the author) in which persons who kept the teeth

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very clean have failed to present the line, although they were indubitably affected by lead. In many cases the line is exceedingly partial. There may be only two or three black dots on one or more of the processes of gum projecting up between the teeth; and a lens may be necessary to enable their true nature to be determined. The lead, with which the sulphuretted hydrogen combines, probably comes directly from the circulating blood, whence it is precipitated in an insoluble form. Thus only can we account for the way the dots correspond with the vascular papillæ of the gum.

The author had one opportunity of examining microscopically the gum of a person who had died while affected by lead-poisoning; and he found that the colour was due to the presence of a multitude of minute granules. Some of these were aggregated together within small blood-vessels, the ramifications of which were mapped out by their presence; others were arranged in double lines which probably correspond with the exterior of other vessels. Thus it seems that the "blue line" is really due to an *excretion* of lead from the blood; and this accounts for the undoubted fact that when iodide of potassium is given to a patient suffering from the poisonous action of lead, but in whom the line happens to be ill developed or absent, the appearance in question often becomes well marked within a few days, just as the lead can then be found in the urine, although none was being excreted previously. We have had several instances in which a blue line has thus been brought out by iodide of potassium while the patient was an in-patient in the hospital for symptoms due to lead-poisoning; and the late Dr Frank Smith, of Sheffield, long ago made the same observation.*

It was at one time supposed that other metals, such as copper or bismuth, might be capable of producing similar appearances.† But there is no evidence that this is the case; and, as regards copper, Dr Clapton has shown that what it really causes is a bluish-green line on the teeth themselves.

When the salts of lead are given medicinally in considerable doses the line often makes its appearance very quickly. Dr Burton met with instances in which it was developed within two days—one within twenty-four hours—the quantity of acetate of lead taken by each patient having then been only from fifteen to twenty-four grains. When the blood is richly impregnated with lead, and when the teeth are so neglected that plenty of sulphuretted hydrogen is provided, the line may go far beyond what has just been described. The spaces between the dots may be filled up by a uniform black discoloration, which spreads over the gum for some distance from the teeth. The insides of the lips may also present a similar staining; in one patient at Guy's Hospital this was half an inch broad.

Morbid anatomy.—At the present day lead-colic is seldom or never fatal, at least in England; but formerly patients seem not unfrequently to have

* These views appear to afford an explanation of a circumstance, noticed by Mr Tomes, which has given rise to some doubt in regard to the clinical value of the "blue line" as an indication of the presence of lead. This is the fact that such a line has sometimes been observed in persons who have not been known to be exposed to the influence of the poison, and who show none of its symptoms. Evidently, if the black granules consist of precipitated sulphuret of lead, the introduction of the smallest quantities of the metal into the blood from time to time might ultimately lead to the formation of a blue line. And there is no one to whose body such minute proportions of lead might not have access.

† Dr Bristowe still believes that bismuth, and perhaps other metals, may sometimes produce a line on the gums resembling that caused by lead. But this seems very doubtful. His account of the appearance which he had observed in one or two instances is that the line was "bluish-red," "wider and redder" than the lead line.

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died of it. In such cases, and in those in which during an attack death occurs from some other cause, the alimentary canal is generally said to present no morbid change of importance. Several *post-mortem* examinations were made by Andral and by Mérat ('Traité de la Colique métallique, vulgairement colique de Poitou,' 1810). The former found the intestines free from inflammation, and neither dilated nor contracted; the latter, however, observed the colon to be contracted, and he also noticed the same thing in rabbits poisoned by lead. It is true that contraction of the large intestine is not very uncommon in persons who have died from various causes, but if constantly found in cases of plumbism it would be significant.

Some time ago a *post-mortem* examination was made upon a patient of Dr Moxon's who had died of heart disease, but who was a painter and had a well-marked blue line. Several years before he had had colic, and shortly before his death he complained greatly of pain about the splenic flexure of the colon. At this part of the bowel, and also in the transverse colon, there were scattered several slate-coloured patches, which, although not indurated, looked puckered, and which, if they were not actual cicatrices, must have resulted from extravasations of blood at some former period. Dr Bristowe showed, at one of the meetings of the Pathological Society, the mucous membrane from the colon of a painter, who had died in St Thomas's Hospital of what was supposed to be colic; the intestines were enormously dilated, and some parts were black from hæmorrhage into the mucous and submucous tissues. Intestinal hæmorrhage, however, is not a symptom of lead-colic, so that possibly the remarkable appearance noticed in these two cases may have been only a coincidence with the patient's exposure to lead.

Prophylaxis.—The directions given to workmen whose occupations bring them into contact with lead are chiefly that they should pay great attention to personal cleanliness, that they should prevent as much as possible the poison from entering their air-passages, and above all that they should not swallow any particles with their food. They should have an outer suit of linen clothing, worn only while they are at work, and washed at least once a week. They should never take their meals in the workroom. When there is much dust, masks or respirators would probably be useful, but the men can seldom be induced to wear them. Many years ago Liebig recommended the habitual use of "sulphuric acid lemonade"—a liquid containing a small quantity of sulphuric acid sweetened by sugar, which it was supposed would render any compounds of lead that might enter the stomach innocuous, by converting them into an insoluble sulphate; and Sir Thomas Watson stated that in some works at Birmingham the addition of the acid in question to the treacle beer which the men drank caused the disappearance of colic. It had before prevailed to a distressing extent; afterwards not a single case occurred for fifteen months.

In the case of the water supplied to Sheffield, referred to above, it was believed, after consultation with chemists and engineers, that the most easy, harmless, and effectual remedy was to add chalk to the water in the great "dams" or reservoirs on the surrounding moors from which the town is supplied.

Treatment.—Colic arising from lead must be treated immediately in the same way as any other form of the complaint. Sometimes there is considerable difficulty in bringing about an action of the bowels, so that two or three successive doses of castor oil with laudanum may be required, and it may even be necessary to add one or two drops of croton oil; but when once a

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free evacuation has occurred all the symptoms generally disappear. The patient should, however, take a course of iodide of potassium. This salt possesses the power of eliminating from the body the lead which had been deposited in the tissues, forming with it a soluble compound, which is absorbed again into the blood and then excreted by the kidneys. This was long ago established by the observations of Nicholson and Parkes. In a marked case at Guy's Hospital, the urine had contained no lead before the patient began to take the iodide, whereas the presence of the metal was afterwards detected without difficulty. Probably it is because the lead is apt to remain in the body all through an attack of colic, that the complaint sometimes relapses after it has been cured, without such a course of depuration.

Other effects of plumbism beside colic are a peculiar form of atrophic palsy chiefly affecting the extensors of the forearm, atrophy of the cerebral cortex, epilepsy or epileptiform convulsions, sometimes accompanied by mania, anæmia, myalgia, menorrhagia, abortion, chronic atrophic nephritis, and saturnine gout. Some of these have been already described (vol. i, pp. 519, 845), and the rest will be noticed in their place in chapters on Bright's disease and on gout.

CONSTIPATION.*—This is the most frequent disorder of the bowels. It has already been mentioned as a symptom of most fevers, of cerebral disease, of dyspepsia, and of colic; it is frequent in cases of jaundice, and is one of the most obvious effects of mechanical obstruction of the bowels. But constipation is still more often met with as a primary complaint.

Pathology.—There are great differences in the frequency with which healthy persons go to stool. In some an action of the bowels occurs only at intervals of three or four days, and yet they suffer no inconvenience. This is not a condition which calls for, or would justify, any medical interference. But in children and young persons the fæces are often retained for several days at a time, merely because they are careless, or unwilling to face the outside air, or too modest to be seen going to a water-closet. Thus the periodicity, which is so important an element in most of the bodily functions, is lost.

Apart from such cases as those last mentioned, in which at first the will is alone concerned, the exciting cause of constipation is either that the peristaltic action of the bowel is too slow or deficient in force, or that the fæces are too dry and hard, so that they do not readily pass down the intestinal canal. In other words, there is either paresis of the muscular coat of the intestines, or deficiency in the secretion of succus entericus. Both functions are under the influence of the intestinal nerves; and some writers have supposed a "sluggish state of the nervous system" as the prime cause of constipation, arguing from the undoubted fact that a tendency to the complaint is often inherited.

Of the more immediate causes may be mentioned excessive abstemiousness in eating, and habitual restriction to a diet which is too exclusively animal. Sedentary habits also help in preventing the bowels from acting properly.

When there is mechanical interference with the passage of the intestinal contents, mere constipation is merged in a more serious condition—that of obstruction of the bowels—which will be described separately. A caution may, however, be given here, that when a woman suffers from habitual tor-

* *Synonyms.*—Obstipatio—Alvus adstricta.—*Fr.* Constipation.—*Germ.* Hartleibigkeit.

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pidity of the intestines, the possibility must always be borne in mind that this may be due to the presence of a uterine or ovarian tumour, or of a prolapsed or displaced womb.

According to O'Beirne, the rectum in health is generally empty, and when a fæcal mass, even of small size, has entered this part of the bowel it ought at once to excite sensations which lead to its expulsion.

Among the discomforts caused by slight constipation not the least is that which results from the passage into the rectum of isolated round pellets which had been moulded in the sacculi of the colon; these may excite a great desire to go to stool, and yet they are passed only after violent straining efforts. Patients in whom this occurs often speak of themselves as suffering from diarrhœa, and only a strict cross-examination can elicit the real state of the case. Dr Bright was once summoned into the country, in consultation with an eminent surgeon and a general practitioner, to see a lady who had been in vain treated with astringents, being supposed to suffer from a relaxed state of the bowels. He asked to see the evacuations, whereupon a single little hard pellet of fæcal matter was shown to him, and it was at once clear that a purge alone would give relief.

Even when the colon or sigmoid flexure is the part in which the fæcal masses accumulate, their presence often gives rise to a sense of weight and discomfort, and to colicky pains. But in persons who are habitually constipated the rectum itself loses its natural sensitiveness, and may then become obstructed by hard, dry scybala of enormous size. Under such circumstances the bowel sometimes becomes irritated and pours out mucus, and this, or fluid fæcal matter, may pass down by the side of the retained masses, so that a condition of diarrhœa may be closely simulated.

Habitual constipation has a marked influence on the general health and spirits of the patient. It is well known to make the tongue furred and the breath foul. It causes an unpleasant taste in the mouth. It gives rise to feelings of languor and melancholy, and makes the countenance depressed and sallow.

Treatment.—One cannot wonder that those who suffer from such discomforts are ready to take purgatives almost every day and in constantly increasing doses. One hears of persons who for years have never had an action of the bowels except as the result of aperient drugs, and most quack medicines consist of aloes, gamboge, or purgative salts. Each time that they are taken they cause free evacuations, but they exhaust the susceptibility of the intestine, and render it less capable than before of responding to natural stimuli.

The proper treatment of inveterate costiveness is the reverse of this. One may be obliged at first to prescribe a draught, or to clear out the lower part of the intestine by enemata. But from that time all ordinary purges should be scrupulously avoided. The following plan of treatment was recommended by Dr Spender, of Bath, in the 'Medical Times and Gazette' for 1870. It consists in the regular administration of a pill containing from one to three grains of sulphate of iron, and about a grain of the watery extract of aloes, or the compound extract of colocynth, or the compound rhubarb pill. At first the patient should take three pills a day, one after each meal. He should be told that for two or even three days he is not to expect a motion, but that when the bowels have once acted they will be moved more frequently. And now comes the point of importance—that whenever there is a loose evacuation he should instantly decrease the number of pills which he takes. Nothing approaching to a pur-

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gative effect should ever be permitted. Very soon two pills a day are sufficient; and a fortnight later a single one perhaps produces the desired effect. Within another month he is able to do with a pill once or twice a week. If the patient should make a difficulty about taking pills, the best substitute for them is a combination of the compound decoction of aloes and Griffiths' steel mixture.

The cases which Dr Spender reports are very striking, and after repeatedly putting this plan in practice the author can confirm his testimony to its value. Patients may at the same time take a spoonful of olive oil once or twice a day, or eat brown bread, or take a glass of cold water on first rising in the morning. The diet, indeed, should always be carefully attended to, and regular bodily exercise must be taken. Another important point is that the patient should seek relief at a regular hour each day, and allow the necessary time for the bowels to act. If the *habit* can be acquired half the battle is won.

When dieting fails, Trousseau and many French physicians advise enemata of water, at first with the chill off, afterwards quite cold; or suppositories of cacao-butter or soap. But it is surely unadvisable to employ measures of this kind, which must tend to render the bowel less susceptible to its natural stimulus. The drug which Trousseau chiefly recommended in cases of habitual constipation was the extract of belladonna.

No plan, however, is always and in all cases the best, and one must adapt one's treatment more or less to the patient.

(*a*) In children constipation is seldom obstinate, and is usually the result of the acuter form of dyspepsia: they are, as a rule, more inclined to looseness of the bowels.

Costiveness in infants often depends on too much starchy food being given, and is best treated by animal broths being added to their diet, or by substituting well-made oatmeal porridge for wheaten flour. Lactose, maltose, and manna are each useful in such cases. Castor-oil should only be used on occasion, not as a habitual aperient. Friction of the abdomen is useful, particularly when constipation is combined with colicky pains. A small piece of soap is a harmless and useful substitute for an enema.

With older children, constipation is often the mere result of carelessness, or of over-eating, and needs no drugs for its cure. As an occasional laxative castor oil is the safest and most effectual. In the less frequent chronic cases, the combination of rhubarb and magnesia (or rhubarb and soda) with an aromatic, known as Gregory's powder, the Pulv. Scammonii Comp., or the Pulv. Glycyrrhizæ Comp., are each useful, and better than sulphur or senna, or saline laxatives. Cascara sagrada is also a harmless domestic medicine. But the diet to be presently described is as a rule sufficient.

(*b*) When constipation is associated with anæmia and amenorrhœa in young women, it is necessary to give iron in addition to laxatives. Griffiths' mixture and Blaud's pills, which are equally famous in Germany, consist of sulphate of iron and carbonate of potash; pilulæ Rufi (pil. aloës c. myrrhâ) are usually combined with these, or decoction of aloes is given separately.

(*c*) In the most numerous cases of all, those of habitual idiopathic constipation in adults of both sexes, the disorder not infrequently disappears of itself during a holiday. Sedentary occupations favour it, and exercise is often a cure. Yet we find that the demand for patented purgative pills is scarcely greater in European capitals than amid the

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patriarchal simplicity of Oriental life, the ruder barbarism of Central Africa, or among Australian herdsmen, who live in the open air and are always in the saddle.

The first indication is to avoid habitual medicine if possible. Exercise and diet should be tried first. Oatmeal porridge at breakfast, treacle or honey, brown bread, chocolate, and fruit—particularly figs, stewed prunes, and baked apples—are all valuable. A larger proportion of vegetable to animal food and of liquids to solids should be taken; a tumbler of water (cold or hot) while dressing, and a baked apple for supper, is an excellent prescription for habitual costiveness. With many persons a pipe or cigarette after breakfast seems to act as an efficient stimulus to peristalsis, perhaps only by directing the thoughts and favouring regular periodicity.

When drugs are necessary, small doses of belladonna at night, and nux vomica and aloes or rhubarb before dinner, form the best laxative pills; while aperient salines may be added to the early draught of water.

Some persons are certainly better if they take a purge once a week, once a fortnight, or (as was once stipulated in the indentures of London apprentices) once a month. For this purpose two compound rhubarb pills, with or without two or three grains of blue pill, may be taken overnight, or a calomel and colocynth pill may be resorted to, followed by a seidlitz powder or other saline draught the next morning.

(*d*) In patients who are subject to gout, and in those who habitually live too freely, a blue pill followed by a black draught is still the best occasional remedy; and in chronic cases of gouty dyspepsia with constipation a saline laxative before breakfast is often the best treatment, taken for weeks or months together. Rochelle salts, Carlsbad salts, Friedrichshall, or Hungarian bitter waters (Hunyadi-Janos), are best adapted for this purpose.

(*e*) The constipation of women at the climacteric period of life is generally best treated by a combination of sulphate of iron, sulphate of magnesia, and aromatic sulphuric acid, attention to diet, and substitution of cocoa or coffee for tea.

(*f*) Lastly, the atonic costive state of the bowels which may be called senile constipation is perhaps the only one in which the habitual use of clysters is advisable. Of drugs, belladonna is in these cases the most valuable; nux vomica comes next, and may often be combined with a small quantity of aloes as a daily pill. Many cases of what looked like malignant disease of the rectum or sigmoid flexure have been cured by the persevering use of unmedicated enemata and belladonna. Of this we had in 1887 a striking instance in Philip Ward, where a case of most obstinate constipation with tympanites and visible peristalsis was thus treated.

DIARRHŒA.*—A frequent result of intestinal disorder is *diarrhœa*—the discharge of the contents of the bowel in a fluid condition, and with excessive frequency.

This depends upon one or both of two causes; an increase in the peristaltic movements of the bowels, or an increase in their secretion.† It is

* *Διάῤῥοια, i. e.* a running through: *διαῤῥοίας ἅμα ἀκράτου ἐπιπιπτούσης, οἱ πολλοὶ ὕστερον δι αὐτὴν ἀσθενείᾳ ἀπεφθείροντο*—of the Plague of Athens, Thuc., ii, 49.—Alvus soluta—Looseness of the guts.—*Fr.* Diarrhée.—*Germ.* Durchfall.

† See a report to the British Association by Dr Brunton and the writer, on the nervous mechanism of intestinal secretion and movement (1874—1876).

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often difficult to distinguish between these two conditions; and no doubt, in many cases, both are in action simultaneously.

Subacute diarrhœa.—The exciting cause of an attack of diarrhœa is often very definite. Sometimes mental influences give rise to it; it attacks the child who is in dread of being punished, or the man who is about to preach or to lecture. Exposure to cold is undoubtedly in some persons its starting-point, and in certain cases such diarrhœa takes the place of coryza in persons whose mucous membranes are susceptible to a chill. It often follows quickly upon the ingestion of some irritating substance, medicinal or dietetic. In infants it is apt to be set up by amylaceous food, which at that time of life cannot be digested, since the secretions which convert starch into sugar are not formed during the first few months after birth. Some infants, however, have diarrhœa even if they are fed with the best cow's milk.

Impure drinking-water is another frequent cause of diarrhœa. In Parkes's work on 'Hygiene' references will be found to numerous observations in which this effect has been produced by water containing suspended or dissolved mineral substances of various kinds, or suspended vegetable matters. Such water, however, would scarcely be used for drinking purposes except by necessity. As a rule, the danger arises rather from the unsuspected presence of animal matters, especially those of fæcal origin. Thus diarrhœa has often been traced (as in Croydon in 1854 by Dr Carpenter) to sewage contained in suspension in the water. Probably dissolved products of animal origin may have the same effect, but this does not appear to have been proved. Dissolved sewer gases, however, are certainly known to be capable of causing diarrhœa. A striking instance occurred in Salford Gaol in 1859. Within four days 266 out of 466 prisoners were attacked with the complaint, whereas none of the officers nor any members of their families suffered. The water which the prisoners drank was at once examined, and found to have a yellowish colour and an unpleasant taste. The cause of this was that the overflow pipe from the cistern led directly into a sewer, and conveyed a most foul stench to the cistern, which was covered in closely with boards. The water supplied to the officers, on the other hand, was clear and refreshing. Both waters came from the same source, being merely stored in separate cisterns.

It is well known that in late summer and early autumn diarrhœa is very apt to prevail *epidemically*, particularly in large manufacturing towns. In 1859 Dr Greenhow was investigating the causes of this form of the complaint. The general conclusion at which he arrived was that in those places in which it prevailed most severely, some local cause could always be traced; either the air was tainted with the products of organic decomposition, especially of ordure, or the water which the people habitually drank was impure. And the outbreak at Salford, which occurred in the end of September, was naturally regarded as a crucial instance, proving that such conditions are really the cause of epidemic diarrhœa.

Subsequent inquiries, however, have tended to throw doubt upon the validity of this explanation of epidemic diarrhœa in general. Dr Greenhow himself observes that as a fatal disease it is almost wholly confined to children under five years. This might, indeed, be attributed to the greater susceptibility of the young, particularly as there is as yet no evidence to show in what proportion attacks of non-fatal diarrhœa occur in older subjects. But it further appears that most of the cases of fatal diarrhœa are

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in children under one year of age. Dr Crane, of Leicester, investigated the conditions under which 283 children had been placed who died from this cause in the summer of 1873, with the result that a large majority lived in houses not in bad sanitary condition, and that 107 were wholly suckled, 98 partially suckled, and 78 fed by the bottle alone. Impurity of drinking-water clearly was not the cause of the disease in the 107.

Dr Buchanan, while not denying that summer diarrhœa has associations with filth, is evidently inclined to think that it is really due to a specific poison developed by heat at a particular season. He insists that whatever may be the heat of the weather before July, it does not cause epidemic diarrhœa. Yet the connection of the disease with autumnal heat is certain, for the mortality from this cause is much greater in hot than in cool seasons. A curious circumstance, to which Dr Buchanan draws attention, is that the disease seems to be of modern introduction, so far as can be learnt from the old bills of mortality. At the beginning of the present century there is no direct mention of it, under whatever name, nor can any special mortality among infants be traced as having occurred in the summer or autumn months. But for many years past epidemic diarrhœa has been a very fatal disease, sweeping away thousands of children annually.

Another form is that which occurs as a complication of acute diseases, particularly of puerperal fever.

The diarrhœa set up by the causes hitherto mentioned is an acute disease; one that may run its course in a few hours, and that perhaps never lasts more than a week or ten days—if we except those cases in which the complaint may be kept up by the repeated ingestion of irritating food or bad water.

The *anatomical changes* discovered when acute diarrhœa proves fatal are generally very slight. The inner surface of the small intestine may be reddened and lined with mucus, or softened. But, as in the case of other mucous membranes, vascular injection probably often disappears after death, so as not to be seen at an autopsy, though it existed during life; and there are great doubts whether softening, on which French pathologists formerly laid great stress, is not a cadaveric change.

Sometimes, however, the mucous membrane of the small intestine is attacked with a much more marked and severe form of inflammation. A *pseudo-diphtheritic* state, especially marked in the valvulæ conniventes, may be found throughout a large part of the jejunum. In all such cases relaxation of the bowels is a principal symptom, and it is doubtful whether they can be distinguished at the bedside from the more severe instances of ordinary diarrhœa, although the pathologist is naturally disposed to place them in a separate category.

Chronic diarrhœa.—Another and a very different kind of diarrhœa is that which runs a chronic course, lasting for months or even for years. The intestine then often presents appearances which are regarded as indications of chronic catarrhal inflammation. Thus slaty or black patches may be seen in its mucous surface, or black dots and rings corresponding with the solitary follicles. Its coats may be thickened, and it may be lined with a viscid opaque mucus. These changes are particularly well marked when they are secondary to mechanical congestion, as in cases of disease of the heart, or hepatic cirrhosis, either of which may be complicated with diarrhœa.

Sometimes, however, one may be unable to detect any definite change in

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the intestine although the diarrhœa had been present for a long time before death. The author once made a *post-mortem* examination in the case of a gentleman who had come home from China with what is termed "white flux," in which there is constant diarrhœa with discharge of matters devoid of bile. At the time of his death the complaint had lasted some years, and the only morbid appearance discovered was extreme thinning of the intestinal tunics.

In the bowels of infants, with whom chronic diarrhœa is a frequent cause of death, one seldom finds any pathological change. Dr Eustace Smith mentions that ulcers are sometimes present in the large intestine; but, as he says, these are probably secondary, and the result of the irritation set up by acrid matters which had been secreted by the bowel higher up.

Tubercular ulcers.—Diarrhœa is a principal symptom of ulceration of the bowels. The symptoms and the intestinal lesions which accompany enteric fever have been fully described in the first volume (p. 154).

Equally definite and important is diarrhœa from *tuberculous* ulceration. The earliest stage in which this can be recognised is that in which opaque yellowish spots are seen in or beneath the mucous membrane. They doubtless are the result of caseation of the tissue of solitary follicles, or of the follicles of a Peyer's patch, the lymphoid tissue of which had before undergone augmentation.* The next step is that the mucous membrane covering the little yellow spots breaks through, and a small circular ulcer is formed. This almost at once acquires a smooth rounded edge, which is indurated, so that to the finger it feels almost like a rim of leather. The increase in size of the ulcer always takes place chiefly in a direction transverse to the axis of the bowel. Thus its form becomes elliptical, or roughly oblong, and it may become so broad as completely to encircle the bowel. Its floor is generally formed by the muscular coat, which is thickened by inflammatory products, and may still have some yellow cheesy granules adherent to its surface. The subserous tissue and serous membrane also become thickened and opaque, and these changes, and the presence of an injected zone of blood-vessels round the ulcer, enable its seat to be clearly recognised on the outer surface of the intestine. A more important character still is the presence, in many cases, of distinct tuberculous granulations in clusters, or forming long ridges, which are believed to correspond with the sheaths of lymphatic vessels, or (according to Rindfleisch) with the smaller arteries.

Tuberculous ulcers are more common in the lower part of the ileum than in any other part of the intestine; they are often very numerous, and just above the ileo-cæcal valve they may form extensive patches of very irregular shapes. Sometimes, however, only one or two are present, and they may be confined to the upper part of the ileum, or even to the jejunum; or those which occur there have much more marked characters than any which can be found lower down. Again, they may be seen only in the cæcum or the colon; these portions of the bowel are very liable to be affected in common with the ileum. Tuberculous ulcers of the intestine

* In all probability the augmentation arises by a formative process. No doubt it might be due to simple inflammation. But after careful investigation of the question the result to which the author has been led is, that in the *post-mortem* room one seldom or never finds caseation of solitary follicles in the intestine without tuberculous lesions being present in other parts.—C. H. F.

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are probably never seen in the *post-mortem* room without the lungs being likewise affected with active tuberculous disease. And their clinical importance is generally altogether subordinate to that of the pulmonary phthisis, to which the patient succumbs. They do, indeed, afford an explanation of diarrhœa when it is present; but in many cases this and all other symptoms of intestinal lesions are wanting, so that the autopsy alone reveals the fact that such ulcers have been forming. Sometimes, however, before any symptom or auscultatory sign of phthisis is discoverable the patient suffers for a very long period from diarrhœa, and this is ultimately proved to have been due to a tuberculous affection of the intestine. Trousseau quotes Chomel as having especially insisted on the importance of fever and night-sweats as indications of the presence of such an affection.

It is said that tuberculous ulcers sometimes heal, and that their cicatrices may produce stricture of the bowel; this will come under consideration in the next chapter. Acute peritonitis has been caused by ulcers of this kind giving way into the serous cavity. More commonly the affected coil of intestine becomes adherent to a neighbouring coil, and an opening forms between them; in this way a series of communications between one part of the bowel and another may be formed.

Lardaceous disease.—Another cause of chronic diarrhœa is the presence of lardaceous change in the intestinal mucous membrane. This seems never to occur without other organs being affected, and in a marked degree. It may be caused either by syphilis or by protracted suppuration, of which one instance is that which accompanies chronic pulmonary phthisis. It may be worthy of note, so far as concerns syphilis, that Trousseau lays stress on it as an occasional cause of chronic diarrhœa; but in the particular case to which he refers the affection can hardly have been lardaceous, for the symptoms yielded to mercurial treatment. The peculiar change is said by Dr Moxon to begin in the walls of the minute arteries, and to spread from them into the tissues around. To the naked eye the mucous membrane presents an appearance which one can more easily recognise than describe; Dr Moxon compares it to wet wash-leather. Iodine stains it of the colour of dark walnut wood. Peyer's patches are generally less affected than the rest of the mucous membrane.

Malignant growths.—Yet another cause of chronic diarrhœa—comparatively a rare one—is the development of a new growth in the intestinal walls. A carcinomatous ulcer sometimes gives rise to this symptom, but very seldom, for such ulcers, as a rule, narrow the bowel and cause obstruction instead. But it is important to remember that cancer of the colon or rectum may produce, not obstruction, but diarrhœa, or obstruction with diarrhœa.

Cancer of the small intestine is one of the rarest diseases. In a recent case reported by Dr W. H. Ransom in the 'Lancet' (November, 1890) the symptoms during life were those of chronic intractable diarrhœa.

More frequently diarrhœa is set up by a form of lympho-sarcoma, the distinctive characters of which were pointed out by Dr Moxon. It may invade a large extent of the intestine, and completely surround it at various points, but always with the effect of making it wider than natural. It constitutes a white, soft, medullary growth, and has little or no tendency to ulcerate. A marked instance of this affection occurred in a child who died under the author's care in the Evelina Hospital; the growth everywhere seemed to have entered the coats of the intestine along the line of its attachment to the mesentery.

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Symptoms.—The discharges may consist of fluid fæcal matter like that which the small intestine normally contains at a certain period after digestion of food. It may be of a bright yellow colour, or more or less brown. In infants the evacuations of diarrhœa are often green. This was formerly supposed to be a result of the administration of calomel, but it is now known to be due to changes in the bile-pigment independent of any medicine. We shall hereafter find that bilirubin (as it is called) is apt to turn green when acted on by an alkali; and the statement has been made that green diarrhœal matters are always alkaline, but, according to Kühne, this is a mistake. In adults the evacuations often look as if they consisted of pure bile, and are said to respond to the tests for the biliary constituents much better than ordinary fæces. In other cases diarrhœal discharges are pale and watery, and they may even approach in character the "rice-water stools" of cholera. Under the microscope crystals of triple phosphate can often be detected in the matters voided from the bowels in all forms of diarrhœa. Mucus is sometimes present in considerable quantity, but pus can seldom be identified, either with the naked eye or microscopically. Hæmorrhage forms no part of mere diarrhœa, and blood can seldom or never be detected, even when there is extensive tuberculous ulceration.

Acute diarrhœa is generally accompanied by some colicky pain, by sickness, and by slight tumefaction of the abdomen. In chronic diarrhœa these symptoms are commonly absent; the abdominal walls often become deeply sunken and retracted. In infants prolapse of the rectum is very apt to occur as a complication, and the anus generally becomes sore and excoriated.

Mild cases, in adults, are attended with little or no disturbance of the general health. But in infants even very slight diarrhœa may give rise to great depression of strength, indicated by coldness of the surface, by dark pigmentation and sinking in of the spaces round the eyes, and by depression of the fontanelles. This last is a most valuable sign, and must always be borne in remembrance; it often gives a warning of danger at a comparatively early period, when the child would otherwise seem to have but little the matter with it, and when neither the pulse nor the respiration is accelerated. As already mentioned, in infants the disease is very apt to terminate fatally. Death may also occur in old people and even in adults, if exhausted by previous disease or privation; the symptoms are then always those of collapse. Such cases, in which the evacuations are generally profuse and watery, commonly receive the designation of "choleraic diarrhœa" or of "English cholera," and these names regularly appear in the returns of the Registrar-General every autumn. The question whether English cholera ever proves fatal to a healthy grown-up person bears on the relation of this form of diarrhœa to Asiatic or epidemic cholera (vol. i, p. 242).

The treatment of diarrhœa is a matter which requires judgment on the part of the practitioner. Detailed rules can hardly be laid down, but some general principles may be stated.

In the slighter forms of diarrhœa it is sufficient to see that the patient is *warm*, particularly his belly and his feet; to make him lie down and keep perfectly still, and to give him arrowroot. A flannel binder or a hot bran-poultice is often comforting.

Next to warmth rest is essential; a patient who has been tormented by

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loose evacuations and griping while going about finds rest and comfort by lying in bed. Thirdly, the food should be much restricted, and only bland farinaceous diet eaten.

In many cases the diarrhœa is the result of irritating food, and then a dose of castor oil is the appropriate remedy, just as an emetic may be the best cure for vomiting.

In acute diarrhœa it is sometimes desirable to administer a dose of castor oil with a little opium; or, what is perhaps better, a scruple of Gregory's powder, or some other preparation of rhubarb. But, as a rule, the contents of the bowels are being swept away by the diarrhœa itself. The best medicine is then a stomachic with a little alkali. A formula which is widely used consists of a scruple of carbonate of soda, twenty minims of aromatic spirits of ammonia, and an ounce of peppermint water; this may be repeated every two or three hours. Another valuable remedy is the subnitrate of bismuth.

It is not advisable to prescribe opium, or even morphia, in acute diarrhœa, at least until other remedies have had a fair trial. Nor should astringents be given at the commencement of the attack. On the other hand, in chronic diarrhœa, astringents are often very valuable, and may suffice of themselves to cure the patient. Hæmatoxylum, krameria, kino, catechu; the extract of Indian baël; the compound chalk powder and the aromatic confection; alum, pernitrate of iron, nitrate of silver, and sulphate of copper—each of these may be used. Many medical men employ sulphuric acid largely, particularly in children; others believe it to be useless.

Sometimes, however, each astringent seems to lose its effect after the patient has been taking it for a few days, and one is often obliged to prescribe many in turn. This is particularly apt to occur when there is tubercular ulceration or lardaceous disease of the intestines; but diarrhœa may last for a very long time without there being any evidence of organic change in the bowel. In some cases of this kind opium is very serviceable; and it may be continued in free doses for several months without appearing to affect the patient injuriously in any way. For cases of chronic intestinal catarrh, Trousseau speaks highly of the arseniate of soda, and one is prepared to believe that this may be useful from what we know of the influence of arsenic in other catarrhal affections.

A method of treating diarrhœa, which in infants often succeeds, consists in giving no food whatever except raw meat, finely grated into a pulp and mixed with powdered sugar or currant jelly to make it palatable. Trousseau calls this "conserve de Damas" for the sake of mystification. He relates the case of a young lady who had had intractable diarrhœa for six months, and who was quickly cured by raw meat.

Enteritis.—This term is sometimes applied to inflammation of the bowels generally, sometimes to inflammation of the small intestine as distinguished from *colitis*, or inflammation of the large intestine. French writers use the terms *gastro-entérite* and *muco-entérite* to denote catarrhal gastro-enteritis or enteritis; but the terms are scarcely needed, for acute gastritis or gastro-enteritis is better described clinically as acute dyspepsia or acute diarrhœa, and chronic enteritis as chronic diarrhœa.

Severe enteritis occurs as a complication of mechanical obstruction of the small intestine, as in invagination and strangulation and volvulus, and

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will be referred to in the following chapter. The affection in cholera is probably not true inflammation.

Ulcerative enteritis has already been described when due to typhoid fever and to tubercle. Otherwise the small intestines are very little liable to serious primary diseases, and the jejunum particularly is remarkably exempt. Colic, constipation, and diarrhœa affect the large more than the small intestine. In dysentery the lower part of the ileum is sometimes affected, but only by the disease spreading from the rectum and colon.

TYPHLITIS.*—There are three parts of the intestinal canal in which the contents are apt to accumulate; the caput cæcum coli, the sigmoid flexure, and the rectum. All three are somewhat dilated, and in the cæcum and sigmoid there is a sudden change of direction which favours delay. Moreover there is the appendix cæci, which, like many other vestigial organs, is a frequent source of injury. No wonder, therefore, that inflammation of the cæcum, or typhilitis, is a common and important disease.

Some writers distinguish *typhlitis* and *perityphlitis* as two distinct affections. The former, they say, is an inflammation of the connective tissue behind the cæcum, which runs a chronic course, and seldom destroys life, except as the result of protracted suppuration. The latter includes the rapidly fatal cases of perforation of the appendix vermiformis, which some pathologists describe separately under the barbarous name of *appendicitis*.

Dr Wilks, however, has repeatedly expressed the opinion that both in typhlitis and in perityphlitis the disease begins in the appendix cæci, and that variations in the intensity of the same morbid process are the real cause of the supposed distinction. The evidence which morbid anatomy affords points strongly in this direction. Dr Theodore Williams has recorded in the 'Pathological Transactions' a case in point. A man who was being treated for pleurisy was attacked with pain in the right iliac fossa and vomiting; the bowels were confined, and a tumour as large as an orange could be felt in the right iliac fossa. Some days elapsed, and all the symptoms were subsiding, when pneumothorax supervened and he died. He had passed several dark and offensive motions containing scybala, and in consequence the swelling had diminished in size until it could scarcely be detected. Thus the case was a perfect example of what would commonly be called perityphlitis, and regarded as the result of the accumulation of fæcal matters in the cæcum; for in the common use of the term "perityphlitis" there is probably no intention to limit it to cases in which the connective tissue behind the bowel is the exact seat of the disease, but rather an unacknowledged feeling that the term has a wider signification than typhlitis, and means that the disease is "about" or "in the neighbourhood of" the cæcum. However this may be, the real nature of Dr Williams's case would not have been cleared up if the patient had not died accidentally from another cause. Then it was found that the cæcum was surrounded by adhesions, but that there was a small collection of purulent matter round the appendix vermiformis. Here Dr Powell found a minute perforation, and outside the aperture lay a small mass of hardened fæcal matter. Two cases which came under Dr Wilks's observation tend to establish the same con-

* Typhlitis, from τυφλόν, *cæcum* (*sc. intestinum*), the blind gut, or *caput coli*. It is perhaps well to remind the reader that the cæcum of human anatomy is a mere dilatation of the colon, found only in man and certain apes. The homologue of the greatly developed true cæcum of many of the lower animals (*e. g.* the horse and rabbit) is the atrophied *appendix cæci*, which only occurs thus reduced in man and the anthropoid apes, and in the wombat.

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clusion. In each of them the patient had a comparatively mild attack and recovered; afterwards he was seized a second time with the disease and died, when a *post-mortem* examination showed that the disease had originated in the cæcal appendix, and that this was perforated. The name typhlitis, therefore, seems preferable to perityphlitis. Some authors, adopting the nomenclature of uterine disorders, use *paratyphlitis* to denote inflammation of the subperitoneal tissue behind the cæcum, perityphlitis for local peritonitis, and typhlitis for inflammation of the mucous membrane.

Origin.—The affection spreads from the mucous to the serous coat of the appendix, either by a process of ulceration or by one of sloughing; in the latter case the blind extremity may be found gangrenous. The cause of the inflammation is generally the presence of a concretion. This may be the size of a pea, or as large as a plum-stone. It sometimes consists of a substance like wax, but usually is composed of hard, dry fæcal matter mixed with mucus, and containing a large proportion of earthy salts. A mass of this kind may look very like the stone of a cherry or some other fruit, and has often been mistaken for such a substance. Indeed, supposed "foreign bodies" from the appendix have so frequently been found on examination to be of fæcal origin that many pathologists are disposed to doubt whether typhlitis is ever set up by such a thing as a fruit-stone. It is, however, certain that seeds, pills, bristles, pins, pieces of bone, and shot have all been found in the appendix, and that some at least have led to its perforation.

In certain cases ulceration of this part of the bowel, penetrating its serous coat, has been tuberculous; and in yet other cases no exciting cause for the inflammation can be discovered. It is possible that the walls of the appendix may have given way as the result of its distension with fluid, for such a condition is now and then met with, the opening into the cæcum being closed. Dr Wilks saw a case in which the appendix was dilated to the size of the ileum, and distended with three or four ounces of white odourless mucus.

Symptoms and course.—The earliest symptom of typhlitis is usually pain, referred mainly to the right iliac fossa. This is more or less paroxysmal, often of extreme severity; and associated with tenderness, so that sometimes the patient cannot bear even the slightest touch. Nausea and vomiting are generally present, and also constipation. The amount of constitutional disturbance is very variable. The principal local sign is the presence of an ill-defined rounded swelling extending upwards from the iliac region towards the right loin. This is doubtless formed in part by the thickened coats of the affected portion of bowel, but in much greater part it is due to the accumulation of fæcal matters within it. The size and form of this swelling may vary from day to day. If the disease subsides it gradually disappears. The tumour is dull on percussion; but light and careful percussion is necessary to bring this out. The patient often lies with the right thigh flexed, and is unwilling to stretch it down.

The extent to which the inflammatory process spreads over the peritoneal surface varies greatly in different cases of typhlitis. Sometimes it lights up almost instantaneously throughout the whole serous cavity. There is then during the early part of the case no possibility of determining the fact that ulceration or sloughing of the appendix formed the starting-point of the disease; one can only set it down as an instance of acute peritonitis, the cause of which is unknown. Such cases will be again considered further

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on. When they terminate favourably it becomes possible after a time to detect a hard swelling in the right iliac fossa, and this clears up their nature.

In other instances the symptoms of acute peritonitis are wanting, or but little marked. The main clinical feature depends upon the inability of the intestinal contents to pass through the affected part of the bowel. These cases are sometimes undistinguishable at the bedside from those of primary mechanical obstruction, and will be referred to again in the next chapter.

Diagnosis.—The local symptoms, together with the age of the patient, and the way in which the case began, help us in most cases to distinguish typhlitis from ileus; but in cases only seen after peritonitis has developed the diagnosis may be difficult or perhaps impossible. From tubercular peritonitis the diagnosis is usually easy; but here also it is sometimes impossible to make, and what began as typhlitis not infrequently ends as tabes mesenterica.

Cases of typhlitis have been mistaken for enteric fever; in the former diarrhœa may take the place of constipation, and in the latter it is far from rare for diarrhœa to be entirely absent. The rose-rash is often wanting in children, and the course of the temperature will not always be distinctive, or at least not at first.

Lastly, the existence of inflammation or suppuration in the right iliac region may be the result, not of typhlitis, but of ovaritis, of morbus coxæ, of caries of the ilium, or of post-peritoneal abscesses originating in the kidney or the vertebræ.

Thus the cases of typhlitis that can be satisfactorily diagnosed form only a part of the whole, and, as a rule, they are the milder ones—those in which the inflammation does not spread far beyond the cæcum.

Sex and age.—Typhlitis is much more common in males than in females. Out of 10 consecutive cases of Dr Fagge's, 8 occurred in boys or men; out of 31 other patients under the present writer's care, 22 were males. It is a disease of an early period of life; in 7 of the 10 cases the patients were between thirteen and twenty-one years old; in 8 of the 31 cases the patients were between five and twelve, in 19 between fifteen and thirty, and in 4 between thirty-eight and forty-five years old.

Treatment and prognosis.—The course and event of typhlitis depend very much upon the treatment which is adopted. Few cases in which the disease can be diagnosed as seated in the caput cæci terminate fatally if judiciously managed; the more severe cases are those which depend on the presence of ulceration in the appendix, and these sometimes need surgical interference.

The treatment in a doubtful case should be that for typhlitis. If there is none, no worse harm than loss of time will ensue; if there is suppuration of the appendix, a subsequent operation will be rather more than less likely to succeed. The essential points are that the patient be kept entirely in the recumbent posture; that he should be strictly confined to liquid diet, that he should not be allowed to take a single dose of aperient medicine, and that opium should be given freely. The treatment should, in fact, be exactly that of acute peritonitis. When the attack subsides, the greatest possible care must be taken to prevent a relapse. Even then the action of the bowels should be solicited by enemata only, and never by medicines taken into the stomach; and the restriction to fluid food should be continued for several days longer than seems at first sight to be necessary. One repeatedly sees relapses occur from disregard of precautions that seemed

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to have been enforced with sufficient emphasis to ensure their being attended to. The disease, indeed, is one which is very apt to recur, even at considerable intervals of time. In more than one instance repeated attacks have taken place with a few weeks or months between them, until at length there has been one so severe as to place life in imminent danger. The patient has then at last submitted to being kept in bed for a considerable time, and has observed the greatest possible care during convalescence; probably for this reason this alarming attack has often been the last.

When typhlitis ends in suppuration, the pus collects in the loose connective tissue, between the cæcum and fascia iliaca, and forms an external abscess, marked first by œdema and then by fluctuation. Sometimes it passes backwards out of the pelvis through the sciatic foramen, and points below the fold of the buttock; sometimes it passes downwards under Poupart's ligament; it very rarely reaches the rectum, but may open into the bladder, as in the case of a boy under the writer's care some years ago, who discharged the pus *per urethram* and made a good recovery.

When suppuration has occurred the abscess should be opened by Hilton's method and drained. In one case lately in Mary Ward, the pus had been allowed to find a way for itself, a fæcal fistula formed externally, and this ended in lardaceous disease of the viscera.

In a remarkable case under the late Dr Mahomed, Mr Symonds cut down upon the appendix cæci and removed the concretion which had caused the mischief ('Trans. Clin. Soc.,' 1885, p. 285). The result was successful.

This method of treatment has of late years been widely followed, and still more widely advocated in this country and in America.* It has even been advised to cut down upon the appendix, ligature, and remove it in all cases of severe typhlitis. Such practice, however, would in all likelihood much increase the mortality of the disease, and may justly be regarded as meddlesome. When suppuration has occurred and the abscess must be opened, it is probably wise in most cases for the surgeon to explore the seat of disease. And when repeatedly recurrent attacks of typhlitis interfere with a patient's health and prospects, an operation for the removal of the appendix in the interval when there is no active inflammation is in certain cases justified. Such a case in a young man, who for several years had been prevented from earning his living by frequently returning typhlitis, was successfully treated by Mr Lane in August, 1888. On the other hand, the writer has two patients at the present time who have suffered from two or three severe and several slighter attacks, and who, after persevering medical treatment, are now well without the inevitable risks of a serious operation.

Even when typhlitis presents itself clinically under the guise of intestinal obstruction or of diffused acute peritonitis, it seldom destroys life if judiciously treated. There may be cases in which a considerable part of the appendix sloughs away, and in which death is inevitable without, and probably sometimes even with, the most skilful surgical interference; but only one fatal case occurred during five consecutive years at Guy's Hospital, if we exclude those in which death took place a few hours after admission and in which purgatives had been given before. The following is the result of the present writer's personal experience:—Out of 31 cases, seven died, but of these two were exceptional cases—the one being a child who had a foreign body, to wit, a pin in the appendix, and a secondary abscess of the liver;

* See Mr Treves's valuable paper in the 'British Medical Journal' for Nov. 9, 1889.

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and the other a woman in whom typhlitis was complicated by tubercles of the peritoneum and other organs. Of the 24 other cases, one died a year after from tubercular peritonitis, and another was last seen with symptoms of lardaceous disease of the liver and kidneys. In two of the cases which recovered there was general peritonitis with high fever; in five the typhlitis was recurrent a second, third, or fourth time; in only one an abscess was opened, and in another the appendix was removed. There was no operative interference in the five fatal cases, and in three of these there was obvious reason for their ill result.

DYSENTERY.*—This disease, the most severe inflammatory form of diarrhœa or "bowel complaint," is now happily rare in England. It is closely allied to malarial disease in its ætiology and distribution; and, like ague, has been gradually banished from most parts of England, while still one of the most important endemic diseases in India and China. Though most severe in the tropics, it is as well known in warm temperate climates at the present day as in the time of Hippocrates, and is still apt to break out in camps and prisons.

The patient has frequent desire to go to stool, but passes little except mucus or blood; at the same time he suffers from tenesmus, and griping pains in the course of the large intestine.

But the symptoms are not by themselves sufficient to characterise dysentery. We will therefore first describe the morbid appearances which belong to the disease.

Anatomy.—The structural change which characterises dysentery consists in an inflammatory process which has its principal seat in the mucous membrane, and which is not limited to any one part, but spreads more or less extensively through the large intestine. Cases have occurred in Guy's Hospital in which the last few feet of the ileum have been diseased as well as the whole length of the large intestine.

In some instances the whole gut, from the rectum to the cæcum, shows morbid changes of the same kind and in the same stage. But in others the disease is more advanced or more severe in one part than in another. Commonly the rectum is the seat of the most intense changes, and these gradually diminish towards the cæcum; but sometimes the reverse is the case, as was noted by Sydenham. The flexures often suffer more than the intervening parts of the bowel.

The appearances presented by the affected parts in dysentery are exceedingly varied, but most writers are now agreed that the processes concerned in their production may be reduced to two. Virchow designates these respectively "catarrhal" and "diphtheritic." Excellent descriptions of them have been given by Heubner, of Leipzig, and by Woodward, of the American army.

The catarrhal form.†—The mucous membrane at first shows lines and

* *Synonyms.*—Bloody flux.—*Fr.* Dysentérie.—*Germ.* Ruhr, blutige Ruhr. The Greek word is classical: a passage in Herodotus clearly alludes to the dysentery of camps:—(of the army of Xerxes during its retreat from the invasion of Greece) ἐπιλαβὼν δὲ λοιμός τε τὸν στρατὸν καὶ δυσεντερίη κατ' ὁδὸν διέφθειρε (lib. viii, cap. 115); and the term is common in Hippocrates, who correctly refers it to ulceration of the intestine. It is also used by St Luke, himself a physician, in its natural connection with intermittent fever: πυρετοῖς καὶ δυσεντερίᾳ, συνεχόμενον (Acts xxviii, 8).

† *Synonyms.*—White dysentery.—*Germ.* Katarrhalische Ruhr—ῥεῦμα γαστρός (Galen).—Intestinorum rheumatismus (Cœ. Aurel.)—Coryza ventris—Tormina—Dysenteria fiens (Sennert), a translation of Galen's Δυσεντερία γινομένη.

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patches of a dark red colour, with points which are almost black. The summits of any ridges or folds projecting into the interior of the bowel are more injected than other parts. The mucous membrane is lined with a rather thick layer of mucus streaked with blood, and is much swollen, as well as the submucous tissue. In the earliest stage of the disease, all that the microscope reveals is a dilatation of the minute blood-vessels, which are gorged with blood. Soon, however, inflammatory products are poured out. The mucous membrane becomes now still more œdematous and bulky; it is less uniformly reddened; the solitary follicles are enlarged, and appear as white points with red rings round them. The submucous tissue is increased from three to five times in thickness; and even the muscular coat is swollen.

Under the microscope all the tissues are now seen to be infiltrated with pus-cells, which are also present in large numbers in the mucus lining the interior of the intestine. In the submucous tissue the corpuscles occur chiefly in the spaces round the blood-vessels. The solitary follicles are markedly increased in size; the lymph sinus which surrounds each of them is wide, but does not contain pus-cells; leucocytes, however, are collected in large numbers under the thin mucous membrane which covers the follicles, and which evidently was about to give way and rupture.

In former days there was great discussion as to the importance of these changes in the solitary follicles, in relation to those in the rest of the mucous membrane. In 1843–4 Dr Parkes examimed the intestine very carefully in numerous fatal cases of dysentery, and stated that the earliest lesion was the alteration in the follicles; and Dr Baly arrived at the same conclusion from his investigations at Millbank Prison. Before this Cruveilhier had insisted that the solitary follicles had no share in it. Since the publication of Dr Parkes's observations, the objectors to his views have taken up the ground that the white granules visible in the early stage of dysentery are not really solitary follicles, but new formations. The right opinion, however, appears to be that which has been stated in the previous paragraph, namely, that although the solitary glands are undoubtedly enlarged in the early stage of the catarrhal form of dysentery, this is only part of a general process of inflammation which affects the whole mucous membrane.

After a time the mucosa softens down with the increasing infiltration of pus-cells, and ulcers are formed. A peculiar appearance is now produced by the changes round the solitary follicles. Their roofs give way, and minute round holes are produced, each of which leads into a small cavity having in its interior the substance of the follicle, which is isolated from its attachments, and in fact forms a small slough. The destruction of the mucous membrane, however, is by no means confined to those parts which surround the follicles. It also takes place between them, so that for a time each orifice is surrounded by a little ring, which appears to be raised, and looks like a deposit upon the surface instead of being a remnant of the original tissue. Even when the ulcers have increased in size, and run together so as to form large patches, there remain irregular islands of still undestroyed mucous membrane, which are of a bluish-red colour and covered with grey or greenish layers of tough mucus. The ulcerated surfaces themselves have a yellow or yellowish-red colour; their floor is formed by the submucous tissue.

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*The sloughing or necrotic form.**—Widely different are the appearances in the other form of dysentery, which, in obedience to the teaching of Virchow, all modern German writers call *diphtheritic.* To digress for a moment, this term diphtheritic has now two meanings, which must be carefully distinguished from one another. In the first volume *diphtheria* was described as a specific disease, generally affecting the throat. But in Virchow's sense "diphtheritic" inflammation of a mucous membrane or of the skin need not have anything to do with that disease. It does not imply the presence of a false membrane, but a putrid, destructive, more or less necrotic inflammation. See the exposition of this point in the chapter on Inflammation (vol. i, p. 52), and in that on Diphtheria (vol. i, p. 257).

In this form of dysentery the whole thickness of the intestine is from the first affected in a marked degree. Even the serous surface is intensely injected, so that it is of a dark bluish-red colour. The bowel feels hard as well as massive. In its interior there is a thin reddish fluid, or, in some parts, a little fæcal matter. Its lining is of a greyish-red colour, and here and there exhibits what looks like a raised deposit on its surface. This, in its earliest stage, is present only on the summits of the ridges of the mucous membrane. It may be seen forming transverse lines in the ileum, the lower part of which is commonly affected in this form of the disease. In the cæcum, the seeming deposit becomes more extensive; and in the colon it occurs in large patches, or may occupy the whole surface; being, however, broken up into plates by deep grooves or fissures. The parts affected in this way look dry and granular; to the touch they feel rough and hard. Their colour varies to some extent with that of the intestinal contents, which possess the power of staining them. Thus they may be yellowish, greenish, dark red, or even black. On making a section through the intestine one finds that it is enormously thickened; the muscular layer is much thicker than natural, and folded in and out. But the most striking change is in the internal coat. Instead of the dry rough substance above described being a deposit on the surface of the mucous membrane, it is now seen to take the place of that structure, and perhaps of the submucous tissue also. The whole thickness of the intestinal wall within the muscular coat may thus be made up of a tough, homogeneous, yellowish-red material, which offers considerable resistance to the knife, and in which the natural strata can no longer be recognised. Even under the microscope one can hardly make out the original elements of the tissue. One sees nothing but a mass of extravasated blood, of hard amorphous fibrinous exudation, and of pus-cells in greater or less number. But Heubner states that, in very thin sections, lines of epithelium arranged in double rows may be identified as the remains of Lieberkühn's tubules.

According to recent observers, however, the fibrinous material described as amorphous contains elements of great importance. It is highly granular, and these granules appear, with the aid of high powers, good illumination, and appropriate staining, to be really bacteria.

It is evident that the apparent "deposit" or "membrane" in the diphtheritic form of dysentery is really formed by the exudation of fibrin and the extravasation of blood into the tissues themselves, not upon the surface of the mucous membrane. The mucosa as well as the epithelium is

* *Synonyms.*—Red dysentery.—*Germ.* Diphtheritische Ruhr.—Dysenteria facta (Sennert), a translation of Δυσεντερία ἤδη γεγενημένη.

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itself destroyed.* This process can have but one termination—the death of the affected structures. Accordingly, whenever there has been time for the occurrence of further changes, eschars are found; and at a still later period these break down into shreds or detritus and are cast off, exposing deep and ragged ulcers of dark green or brown colour.

For detailed information of the morbid anatomy of dysentery, illustrated by photographs and coloured plates, the reader is referred to the valuable monograph on the diarrhœa and dysentery observed during the war of the Rebellion in 1861—1866, by Surgeon-Major Woodward, U.S.A. Among early accounts of autopsies may be mentioned one by Hewson, and two others (likewise communicated to Sir Geo. Baker) by Dr Wollaston.

The changes presented by the intestine in the two forms of dysentery are so different in appearance that one would at first sight be disposed to regard them as belonging to different diseases. It is, however, certain that they merely indicate different degrees of severity in the morbid process. For they are very frequently found side by side in the same intestine, the more intense inflammation being present in those parts (commonly the rectum or cæcum) which were earliest attacked. And this being so, the fact that the solitary follicles appear not to be specially affected in even the earliest stage of the diphtheritic form affords strong corroborative evidence in favour of the view that these glands are not the seat of any primary or special change in the catarrhal form of dysentery.

Later anatomical course.—In severe cases abscesses also arise in the submucous tissue, and these may burrow, so that undestroyed parts of the mucous membrane over them are detached in the form of bridges, and when pressure is made upon these the pus exudes at several distant spots. The inflammation extends at one or more points through the muscular coat, and perhaps penetrates to the serous membrane, so that perforation of the bowel occurs, with consequent peritonitis; or the connective tissue at the back of the bowel may be reached by the ulcerative process, the result being that a fæcal abscess is formed. In one case at Guy's Hospital such an abscess formed a large tumour filling the left side of the abdomen, extending into the psoas muscle and the spleen (which was sloughing), and denuding the ilium of its periosteum over a considerable space.

It is probable that the most intense form of dysentery, in which the whole large intestine and the last few feet of the ileum are uniformly affected, is necessarily a fatal disease. But if the morbid changes in the bowel be not too extensive and severe, recovery may take place, even though in some parts they may have assumed a necrotic character. In the catarrhal form, the inflammation probably often subsides before any breach of surface has occurred. When ulceration takes place, and the ulcers subsequently heal, a thin membrane is formed over their surface which is at first depressed below the level of the parts that had been unaffected; but this difference gradually becomes less obvious, and ultimately disappears. In the "diphtheritic" form of dysentery the ulcers left by the separation of the sloughs become covered with granulations; their undermined edges adhere to the submucous tissue, and thickened and irregular cicatrices gradually develop themselves. The cicatrices which follow dysentery are always of a dark grey or black colour, which probably results from a chemical reaction between the colouring matter of

* "Muco penitus absterso, nulla membrana superficiem quæ tegeret inventa est" (Hewson).

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blood extravasated during the course of the disease, and the sulphuretted hydrogen which is one of the gases contained in the interior of the bowel.

In many cases, however, there is no definite termination of the morbid process, either in the death of the patient or in his recovery. The disease passes into a chronic form, and may continue for months or even years. The ulcers remain unhealed, or fresh ones form in succession as others cicatrise over. But, as Dr Maclean points out, it is a mistake to suppose that ulcers must remain unhealed so long as symptoms of dysentery in the chronic form persist. Cases are often observed in which not a single breach of surface is discoverable after death. Numerous black cicatrices may be seen, but the essential pathological change is an atrophy of the coats of the bowel, the glandular structures having disappeared, and the wall being so attenuated as to be transparent.

Sporadic, endemic, and epidemic forms.—Probably there is no part of the world in which dysentery does not sometimes occur *sporadically*, but in London it is now decidedly a rare disease. Formerly it was common there and in many other parts of England, and has become rare along with ague and other results of malaria. Cardinal Wolsey died of English dysentery. In 1762 it occurred epidemically in London, and was described by Sir George Baker. The account of it by the late Dr Baly, as a local disorder in Millbank Prison, is, we may hope, the last. At present most cases met with by London physicians are imported, and are mostly confined to soldiers and sailors. At the Seamen's Hospital, Greenwich, dysentery may still be studied in its less acute forms.

There are, however, countries in which it is exceedingly prevalent, so that it may be said to be *endemic* there. These are Greece and other parts of the Mediterranean shore, most districts of India and Ceylon, Java, the coast of China, almost all tropical Africa, Madagascar and many islands, and Central America.

Dysentery sometimes affects large numbers of persons as an *epidemic*, and then is apt to assume a severe type. Heubner, indeed, is disposed to think that a primarily "diphtheritic" dysentery never occurs sporadically. But among fourteen or fifteen cases of acute and rapidly fatal dysentery that in the course of the last twenty years have from time to time occurred at Guy's Hospital, there have been several in which the inflammation showed the most marked pseudo-diphtheritic or necrotic character. It is therefore evident that, so far as concerns the anatomical changes in the intestine, no absolute distinction exists between sporadic and epidemic dysentery.

As a rule, however, the disease is much milder when sporadic than when endemic or epidemic.

Ætiology.—The origin of *sporadic dysentery* is commonly attributed to the ingestion of irritating articles of diet, such as unripe fruit, decomposing meat, or bad water. But Trousseau and others have disputed the correctness of this opinion. It has been urged that when anything which disturbs the intestine is swallowed, active peristaltic movements are excited which expel it from the body, and that ingesta are not likely to leave the small intestine unaffected and to exert an irritant action first upon the cæcum and colon. But it is well known that this very thing occurs in cases of poisoning by bichloride of mercury, in which violent inflammation and even ulceration of the cæcum and colon have repeatedly been observed although the small bowel has escaped entirely.

There is some reason for thinking that one factor in the causation of

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dysentery may be an habitual torpidity of the large intestine. This is a point on which Virchow lays stress; he remarks that the cæcum and the flexures of the colon, which are particularly liable to be affected by the disease, are also especially apt to become loaded with fæcal masses. And it is evident that if there is any irritant substance among the intestinal contents its action must be favoured by their retention in the bowel as the result of imperfect peristalsis. Now, Annesley long ago pointed out that in India the disease often commences with the characteristic signs of morbid accumulation in the large bowel. And in connection with this the suggestion of Dr Dickinson may be remembered, that certain transverse ulcers in the colon, which are not uncommonly found in the bodies of those who have died as inmates of London hospitals, and which have been known to perforate the bowel or to cause a fæcal abscess, are due to irritation from the fæces.

Another cause to which sporadic dysentery has been attributed is cold. Heubner alludes to a case which occurred in a washer-woman who had been standing for a long time with the clothes about her body wet through. The cases of catarrhal colitis with hæmorrhage, which are not uncommon in women and children and are commonly regarded as dysentery, can quite as often be traced to exposure to cold as to indiscretions in diet.

As regards the causation of *endemic* and *epidemic dysentery* very little is as yet certainly known. On each side of the equator, to about 35° or 40° of south or north latitude, there are in all parts of the globe territories in which it prevails, but it is a great mistake to suppose that it is endemic in every country with a hot climate. Hirsch mentions Gujerat in India (particularly the peninsula of Kathiawar), and Senegal in Africa, as regions in which the heat is intense, but in which there is no dysentery. So, again, Singapore is said to be free from the disease, which yet exists in all other parts of the peninsula of Malacca. The rainy season is generally the period of the year at which dysentery is most apt to prevail. The alternation of hot days and cold nights has been supposed to have a special influence in producing it.

In temperate climates epidemic dysentery occurs, at the present day, chiefly in camps and armies. It was very fatal in 1854 among the British troops engaged in the Crimean war, as it had been among their forefathers in 1415 before the battle of Agincourt. It raged terribly in the armies of the United States in 1862—1865, and in the camps of the Northern prisoners. It was again prevalent in 1870, during the campaign between France and Germany, particularly among the miserable fugitives who sought refuge in Switzerland after the dispersal of Bourbaki's army.

Even in time of peace the large cities of Europe were formerly liable to epidemic dysentery, and Paris suffered severely from it as lately as 1859, after having been free for a hundred years. In London, in the seventeenth century, it is believed to have caused from 1000 to 4000 deaths annually; in the following century it gradually disappeared, the last general epidemic being that of 1762. In Millbank prison, however, small outbreaks of the disease were of frequent occurrence until a few years ago.

There was lately a severe local epidemic of dysentery in a lunatic asylum in Ireland. At a meeting of the Royal Irish Academy of Medicine (December 2nd, 1887), Dr Conolly Norman reported that of the 1100 inmates 120 were attacked, and 22 died. In two of the latter cases there was perforation of the colon, and in a third multiple abscesses were found in the liver.

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As in the tropics, so in temperate climates, the autumn is the season at which the disease is most apt to break out. The years in which it has prevailed have sometimes been exceptionally hot.

Dysentery occurs in persons of all ages. In this country the sporadic form, or a closely related disorder, is not uncommon in infants who are brought up by hand. When it is epidemic, it attacks especially those who are weak or old, or whose health is impaired by intemperance.

The conditions which are concerned in the production of endemic dysentery have been studied with the utmost care and attention; but, as one might have anticipated, it is difficult to make out which of them is really the exciting cause of the disease.

Certain facts appeared at one time to point to the conclusion that it was a telluric poison, like that which was supposed to generate intermittent fever. Thus it has long been known that even in temperate climates, and still more in the tropics, the countries in which dysentery prevails are also those in which ague is common. Indeed, the two diseases frequently occur together, in the same patient and at the same time; and Dr Aitken remarks that if a boat's crew be sent ashore in a tropical climate, and exposed to paludal miasmata, the probabilities are that of the men returning on board some will be seized with dysentery and others with remittent fever. So, again, the gradual extinction of endemic dysentery within the last two centuries in England has coincided with a very marked decrease in the amount of ague throughout the country, and with its complete disappearance from certain parts. It was, indeed, known that the very same miasm could not be the cause of both diseases, for dysentery prevails in many places where there is no intermittent fever. But, from his investigations at Millbank prison, Dr Baly came to the conclusion that the epidemics of dysentery there were due to a miasm arising from the soil; and Dr Maclean, who endorsed this opinion, attributed the poison to the decomposition of organic matter in the ground.

In the meantime, however, the course of events at Millbank proved that Dr Baly was wrong. In the year 1854 the prisoners ceased to be liable to dysentery; and during the next eighteen years (up to 1872) one death only occurred from that disease or from diarrhœa. Now, as Mr de Renzy showed, one, and one only, change in its hygienic arrangements has coincided in time with this improvement in the sanitary state of the prison. Formerly, the water which the convicts drank was taken directly from the Thames as it ebbed and flowed beneath the walls. But on August 10th, 1854, the artesian well in Trafalgar Square was made the source of water supply to the prison, and has since been exclusively used. The change was effected in the middle of a cholera epidemic; six days afterwards the disease suddenly ceased. Enteric fever, too, no longer attacks the convicts, and the death-rate has declined to an extraordinary extent. It seems impossible to avoid the conclusion that the exciting cause of dysentery in Millbank prison was the Thames water; and in all probability the noxious ingredient was derived from the sewage it contained.

Another series of epidemics, which have been traced to a somewhat similar origin, occurred in the Cumberland and Westmoreland Asylum. In 1864, chiefly between May and August, twenty-six persons were attacked with dysentery, and in March, 1865, five others. For a long time Dr Clouston, the medical superintendent, was completely at a loss to account for the disease. It had often occurred to him that the cause might be connected

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with the distribution of the sewage of the asylum, which, after being thrown into a large tank, was allowed to flow over a field about 300 yards distant; but it was not until August, 1864, that an offensive smell was noticed at the asylum during several hot and sultry evenings. Dr Clouston had the sewage carried away in a covered drain to a distance; from that time no fresh cases of dysentery occurred. An investigation was then made as to the exact meteorological conditions which had existed during the prevalence of the epidemic, and it was found that within a week before the day on which each patient fell ill there had always been either hot sultry evenings with no wind in the night, or northerly winds which blew from the direction of the field which was being irrigated. Male and female patients, too, were attacked at different times, according as the exact direction of the wind was such as to carry the sewage emanations either to one or to the other of the parts of the building which the two sexes severally occupied.

The probability that these observations pointed to the real cause of the dysentery was greatly increased by the fact that the five cases of dysentery in March, 1865, all occurred within a week after the sewage was again allowed to flow over the field, during one night, when the direction of the wind was towards the asylum. The evidence may be said to have amounted to proof in the year 1868. There had then been two years' immunity from dysentery, and, the most approved precautions having been taken, it was determined to run the sewage over another field. Two months later six patients were attacked with dysentery and diarrhœa within a few days of each other; they were all in that part of the asylum nearest the field, and the wind had been blowing towards it continuously for eight days before the outbreak occurred.

In time of war, as well as in tropical epidemics, the conditions are generally so complicated as to defy analysis. Dr Chevers, however, has expressed the belief that much of the dysentery (as well as cholera) occurring on board vessels in the port of Calcutta is caused by men drinking the water taken up in buckets over the ship's side, this water being loaded with sewage and the products of putrefaction. Heubner was told by several military surgeons that where many severe cases of dysentery were crowded together the disease was often spread by the latrines, and ceased when the proper precautions were taken with regard to them. Prof. Maclean says that in India the barrack-rooms most exposed to the effluvia from latrines always furnish the largest number of dysenteric cases.

We must believe, then, that the cause of dysentery may be, and probably always is, the entrance into the body of some organic matter conveyed either by drinking-water or through the air. So far dysentery resembles enteric fever and cholera.

Specific contagion.—The next question is whether, as in these diseases, the organic matter is a chemical product of decomposition, or a living organism, which undergoes a process of self-multiplication in the human body, so that the discharges possess special powers of infecting others with dysentery. This question cannot yet be finally answered. Repeated attempts to discover a pathogenic microphyte (or microzoon) have been made by Prior, Klebs, and other pathologists, but without success. The contagion is believed to be in the fæcal discharges, and to be taken in by water.

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All observers are agreed that the disease seldom or never passes from the sick to those who are attending on them. And Heubner lays stress on the fact that it has hardly ever been known to spread from the military to the civil population in time of war.

In 'Ziemssen's Cyclopædia' and 'Eichhorst's Handbuch,' however, dysentery is placed among the infective diseases, and Liebermeister regards it as belonging to the same class of miasmatic-contagious maladies as cholera and enteric fever. He goes on to admit that it is a local disease, and that all its symptoms are dependent upon the intestinal inflammation; but, as he remarks, gonorrhœa and the soft chancre are *local*, and yet *infective* in the fullest sense of the term.

Epidemic and sporadic dysentery cannot be separated by any strict boundary-line; but it has been urged that between these two forms of the disease the same relation exists as between Asiatic and English cholera; and since the occurrence of a simple variety of cholera does not prevent the acceptance of the view that, when introduced from the East, this malady is due to a specific contagion, it is argued that the existence of sporadic dysentery is no bar to the infective theory of epidemic dysentery.

Evidence that dysentery is, at least in its severe form, a specific and infective disease has been afforded by an epidemic which occurred in Norway in 1859. Dysentery had not prevailed there for half a century, and as the population was scattered, and the ground very broken, there were unusual opportunities of tracking its course. The disease was studied with great care by Homan and Hertwig, and they believe that they established the fact of its spreading by a definite contagion, which was even conveyed by healthy persons from infected places to other spots where the disease had not before appeared. It does not seem that they traced the poison to any particular source, but the analogies of enteric fever and cholera evidently render it in the highest degree probable that, if there really is a contagion which multiplies itself in the human body, this reaches the outer world in the alvine evacuations.

With regard to the period of *incubation* in dysentery, Dr Clouston states that one of his patients fell ill within three days after first inhaling the poison; but others were attacked as long as three, four, or five days after the cessation of opportunities for its entrance into the body. Homan and Hertwig state that in Norway in 1859 the period of incubation was from two to eleven days.

Symptoms.—Before the characteristic features of dysentery develop themselves, there is generally a period in which the patient suffers from simple diarrhœa, with more or less griping pain in the abdomen. This may last from three to five days, or even a fortnight. He then becomes worse, and perhaps he shivers, or at least complains of greatly increased malaise and weakness. The abdominal pain is more severe and paroxysmal. The desire to go to stool becomes more and more frequent, and, instead of abundant liquid fæces, he begins to pass only very small quantities at a time, with violent straining, and burning pain in the rectum. He is then said to suffer from "tenesmus," but although this term is universally employed there are hardly two writers who are agreed as to its exact signification. Some mean by it the peculiar sensation that there is "something in the rectum that wants to come away," others the straining, and others the burning pain; while others again include two or all in the connotation of the word. But as all these symptoms are constantly associated together it is really not a

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matter of importance whether tenesmus means one or another of them.* It is taken by Dr Woodward as the most characteristic of the symptoms of dysentery. So incessant may be the call for relief that the patient goes to stool twenty or thirty or even as many as two hundred times in the twenty-four hours. Or he may sit there for half an hour at a time, straining violently, but passing little or nothing. In a number of cases of dysentery occurring in camp, Heubner had the total daily evacuations collected. The quantity passed by each patient was found to be only from 28 to 42 oz.

The evacuations in dysentery often contain no fæcal matter whatever. Small hard scybalous masses may be voided occasionally, but the most careful recent observers are agreed that these are comparatively seldom seen. Thus the paradoxical expression of Stoll is justified, who described dysentery as *morbus alvum occludens.* But in some cases fluid fæcal matter is passed from time to time throughout the whole course of the disease, and this is said to be rather an unfavourable sign, as showing that the upper part of the large intestine, or even the small intestine, is involved. In most cases, however, the only result of the straining efforts of the patient is that he passes two or three drachms of gelatinous mucus, colourless, or more or less deeply stained with blood. Or there may be membranous shreds of mucus, and small clots, mixed with more or less of the fluid fæcal matter. Or, again, pure blood may come; or a thin reddish fluid, which has floating in it a number of little yellow or red masses, soft, and looking like pieces of sodden meat. This kind of discharge is often described as resembling the washings of meat, and was formerly called *lotura carnea.* The masses suspended in it were supposed to be cast-off portions of the intestinal mucous membrane, but it is now known that this is not the case. Dr Goodeve, in Calcutta, is, indeed, said to have found membranous sloughs from half to one inch or more in diameter, in the sediment obtained by washing the evacuations after the eighth to twelfth day of the disease. But, according to Heubner, it very rarely happens that masses are passed the nature of which can be recognised. Another dysenteric product is pus, which may occasionally be discharged pure and odourless, and just as if it had come from an ordinary abscess, but which is much more commonly mixed with fluid fæcal matter and blood. In some cases, again, a substance is voided which looks like frog's spawn or boiled sago. This consists of rounded bodies which were once thought to be sloughs derived from the solitary follicles. Heubner, however, says that they are too large for this, and that they consist of mucus. Lastly, the discharge may be a brownish-red or blackish fluid, of a most horribly offensive odour; this is an indication that sloughing is going on in the intestine. It is to be noted that in all but the earliest stages of dysentery the matters passed from the bowel have a fœtid odour, which Parkes regards as peculiar to the disease.

The abdomen is not at first distended, but it may become so as the disease advances. Tenderness may be altogether absent, or pressure over different parts of the large intestine may give more or less pain. In some cases one can obscurely feel an induration in the course of the bowel, and it has even been suggested that the extent to which the upper part of the colon is affected may be determined in this way.

* Tenesmus (τεινεσμός, from τείνω): "Est autem affectus hic continua et implacabilis desidendi cupiditas (Paulus Ægineta apud Stephanum)—Quod τεινεσμόν Græci vocant: in hoc frequens desidendi cupiditas est, æque dolor ubi aliquid excernitur" (Cels., lib. iv, cap. 18).

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Another symptom which is sometimes present in severe dysentery is strangury. It may be necessary to use a catheter.

The most marked general symptom is the great prostration. The patient not uncommonly faints while he is at stool. Anæmia is very rapidly produced, and the face assumes a pale, waxy look.

The febrile disturbance is generally slight. Even in severe cases the temperature may be normal or below normal, but sometimes it rises to 100° or 101°. The pulse is not much quickened, except in certain epidemics formerly characterised as "inflammatory dysentery." The patient often makes great complaint of thirst, and although his appetite is not always lost, griping pain is frequently brought on by food, so that he is unwilling to take any but the blandest nourishment. Cold liquids frequently have the same effect, and therefore it is well that what the patient drinks should be lukewarm.

Diagnosis.—A question of great importance is whether dysentery is always attended by the striking train of symptoms which have just been described. Writers mention that some patients pass no blood, and it is said that certain epidemics are characterised by the absence of this symptom. Again, it is well known that tenesmus and the other symptoms which are constantly associated with it depend upon disease of the lower part of the large intestine, and that they are absent when the inflammation is limited to the cæcum and the upper part of the colon. Dr Clouston says that in the epidemic which he observed at the asylum near Carlisle, some patients experienced scarcely any pain throughout the whole course of the disease, and at first, having no fever or want of appetite, they refused to believe that they were ill, although they were passing glairy mucus mixed with blood. It does not appear that tropical dysentery is ever latent when the disease is epidemic. We have, however, had at least two well-marked instances of fatal sporadic dysentery, in each of which there was very extensive diffused ulceration of the large intestine, the presence of which had been entirely unsuspected during life. Both patients died in Guy's Hospital, the one of an enormous hepatic abscess, the other of a large abscess in the left iliac fossa, resulting from perforation of the bowel into the post-peritoneal connective tissue. Dr Dickinson has recorded a similar case, that of a woman who died in St George's Hospital of abscess of the liver, and who, during the week which she passed as an inmate of the hospital, was so constipated as to require aperient medicines. She would not allow that she had ever had any looseness of the bowels, but after death the upper part of the large intestine was in a state of ragged ulceration.

In at least three cases of acute dysentery that have proved fatal in Guy's Hospital the disease was supposed during life to be *enteric fever;* the characteristic symptoms were either absent or passed under the name of diarrhœa. It is worthy of inquiry whether a similar latency or obscurity of symptoms may not sometimes occur in epidemic dysentery, both in India and in temperate climates. We shall hereafter see that the question is one of considerable interest in reference to the causation of abscess of the liver.

Even when all the symptoms of dysentery are present, the diagnosis should be made with more caution than would appear necessary from the statements of writers who speak of it as presenting no difficulty whatever.

In young persons, and particularly in male children, one must always bear in mind that *intussusception* causes very similar symptoms, although they no doubt begin in a different way. In this country more than one case of

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intussusception has been mistaken for dysentery, and this very serious error must often have been committed in those parts of the world in which the latter disease, being endemic, is likely to have its presence taken for granted.

Again, in persons more advanced in years, it very frequently happens in England that *cancer of the rectum* is overlooked, and that its symptoms are attributed to dysentery. It may almost be laid down as a rule that a supposed case of chronic dysentery in an elderly patient, who has not been out of England, is really one of local disease of the rectum, most probably cancer. A digital examination generally clears up all doubt as to the nature of the disease; and it would appear in most cases that the blood passed during defæcation is not intimately blended with the motion, but comes before or after it.

Progress, complications, and events.—The course taken by dysentery varies much in different epidemics, as well as in individual cases; we shall presently see that it may to a very great extent be modified by treatment.

In favourable cases the symptoms quickly begin to decline. First the pain and tenesmus pass off, and then some of the evacuations begin to contain fæcal matter. Formed fæces may for a time be passed alternately with blood and mucus.

An occasional *sequela* is an affection which resembles acute rheumatism According to Homan and Hertwig, it attacks several joints at the same time, but these do not become very tender, nor is there much swelling. The febrile disturbance is moderate, and the affection subsides in three or four weeks. Parotitis is also said to occur sometimes during convalescence from dysentery.

Portal pyæmia with multiple abscesses in the liver is a most important complication or sequela of dysentery, which will be considered in the following chapter.

In cases which are about to terminate fatally the prostration passes into collapse. The features become sunken, while the body is covered with cold sweat, and exhales a fœtid odour. The urine may be suppressed. The tongue and lips are covered with sordes, hiccup sets it, and a painful sense of constriction of the epigastrium is complained of. The fæces are passed involuntarily, the anus and penis become excoriated, and the lower part of the rectum is often prolapsed. Bedsores are formed if the patient should live long enough. Consciousness is often retained to the end, but in some cases the mind wanders; and during the last few hours all pain may cease, so that the patient fancies he is doing well, or his delirium assumes pleasing forms. Before death the temperature of the body often rises considerably.

In the later stages of dysentery perforation of the intestines sometimes occurs, setting up fatal *peritonitis*. In one case this was observed in a patient at Guy's Hospital. He was in a surgical ward for disease of the knee, when in the month of August he was attacked with "severe diarrhœa." This continued, and he died at the end of a fortnight. The large intestine was ulcerated and sloughing in its whole length, and at one spot the transverse colon was perforated. But in three other cases of sporadic dysentery there has been acute peritonitis without any perforation being discovered to account for it. This seems not to have been hitherto observed in epidemics of the disease.

Chronic dysentery.—In many cases, as already mentioned, acute dysentery passes into a *chronic form* of the disease, which may last for months, or even

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years. It is doubtful whether chronic dysentery ever arises without being preceded by an acute stage. The discharges still have to some extent the peculiar odour. They are for the most part liquid, but they vary in character from day to day. Sometimes they may contain tolerably natural fæcal matter, at other times they consist of a blood-stained mucus or serous fluid. Instead of gaining flesh the patient becomes more and more emaciated. The appetite is generally capricious. The tongue is red and glazed. Abscess of the liver not infrequently forms in these cases; or death may arise from pneumonia, phthisis, Bright's disease, or lardaceous disease of the viscera; or, again, a fæcal abscess may make its appearance in the iliac fossa or elsewhere. Peritonitis from perforation may also occur, even at this period.

Chronic dysentery is by most writers regarded as an exceedingly intractable disease, and one which generally proves fatal at last, the patient dying by exhaustion, with febrile symptoms of hectic type, even if no complication should arise to cut short his existence. Dr Ward, however, says, from his experience at the "Dreadnought," that in the majority of cases recovery at length takes place, if they are judiciously treated. Even then, however, the bowels often remain irritable and easily disturbed for long afterwards, it may be for the rest of the patient's life.

Prognosis.—With regard to the grounds on which a forecast must be based in a case of dysentery, there is little to say that has not been implied in preceding paragraphs. The severity of the disease is proportional to the extent and intensity of the local inflammation, but this must nevertheless be measured during life by the gravity of the constitutional rather than of the local symptoms. Tenesmus and pain may be slight or even absent, and yet the patient may be in very great danger; or his sufferings may be extreme, and yet there may be no grounds for alarm if the affection is limited to the rectum. But the appearance of his countenance, the state of the circulation, and the presence or absence of symptoms of collapse, generally allow a correct judgment to be formed as to the probable issue of the case. The rate of mortality varies greatly in different epidemics. In Dr Clouston's Asylum it was 64·5 per cent. This is enormous, for even in Western Africa the proportion of deaths to admissions into hospital appears from a table drawn up by Sir Alexander Tulloch to be only 14·2 per cent.; and the death-rate there was higher than in any other of the intertropical stations for British troops.

In epidemic dysentery the prognosis must also be greatly influenced by the presence of *scurvy* as a complication. This is exceedingly frequent, so that many observers speak of scorbutic dysentery as a distinct variety of the disease. But it is needless to describe the symptoms of such a form in detail, because they are in fact a combination of those which belong to the two maladies separately. Only it may be worthy of mention that when scurvy is present, the course of the disease is slower that in other equally severe cases. The patient almost always lives three weeks, and often as long as three months.

Even the most severe form of uncomplicated dysentery is seldom rapidly fatal. Death rarely takes place within the first week, or before the ninth or tenth day. At Guy's Hospital, however, some years ago a case occurred in a child who died after five days' illness. She was in the hospital for chorea when she was attacked, and it was a question whether the sulphate of zinc which she was taking could have been concerned in causing the intestinal

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inflammation. The symptoms were so severe that the case was mistaken for one of Asiatic cholera, that disease being prevalent at the time.

Prophylaxis.—Before entering on the treatment of dysentery we may ask whether any preventive measures can be adopted, when the disease prevails, to protect those who have hitherto escaped. The answer to this question is that all general hygienic measures must be strictly attended to, overcrowding being particularly avoided. It is well for the evacuations of those who are sick to be disinfected with carbolic acid or sulphate of iron, and removed without delay, although, as we have seen, it has not yet been proved that they contain a specific poison. Those who are still well must clothe warmly, wear a flannel binder, and be careful to avoid chills; they should eat and drink moderately, taking no strong wine or spirits, and they should not allow the bowels to become constipated. Ripe fruit taken in moderation need not be excluded from the dietary.

Treatment.—The therapeutics of dysentery, as of other diseases, must be based upon a knowledge of its natural course. Dr Austin Flint once observed ten cases in succession in which no medicine was administered except a little tincture of cinchona as a *placebo;* and he found that the mean duration was eleven days and a fifth, the most protracted case lasting twenty-one days and the shortest six. Or, reckoning from the first dysenteric evacuation (instead of from the commencement of illness), he obtained a mean duration of eight and a half days, the maximum being twelve and the minimum five days. Dr Flint had before analysed forty-nine cases, which had been treated, some with calomel and opium, some with opium alone, some with castor oil, and others with astringents. The mean duration was almost exactly the same in these cases as in those in which the disease ran its natural course.

These particular figures are evidently applicable only to dysentery as it occurs in the city of New York, and in strictness only to the actual period within which the observations were made. It is clear that Dr Flint's cases were mild ones; for he states that the complaint showed no tendency to become chronic and that relapses never occurred, although he allowed his patients to eat solid food as soon as they chose to do so.

Very different is Prof. Maclean's account of dysentery in India. "Speaking from large experience," he says, "I affirm that complete restoration to health, by the unaided efforts of nature, is of extremely rare occurrence; the disease either destroys the patient or it passes into a chronic form."

Dr Flint's observations appear to establish the uselessness of the various remedies employed in his earliest series of cases. But we have for dysentery another medicine of which he does not seem to have made trial, but which is proved by the concurrent testimony of a large number of observers to possess the power of cutting short the disease, and even of curing cases that would otherwise have terminated fatally. The medicine is ipecacuanha. The root of this plant was first employed as a remedy for dysentery in Brazil, where it is indigenous. Towards the end of the seventeenth century it was introduced into France, where it was successfully given to the Dauphin, acquired a great reputation, and was known as the *radix antidysenterica.* In India it was used before mercury came into vogue, and of late years it has become the staple remedy.*

* "Dehinc ad radicem Ipecacuanha confugiendum, qua nullum præstantius aut tutius, cum vel sine sanguine, ad fluxus compescendos natura excogitavit remedium."—'Gul. Pisonis de Medicina Brasiliensi,' lib. ii, cap. xi, Lugd. Batav., 1648: quoted by Woodward.

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The action of ipecacuanha in dysentery is as yet unexplained; it must be regarded as a "specific." Its administration renders unnecessary the use of castor oil, tamarinds, rhubarb, or the sulphates of potass or soda, which are recommended by different French and German writers. It is curious that Heubner, in 1874, seems to know scarcely anything of the value of ipecacuanha, except as an emetic; he had only heard that it was used by some English surgeons with good results in the war between France and Germany. Even Eichhorst, writing so late as 1887, does not mention this invaluable drug among the means of treating dysentery.

The method of administering ipecacuanha in acute dysentery, now general, was introduced by Surgeon Docker, of the 7th Royal Fusiliers, in 1858, and first tried by him in the Mauritius.

The patient having been put to bed, twenty-five to thirty grains of powered ipecacuanha are given to him in as small a quantity of fluid as possible; a little syrup of orange-peel covers the taste as well as anything. Some surgeons think it of importance that thirty minims of laudanum should be given half an hour before, in order to make the stomach tolerant of the ipecacuanha; but Dr Maclean says that he has seen the latter drug well borne without any such precaution having been taken. After the dose the patient should keep perfectly still, and abstain from drinking for at least three hours. If thirsty, he may suck a little ice, or have a teaspoonful of cold water at a time. Under this management he seldom complains of excessive nausea, and vomiting rarely sets in within two hours. A poultice is in the meantime placed over the abdomen, or a piece of spongiopiline, wrung out of hot water with a little turpentine sprinkled over it. Afterwards some bland nourishment is given. In from eight to ten hours, according to the urgency of the symptoms and the effect of the first dose, the ipecacuanha is repeated, its quantity being somewhat reduced, but with the same precautions as before.

"All who have had opportunities of trying this mode of treating dysentery," says Dr Maclean, "can bear testimony to the surprising effects that often follow the administration of one or two doses of ipecacuanha. The tormina and tenesmus subside, the motions quickly become feculent, blood and slime disappear, and often after profuse action of the skin the patient falls into a tranquil sleep, and awakens refreshed." The remedy may, however, require to be continued in diminished doses for some days; and even after the stools have regained a healthy appearance it is well to administer ten or twelve grains at bedtime for a night or two.

Even when the powers of life are very low this remedy may sometimes be given with safety and success. Dr Maclean mentions the case of a lady who landed at Madras, having come from Calcutta, and who was so exhausted that her voice was scarcely audible. With some misgiving he gave twenty-grain doses at intervals of eight hours; after the third dose she was out of danger.

When severe vomiting follows the administration of ipecacuanha, Dr Maclean says that coexistent liver disease may be suspected, or complication with malarious fever. In the latter case he advises that quinine in ample doses should be alternated with the ipecacuanha.

In mild cases he recommends that the treatment should be commenced with a hot bath, which must be brought to the patient's bedside. He is to be kept in it until he feels faint, and after being rapidly but carefully dried he is to be put to bed, and to have a dose of fifteen to twenty

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grains of ipecacuanha. In some cases a few drachms of castor oil, with a little tincture of opium, may be afterwards prescribed. A turpentine stupe should be applied to the abdomen, and repeated from time to time. According to Heubner, enemata of starch and opium often give great relief to the tenesmus in these mild cases.

The value of ipecacuanha in treating dysentery is attested not only by individual experience, but by statistical results, which are most striking. In Bengal, under "the old system," the average mortality among Europeans during forty-two years was 88·2 per thousand; in 1860, under ipecacuanha, it was 28·87 per thousand. In Madras the corresponding numbers were respectively 71 and 13·5. Surgeon Mee, at Madras, treated sixty-eight cases from the 44th Regiment "in the ordinary way," with a mortality of 6 (or 88 per thousand); afterwards he treated fifty-nine cases with large doses of ipecacuanha, and these all recovered. It is also asserted by the advocates of this remedy that, as its administration becomes more general, the number of chronic cases of dysentery diminishes year by year, and the development of hepatic abscess, as a complication, becomes less frequent. Hygienic improvements, however, may have had a share in this result.

Against these statements it is proper to set Dr Clouston's experience in the epidemic at the Cumberland and Westmoreland Asylum. He found the ipecacuanha treatment useless, even if it did not (as he appears to think) take away the last chance from one or two of the patients by causing vomiting that could not be stopped and prostration that was never rallied from. But, as already stated, this epidemic was one of a type far more severe than ordinary tropical dysentery.

The experience of the American surgeons of ipecacuanha in the treatment of acute dysentery during the Civil War was not so favourable as might have been expected. It appears, however, to have been little tried. Dr King reported good results in the Confederate army at Richmond, and several other favourable results are referred to in the official report already quoted; but, on the other hand, its trial at Washington ended in disappointment, as was the case in an epidemic in South Carolina in 1868.

Dr Baly says that ipecacuanha wholly failed in his hands. But it is a cardinal rule—although one which is too apt to be forgotten—that in all the worst cases of a disease a medicine may fail to produce any appreciable benefit, and yet it may be capable of curing those which tend but a little less surely towards a fatal termination.

In a recent sporadic case, the present writer saw ♏xv of ipecacuanha wine every four hours, prescribed by a medical friend who had seen much of dysentery in China, completely relieve the symptoms in twelve hours. The chronic cases of tropical dysentery which he is accustomed to see in London, though far less amendable to this treatment, are also in most cases favourably influenced by it.

Calomel has been definitely abandoned in the treatment of dysentery. Opium is believed by all experienced observers to be injurious if systematically given. Venesection has been laid aside, as tending to exhaust the patient's strength to no purpose.

In severe and malignant forms of dysentery Dr Maclean recommends the solution of pernitrate of iron; he says that he has sometimes prescribed ten drops every hour with advantage. The patient must of course be sustained with nourishment, and stimulants may often be freely given. Dr Clouston found that milk boiled with a little flour, and allowed to cool,

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was taken more readily than anything else, and kept up the patient's strength. He also gave strong beef-tea, jelly and eggs, and wine and water.

In *chronic dysentery* an essential part of the treatment is removal to a better climate. In India it sometimes suffices to send the patient to the sea-side; but more often a voyage to Europe is necessary. Persons invalided home on account of chronic tropical dysentery are occasionally seen in the London hospitals. In such cases rest in bed is a very important part of the treatment. The late Dr Ward especially insisted on this fact, and on the necessity that the diet should be carefully restricted to milk and farinaceous food. His experience at the "Dreadnought" has led him to believe that ipecacuanha is useless in chronic cases. But Dr Maclean says that exacerbations of a subacute character are of frequent occurrence in chronic dysentery, and that at Netley he has often given the ipecacuanha in suitable doses with the happiest effect. Our experience at Guy's Hospital has certainly been that this medicine is of great value long after dysentery has been brought home from the East. In at least one instance it undoubtedly did good, even in out-patient practice.

Dr Ward's patients were probably sailors, whose food on board ship would have aggravated the disease, and it is not surprising that they derived so much benefit from rest in bed and a milk diet as to throw the effect of medicines into the background. Even when a person affected with dysentery is sent home as an invalid, with nothing to do but to take care of himself, the disease is very apt to become worse on board ship, unless special care is taken that he has proper food and puts on warmer clothing as he passes into a colder climate.

In some cases of chronic dysentery astringents are of great value. Dr Maclean recommends the solution of pernitrate of iron, which, besides checking the discharges, removes anæmia. Krameria, catechu, hæmatoxylum, even tannic and gallic acids, may each be useful in turn. Marked benefit sometimes results from the administration of drachm doses of the extract of Indian bael, though this hardly accords with the opinion of Dr Maclean that even the fresh bael fruit is only efficacious in dysentery when a tendency to scorbutus is also present. Acetate of lead and sulphate of copper are sometimes useful. Dr Ward mentions one case in which enemata of nitrate of silver (gr. iv ad aq. ℥ij) were repeated every night with good effects. Dr Galton (late of Shanghai) uses enemata of sulphate of zinc, gr. v, and Liq. Opii Sed., ʒss, in ℥ij of water.

In some bad cases of chronic tropical dysentery, the writer has found that, next to confinement to bed and abstinence from meat, broths, and vegetables, the most useful results may be obtained with sulphate of copper and opium pills, and enemata of silver nitrate.

Sir Joseph Fayrer's ripe experience of the treatment of dysentery in the chronic form, seen in Indian officers invalided home, is that drugs are of far less value than in the acute cases seen in the tropics, and that an exclusively milk diet is the most important means of cure.

Acute catarrhal colitis.—There is a somewhat rare affection of the bowels, which is inflammatory in its character and acute in its course, yet distinct from ordinary acute diarrhœa on the one hand and from dysentery or any form of ulcerative enteritis on the other.

The following is an illustrative case. A previously healthy man, thirty-four years old, after exposure to cold and fatigue, was attacked with shivering

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and diarrhœa, accompanied by severe abdominal pain and tenesmus. The temperature rose to 103° F., and there was complete anorexia, scanty high-coloured urine, a thickly coated tongue, pains in the back and limbs, and slight febrile delirium at night. The symptoms were more severe than those of acute catarrhal dyspepsia (p. 318), and vomiting and nausea were absent. There was no source of irritation from food or other ingesta, nor from arsenical poisoning. Moreover, after the bowels had been emptied there remained tenesmus, with passage of abundant, clear, colourless, and inodorous mucus, entirely free from any trace of fæcal matter or bile, of blood, or of pus. In rather less than a week acute symptoms had subsided, but a somewhat tedious convalescence, with great muscular weakness, followed.

Such cases in a less marked degree are not very uncommon both in children and adults. They often appear to be due to direct chill, and though seated in the colon, correspond pathologically to acute gastric catarrh (p. 153) and acute muco-enteritis of the small intestine (p. 219). Warmth, fomentations, diluents, and opiates seem to be the treatment indicated.

Catarrhal colitis is a frequent complication of Bright's disease, and may often go on to ulceration.

Follicular enteritis has been described as a separate affection, on account of the solitary follicles of the colon being primarily affected.

Ulcerative colitis.—A more common disorder, chiefly met with among women and children, is ulceration of the colon and rectum, running a subacute or chronic course, and resembling dysentery in the passage of blood and purulent or muco-purulent matter with the stools, in the tenesmus and most other symptoms. But it does not appear to depend on any of the causes of true dysentery as above described, and does not occur epidemically. Accordingly Dr Eustace Smith and Dr Goodhart admit as dysentery in children only chronic cases which have been acquired in the tropics.

Some cases of severe and even fatal ulcerative colitis in adults are very difficult to explain. When cases dependent on typhoid fever, tubercle, Bright's disesse, dysentery, or syphilis, have been excluded, a certain number still remain, which must at present be classified rather by their anatomy than their origin and pathology. Dr Hale White showed a good specimen of this morbid condition at the Pathological Society on December 6th, 1887.* Another was recorded in the 'Guy's Hospital Gazette' for the 24th of the same month; and a third (which was diagnosed as general tuberculosis following acute catarrhal pneumonia) died in Miriam Ward about the same time.

Membranous colitis, sometimes called "diphtheritic" or "croupous," is a very rare affection. It is not to be distinguished during life from the so-called infantile dysentery or other sporadic cases of ulcerative colitis, unless casts of the intestine should be voided. Dr Goodhart narrates a marked case of this condition in a girl of eleven. There was high temperature, a purpuric rash, and excessive anæmia. After death the rectum and lower parts of the colon were found covered with a thick adherent membrane.

The true pathology of the *intestinal casts* which are sometimes met with is very obscure, and probably they are not all produced under the same conditions. Sometimes they are associated, not with diarrhœa or dysenteric

* See also his interesting commentary on twenty-nine cases in the 'Guy's Hospital Reports' for 1888 (p. 131).

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symptoms, but with constipation. In 1857 Mr Hutchinson showed several specimens of this kind at one of the meetings of the Pathological Society ('Path. Trans.,' ix, 188); they were cylinders several inches or even feet long. Their walls were from one eighth to a quarter of an inch in diameter, yellowish brown, transparent and gelatinous. Under the microscope their surface showed a regular arrangement of round or oval pits, which had evidently corresponded with the mouths of the tubular glands of the intestine. Their substance was almost structureless, but embedded in it were large numbers of epithelial cells. When they had been retained in the rectum, the casts were apt to be changed into hard, white, round masses, about the size of nutmegs.

More recently Dr Goodhart exhibited some specimens to the same Society ('Path. Trans.,' xxiii, 98) which were almost exactly similar, except that they were solid. Many of them passed at their ends into a clear colourless jelly. Both in Mr Hutchinson's case and in Dr Goodhart's there was much complaint of abdominal pain. It does not appear that medicinal or other treatment led to any definite good result.

Occasionally, as the result of severe ulcerative colitis, fragments of mucous membrane are passed. These are not mere casts, like those just mentioned, nor dysenteric sloughs, nor portions of the entire gut thrown off as in cases of invagination (*infra*, p. 246). A patient under the writer's care in Dec., 1887, had at first an enlarged liver with jaundice and pyrexia, but afterwards passed, on several occasions, fragments of mucous membrane apparently from the colon, one of which formed a complete ring, and showed the tubules of Lieberkühn clearly. There was very little hæmorrhage, and none of the distinctive characters of dysentery. After being extremely ill, the patient, a man of about forty, seemed to be slowly recovering, when fresh hepatic symptoms appeared, and he died of abscess of the liver. At the autopsy no other lesion was found except extensive colitis, which appeared to be on the way to complete recovery.

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INTESTINAL OBSTRUCTION.

"Tu Maximus ille es
Unus qui nobis cunctando restituis rem."—VIRGIL.

"Horæ
Momento cita mors venit aut victoria læta."—HORACE.

INVAGINATION—*Anatomy—Obstruction and subsequent strangulation—Ætiology and pathology—Symptoms: pain, tumour, hæmorrhage—Duration of first and second stages—Diagnosis—Prognosis—Treatment.*

IMPACTION—*Gall-stones—Concretions—Fæcal masses in the rectum or colon.*

Compression of the gut from outside.

CONTRACTION AND STRICTURE—*Cicatrices and constrictions—Adhesions and contractions affecting the ileum—Simple and malignant stricture of the colon and rectum—its locality and anatomical structure.*

STRANGULATION—*Bands—Apertures in omentum or mesentery, &c.—Internal hernia—Volvulus—its seats, mechanism, and effects.*

Anatomical results of obstruction and strangulation—General symptoms: constipation, pain, vomiting—Special symptoms of chronic obstruction—course—diagnosis—treatment—colotomy—Special symptoms of acute obstruction—diagnosis—prognosis—treatment: expectant—by abdominal section—by other mechanical methods—Summary—Statistics.

WE now pass to the consideration of a group of affections which differ widely in their pathology, but agree in this, that they cause mechanical obstruction of the bowels, and thus approximate in their clinical features.

Classification.—Obstruction of the bowels may depend upon varied *causes.*

The lumen of the gut may be plugged or blocked by a solid body. This is called Impaction. In cases of Invagination the plug consists of a portion of the gut itself.

The calibre may be narrowed by thickening or contraction of the intestinal walls—a condition called, by analogy to a corresponding disease of the urethra and œsophagus, Stricture.

It may be compressed, drawn, twisted, or squeezed from without, so that if untwisted or released from pressure it would return to its normal condition.

Each of these modes of obstruction may affect either the *small* or the *large* intestine, and the symptoms and results will differ accordingly.

For purposes of diagnosis it is useful to recognise some forms of obstruction as most commonly occurring in *children*, others in *young adults*, and others in *later life.*

The symptoms caused by obstruction of the bowels may run an *acute* or a *chronic* course. The former is more common in affections of the small, the latter in affections of the large, intestines. Chronic cases are more often caused by impaction or stricture, and acute ones by twists or compressions.

Lastly, some forms of *obstruction* to the passage of the intestinal contents

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are complicated by strangulation, that is, obstruction to the circulation in the part of the gut affected. This important and dangerous additional effect does not follow impaction or stricture, but only extreme external compression, as by the neck of a hernial sac. Strangulation always includes obstruction. Obstruction frequently ends in strangulation; but the two processes are pathologically distinct, and each has its own clinical effects.

(1) *Invagination.**—One of the most interesting and important forms of intestinal obstruction is what is known as intussusception or invagination. It occurs when one part of the gut passes into that immediately beyond it, just as the finger of a glove can be made to slip into itself. Out of 500 fatal cases of obstruction collected by Brinton, 215 were due to invagination. Leichtenstern collected 442 cases out of 1152 of obstruction.

The part of the gut which passes into that below it may be compared to a foreign body plugging the intestinal lumen. The coats of the invaginated portion speedily become congested and œdematous, and thus thickened produce a form of stricture; and the pressure of the swollen gut upon itself, together with the dragging of the mesentery and its vessels, produces first congestion, and at last strangulation with gangrene. Hence the three anatomical types of intestinal obstruction above described may be, and usually are, united in a case of invagination.

The seat of the disease is far most frequently at the junction of the small and large intestines. It begins acutely, but sometimes passes into a chronic condition afterwards.

Invagination may occur at any age, but it is most common in children. Indeed, it is the only kind of intestinal obstruction which is frequently met with under puberty.

Anatomy.—The direction in which invagination occurs is invariably downwards (or "forwards"); *i. e.* the sheath (*vagina, la gaîne, recipiens*) is always on the anal side of the intussuscepted portion (*receptum*).

An invagination must obviously consist of three parts, or, as they are often called, "layers." Of these we may term the outermost the "receiving," the middle the "returning," and the innermost the "entering" layer. The latter two together are called *le boudin* by French writers, which we may translate as "the plug." The returning layer, unlike the others, has its mucous outside its serous coat; it is, in fact, turned inside out The bend which connects the receiving and the returning layers is situated at the upper part, and its convexity is formed by the peritoneal coat of the intestine; that which connects the returning and the entering layers is at the lowest point of the intussusception, and the mucous membrane covers it. Adhesions are apt to form where the two serous surfaces of the entering and returning layers come into contact.

Probably almost every portion of the bowel is liable to intussusception. Sometimes one part of the small intestine enters another part, or one part of the colon another part. A case once occurred at Guy's Hospital in which the rectum with just the lower end of the sigmoid flexure passed down through the anus; there was some difficulty in distinguishing it from a mere prolapsus of the mucous membrane. There is, however, one point at which invagination occurs much oftener than anywhere else, namely, at the junction of the small with the large intestine, when the ileum with the valve pushes forward into the cæcum and colon. Such cases are generally distin-

* *Synonym.*—Intussusception.—*Germ.* Einstülpung.

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guished as *ileo-cæcal*, and make up nearly half of Leichtenstern's 442 cases of intussusception. There is very seldom protrusion of the ileum through the ileo-cæcal valve into the cæcum; perhaps not more than 5 per cent. of this "valvular" or *ileo-colic* intussusception. Next in frequency to the ileo-cæcal variety is invagination of the ileum (about a third), then that of the colon, and it is extremely rare for the rectum or the duodenum to be affected.

In twenty-four consecutive cases at Guy's Hospital (1889), the ordinary ileo-cæcal variety occurred in eleven, the seat of disease was in the ileum in two adult cases and in one infant, and there was one example of the rare valvular (or ileo-colic) form. The remaining nine cases recovered without operation, and their exact seat could therefore only be surmised; but most were regarded as ileo-cæcal.

Collected cases probably show an undue proportion of exceptional characters. In this as in other diseases the common type is probably more common and the rare types more rare than appears from statistics.

An invagination at first affects only a small part of the bowel. Gradually more and more of this is involved, and always by the inclusion of one part after another of what had been the receiving layer. Thus the upper bend of the intussusception is constantly shifting, while the lower bend remains stationary from beginning to end. In an ileo-cæcal case, for instance, the entering layer is the ileum; the cæcum forms the returning and the receiving layers; and the lower bend is situated exactly at the ileo-cæcal valve. As the affection advances, the whole of the cæcum, the ascending, transverse, and descending colon may become included; but the ileo-cæcal valve always remains at the lowest point of the mass. Extraordinary as it appears, an invagination of this kind may pass through the anus; and the valve may actually be seen and felt in this position, as well as the orifice leading into the vermiform appendix. The peritoneal layers of mesentery and the vessels of the invaginated gut must be stretched more than could have been thought possible; but there is no doubt that the protrusion of an ileo-cæcal intussusception from the rectum has repeatedly been observed, and it may be regarded as the natural result of the process. When the small intestine is invaginated, the mesentery tethers it much more closely; it is drawn in, and forming a wedge-shaped mass on one side of the gut, pulls on it and drags its lower end to one side, so as to make it assume a contorted form.

The next step is that the circulation of blood in the invaginated mass is interfered with. Thus to *obstruction* is now added *strangulation* of the intestine. The one condition prevents the passage of fæces, and acts in a way comparable to stricture of the urethra, stricture of the œsophagus, or stenosis of the pylorus. The other condition interferes with the nutrition of the invaginated tissues, and causes congestion, inflammation, and at last gangrene.

Sometimes, it seems, the veins alone are compressed; blood can then no longer return from the affected part of the bowel, which becomes enormously swollen, with hæmorrhage and exudation of serum into its tissues. This is particularly apt to occur in ileo-cæcal intussusceptions; but it is often delayed until an advanced period of the case. The swelling is always more marked at the lower bend than anywhere else. Dr Moxon mentions an instance in which the coats of the bowel were three quarters of an inch thick in this position. The included part of the mesentery likewise becomes dark and thickened by effused blood.

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In other cases the influx of blood through the arteries appears to be arrested as well as its escape by the veins. This takes place especially in intussusception of the small intestine, in consequence of the comparatively narrow diameter of the receiving layer in that form of the disease.

The inevitable result is gangrene of the invaginated mass. And, strange as it may appear, this does not always lead to the death of the patient. The sloughing part of the bowel may be cast off, and may pass down the large intestine, and be discharged *per rectum*. It generally appears as a single tube, with its mucous surface outwards; this perhaps includes both the entering and the returning layers, the former having undergone inversion during the process of detachment; or it may be that the entering layer is cast off separately in the form of soft shreds, so that the inverted mass is constituted by what has been the returning layer only. Some years ago a specimen of this kind was sent up to Guy's Hospital by Mr Higginbotham, of Bruton; it consisted of twelve inches of intestine, and within its channel lay the appendix vermiformis, which opened on to its outer or mucous surface. Only an inch of it was small intestine. Consequently, if the intussusception was of the ordinary ileo-cæcal variety, the cast-off mass must have been derived mainly from the returning layer, the entering layer having doubtless broken down and been discharged separately. Several instances have been recorded in which portions of bowel from twenty to forty inches long have been shed. In different cases there are great variations in the extent of the inflammation that occurs at the line of separation, which of course corresponds with what was the upper bend of the intussusception. In a case which Dr Hare brought before the Pathological Society in 1862, and in which the patient died of phthisis three months after passing some inches of bowel, the line of union could only just be detected on the mucous surface by its shining glazed appearance. Even on the serous surface there was only what is described as "considerable puckering," but below the cicatrix a small pouch existed into which projected a curious little hollow cylinder, evidently a relic of the invaginated part. In other instances, however, inflammatory products have been formed in large quantity at the line of separation, and thus the new channel has been reduced to a very small calibre. Or suppuration has occurred at the same spot, and an abscess has developed itself, containing pus alone, or pus mixed with fæcal matter. Cases of this kind probably always terminate fatally. And even when the expulsion of the cast-off bowel is followed by the recovery of the patient there is always a risk that the cicatrix may gradually contract. Dr Moxon has twice seen a fatal annular stricture of the ileum, with puckering of the mesentery, which he believed to have arisen from a former intussusception.

Age and sex.—Intussusception may occur at any age. In adults, however, it is far more rare than might be supposed from the comparatively numerous cases that Hutchinson, Peacock, Brinton, Leichtenstern, and Treves have collected. Dr Wilks used to say that he had only seen one case in a grown-up person. In young children and infants the disease is very frequent; and probably very many cases are overlooked, so that it really occurs oftener than would appear from the published statistics. In fact, with rare exceptions, intestinal obstruction in a child is due to invagination.

The present writer collected twenty-four consecutive cases from Guy's Hospital in ten years (1879–88); of these twenty occurred in children and only two in adults. The youngest patient was two months old, fourteen

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were less than twelve months, and the eight others were between one and seven years old.

Both the adult patients were women between forty and fifty; but of the twenty-two children, fifteen were boys and only seven girls.

Among children, males are far more liable to intussusception than females. Rilliet and Barthez met with twenty-two cases in boys to only three cases in girls. In Leichtenstern's collected cases of all ages, 285 occurred in males and 157 in females. In adults the difference between the sexes in this respect is far less marked. Thus in one year the Registrar-General's returns (according to Mr Gay) gave altogether 163 male to 93 female patients, while between thirty-five and forty-five the numbers were 55 men to 74 women. Mr Treves has shown that the acute cases belong chiefly to childhood, while the chronic ones are more common in adult life.

Origin.—Sudden movements may perhaps sometimes lead to intussusception. Rilliet and Barthez mention two instances in each of which it came on suddenly in a child who was being jumped in its father's arms. Violent muscular efforts to raise a burden have also been assigned as causes of it; and likewise direct injuries.

It probably suffices for the production of an intussusception that the contraction of the transverse muscular fibres of one portion of the bowel should lengthen it, and push it into the interior of the succeeding portion. The foremost or distal (mucous) part of the "plug" probably continues unchanged from the first; the increase in length of the plug is due to the intussuscipiens continually adding to its base (or proximal end) by doubling over.

Everyone who is accustomed to make *post-mortem* examinations knows that in children, particularly after death from cerebral disease, it is common to find two, three, or more short intussusceptions at different parts of the small intestine. They are sometimes reversed or retrograde. The affected parts are not reddened in such cases, and there have been no intestinal symptoms; hence some writers have supposed that the invaginations have arisen after death as a result of rigor mortis. But it appears more probable that they really occurred towards the end of life, and that they would have undergone spontaneous reduction. It is possible by producing strong contraction of a limited portion of intestine with the faradic current, particularly when death by asphyxia has caused acute peristalsis in an animal, to initiate the pathological process of invagination, and cause the contracted portion to push itself into the next piece of gut. Moreover, on opening the abdomen of a rabbit or cat under chloroform or recently killed by carbonic acid poisoning, one may often see more than one invagination form, and sometimes extricate itself again.

One distinction between such intussusceptions and those which give rise to symptoms is that the former do not occur at the junction of the ileum with the cæcum, which we have seen to be the favourite seat of the latter.

The special liability of this part of the bowel to the disease is probably the combined result of two conditions: one, that the axis of the large bowel is nearly at right angles with that of the small intestine; the other, that the cæcum is much more fixed as well as much larger than the ileum.

In two recorded cases invagination of the ileum appeared to have been favoured by the presence of cancerous induration of the cæcum; this may be supposed to have kept it more widely open than in its normal condition. Many instances have been recorded in which the starting-point of an intus-

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susception has been a *polypus* hanging from the mucous membrane. This seems to have been caught by the contraction of the intestine below, and to have dragged downward in its turn the part of the bowel to which it was attached. Dr Moxon met with a case in which an intussusception appeared to have been caused in a similar way by a diverticulum of the ileum; this must itself have first been inverted into the gut. The presence of the *Ascaris lumbricoides* in cases of intussusception has been noticed by several observers, and it seems not impossible that a worm might have its body grasped by the peristaltic movements of the intestine, so as to invert the part to which its head was attached.

In the great majority of instances, however, we find no probable exciting cause of the mishap, nor can we say why invagination is comparatively common at an early age and rare after childhood.

Occasionally invagination occurs as a secondary complication of some other form of obstruction.

Symptoms.—As a rule, the effects of invagination of the bowel are different from those of obstruction from other causes. In the progress of many cases two distinct periods can be recognised. But sometimes, particularly in infants, the former stage is so short that it is passed before the patient is seen.

The first complaint is of a paroxysmal *pain* in the abdomen, with sudden onset, without tenderness, and usually remittent. This pain is generally referred to the neighbourhood of the umbilicus. It is often extremely violent, so that the patient rolls about the bed or the floor of the room in agony. It is as a rule accompanied by *vomiting*. It usually passes off, and may return only after a considerable interval. In one case the patient, a boy five years old, had for four months only two or three attacks of pain during each twenty-four hours; between them he appeared perfectly well.

At first the intestinal contents still pass through the intussuscepted part. The bowels may act every day, and the evacuations are natural.

Although constipation is often absent for a time, another and a most important symptom of intussusception can almost always be detected, even from the first—the presence of a *tumour*. To detect it one may sometimes have to place the patient under chloroform, so as to relax the abdominal walls, and if there be a great quantity of fat in them and in the mesentery, the examination may yield no definite result. But with these limitations, doubt as to the existence of an intussusception may generally be cleared up by thorough palpation of the abdomen. The tumour which occurs in this disease is felt as a sausage-like mass of greater or less length. The note yielded by percussion over its surface may be dull, or partially resonant, or scarcely distinguishable from that given by other parts of the abdomen. Its seat varies with the part of the bowel which is concerned. In the common ileo-cæcal variety it originally occupies the right iliac fossa. As more and more of the intestine becomes involved, the tumour gradually changes its position. It moves across the abdomen, either at the level of the umbilicus or a little higher; having reached the left side it passes downwards into the left iliac fossa, and ultimately into the pelvis. Brinton says that it often forms an elongated mass, which lies horizontally just above the pubes. Another peculiarity of this tumour is that its size and form are liable to frequent changes. It may be hardly perceptible when one first places one's hand upon the abdomen, and may harden and become prominent under manipulation, particularly if a paroxysm of pain should come on.

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As the disease advances, exploration through the anus affords further aid in diagnosis. The end of the invaginated intestine can often be felt with the finger, and ultimately it may even protrude from the anus, like a prolapsed rectum, for which it has often been mistaken. The *rectal tumour* is soft and smooth, the finger feels a dimple not unlike the os uteri in the cervix, and can be carried round it between the returning and the receiving layers. When withdrawn it is unstained by fæcal matter, but covered with blood-stained mucus.

Before the plug has reached the rectum fresh symptoms commonly develop themselves, belonging to the latter stage of the disease. As already remarked, the veins of the intussuscepted part of the bowel become obstructed, and it consequently becomes intensely congested and swollen; *hæmorrhage* occurs from its surface, and blood is discharged by stool, or a mixture of mucus and blood. The blood is red, never black, and is sometimes voided in large quantity. This symptom was present as often as vomiting, namely, in twenty-two times out of twenty-four. *Tenesmus* is a frequent symptom, but only when the plug has reached the rectum. Or blood may be vomited in the rare event of the small intestine near the stomach being the seat of the disease. At the same time, or somewhat later, the passage of fæcal matter through the invaginated part is obstructed. *Vomiting* returns, there are no longer any remissions in the pain, the vomited matters become stercoraceous, the abdomen now becomes distended, the patient is collapsed, and before long he expires. In the case already referred to of a boy who for four months had no symptoms but paroxysmal pain and tumour, death occurred within three or four days from the time when he first began to have tenesmus and to pass blood and mucus. And in infants such symptoms generally set in at the very commencement of the disease, which commonly proves fatal by the second or third day.

Distension of the abdomen by tympanites only occurs in the chronic cases. *Collapse* is an important feature of the acute form of invagination in infants, and is a common cause of death.

It has generally been supposed that the cases attended with hæmorrhage are those in which the invaginated mass is apt to slough away and to be discharged *per anum*, but Mr Hutchinson showed that in chronic cases of intussusception this very rarely occurs. Moreover, the changes which lead to the gangrene and shedding of the whole of the invaginated part of the bowel involve the complete arrest of the circulation of blood in it. Consequently one would not expect hæmorrhage from the bowels to occur while these changes are in progress, although it is true that the ulceration at the neck of the intussusception may lead to hæmorrhage at the time when the sloughing part is being detached. Out of twenty cases collected by Dr Peacock, in all of which the invaginated parts were shed and passed *per anum*, there was only one in which bleeding is said to have occurred, and in that one it ceased twelve or fourteen days before the expulsion of the gangrenous mass. In fact, the symptoms of those cases in which the affected part of the bowel sloughs can seldom be clearly divided into two stages; and very often they are undistinguishable from those of other forms of intestinal obstruction until the portion of gangrenous intestine is unexpectedly voided from the rectum.

Among those cases which present the more characteristic symptoms of intussusception, the duration of the first period varies greatly. The above-cited case lasted four months, and similar instances have been recorded by

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others. A patient of Dr Brinton's died of cancer of lungs during this stage of an intussusception which had lasted four months and a half, and it remained uncertain whether the abdominal affection had any share in determining the time of the man's death. Such protracted cases appear always to be examples of the common ileo-cæcal variety of the affection. On the other hand, many cases, even of this variety, pass into the second stage from their very commencement; and when the small intestine is alone concerned the disease appears generally to take this course. Sometimes hæmorrhage and the other symptoms belonging to the second period set in and afterwards subside. Mr Sydney Jones met with a case in which they lasted for three days and then passed off, returning again seventeen days later, and then leading to a fatal termination.

Brinton says that when the invaginated mass sloughs off and is discharged, the date at which it commonly separates is the eighth day in cases limited to the small intestine, and that it is expelled on the tenth day. In cases belonging to the ileo-cæcal variety the corresponding dates are, according to him, the fifteenth and twenty-second days respectively. But these last figures are of little value because of the variable duration of the first stage in ileo-cæcal intussusceptions.

An acute course is the rule, and is almost universal in children. Of our 24 cases, 18 lasted from twelve hours to five days; 2, nine or ten days; and only 4 were chronic, lasting three, nine, ten, and twelve weeks respectively. Two of these were in adults; the two others and all the acute cases in infants or children under seven.

Dr Goodhart recorded a remarkably chronic case of invagination which lasted twenty-one months in a girl of nineteen. It was apparently occasioned by a polypus ('Clin. Trans.,' 1886).

Diagnosis.—This may either be perfectly easy or exceedingly difficult. It should be laid down as a rule that the abdomen is always to be carefully explored, by the hand laid upon its surface, whenever a patient (particularly a child) complains of paroxysmal pain there, recurring without obvious cause. A tumour may perhaps be discovered, the form of which, and its seat, would be consistent with its being an intussusception. The alternative diagnosis is generally that the tumour is an impacted mass of fæces. An enema (repeated if necessary) generally solves all doubt upon this score; or the suspicion may be confirmed by finding the tumour harden under manipulation, or by detecting peristaltic movements in it. In one instance a rounded swelling, of somewhat cylindrical form, and only partially dull on percussion, was felt crossing the abdomen just above the umbilicus, and was at first thought to be due to intussusception. But the fixity and unchanged position of the tumour, and the absence of other symptoms, soon showed that it was not. The man died some months afterwards, and the case proved to be one of tubercular peritonitis, the mass that had been felt being the omentum indurated with caseous tubercular matter. A gall-stone or a cancerous nodule in the omentum might simulate an invagination in an elderly patient.

In those cases which are attended at an early stage with hæmorrhage from the bowels, there is danger of mistaking the disease for infantile dysentery. In an adult also one must remember cancer of the rectum. Doubt may often be removed by digital examination of the bowel; but even when there is protrusion of the invaginated mass from the anus, it has sometimes been taken for a mere prolapsus. This occurred in a case

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recorded by Mr Hutchinson; a practitioner, called in to see the patient, returned the bowel into the rectum, and fitted a cork pad to the anus to prevent its coming down again.

Prognosis.—Invagination is a very serious disease. The bowel may occasionally extricate itself, particularly if aided by chloroform, as it did in three cases of our twenty-four. But far more frequently the invagination goes on increasing, and the symptoms pass from bad to worse.

Death happens by collapse in acute infantile cases, by exhaustion from starvation, pain and hæmorrhage, or by peritonitis. If the plug sloughs off, death often results from profuse hæmorrhage or from perforation. In sixty-four cases collected by Dr Peacock, however, twenty-five recovered. This event belongs to chronic and adult cases, and is probably more rare than it used to be when cases were less often recognised.

Treatment.—Our object in a case of intussusception is to replace the bowel in its natural position. This may be effected by several different methods. In at least two cases introduction of a gum-elastic bougie into the rectum has succeeded, but it is applicable only when the invaginated mass lies in the rectum, and probably only in the rare cases when the part of the intestine concerned is limited to the lower part of the colon.

Copious injections of warm water have been known to cure the disease. But a much more effectual measure than this appears to be inflation of the intestine with air. This procedure was many years ago (1838) recommended by Mr Gorham in the 'Guy's Hospital Reports' (1st series, vol. iii, p. 345); and he quoted three cases which had been successfully treated in this way in America. It is now frequently employed, and sometimes with the result of completely curing the disease. More often, perhaps, its success has been partial. The tumour has been reduced in size; or it has changed its position, returning towards the seat which it had occupied at an earlier period; or it has been made to disappear for a time, and all the other symptoms have subsided, but only to recur a few days later, with a fatal termination.

The method of injecting air into the bowel is as follows. After chloroform has been administered and the abdomen again examined, a plug of lint is wrapped round an ordinary pair of bellows, at a little distance from the nozzle, and fixed by adhesive plaster. The pipe is then introduced into the rectum, the plug being kept firmly pressed against the anus. Air is then slowly forced in until the abdomen becomes tense, the physician's hand being placed on the abdomen so as to feel any change in the tumour and regulate the distension.

Sometimes it has appeared to be advantageous to place the patient head downwards while either air or water is being injected into the bowel. Care must be taken that too much force is not used, for in the case of an infant, aged five months, which was treated by inflation in Guy's Hospital in 1873, the bowel gave way, and a large quantity of air passed into the peritoneal cavity. Some of it was let out through a puncture in the abdominal walls, but the child did not rally and died in a few hours. In another instance the serous covering in the bowel was cracked in several places and the muscular fibres torn; in that case both air and water were injected. This is a reason for not giving chloroform in the case of grown patients.

Inflation should be practised in every case of intussusception in which the diagnosis is made at a sufficiently early period; but it is evident that if

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the process of sloughing of the included bowel has once set in, the procedure would not only be useless, but would take away the last chance of the patient's recovery. Unfortunately, there are no definite symptoms which indicate that the invaginated mass is passing into a state of gangrene. Still, as Mr Hutchinson has remarked, the fact that in a particular case the tumour is advancing lower and lower in the large intestine is always proof that this is not the case, and that the upper bend is not yet fixed.

If it should be decided that inflation is unadvisable, the patient must be kept well under the influence of opium. Ice may be given him to suck in small quantities, but he should be allowed to drink as little as possible. The treatment, in fact, should be the same as in a case of acute peritonitis or of ordinary intestinal obstruction. After successful inflation the patient should be kept under opium for a time.

Inflation, however, may fail, even if performed early, and repeated two or three times at intervals of a few hours. When it is obviously ineffectual, the operation for opening the abdomen should as a rule be performed without further delay. As far back as the year 1784 this was done successfully in Paris, in the case of a woman aged fifty. In 1873 Mr Hutchinson advocated it in a paper read before the Royal Medical and Chirurgical Society. He had in 1871 performed the operation on a child, aged two years, who had an ileo-cæcal intussusception for a month, which was protruding from the anus. He opened the abdomen in the median line, introduced two or three fingers, and quickly drew out the invaginated mass at the wound. He then easily effected its reduction and returned it into the abdominal cavity. This was all done in two or three minutes, and the child recovered well.

The cases most suitable for this operation are the common ones of ileo-cæcal intussusception, in which the symptoms come on slowly. In such cases, as Mr Hutchinson remarks, there is but little tendency to sloughing and detachment of the invaginated part, so that on the one hand there is scarcely any prospect of a spontaneous cure, and on the other hand the surgeon is very unlikely to find the bowel in such a state as would render it impossible for him to proceed with the operation. But the included portion of intestine is often highly congested, and this may seriously interfere with the complete reduction of the invagination. Dr Goodhart made an autopsy in one case on a child, aged six months, who had died in less than twenty-four hours, and in that instance he could neither by traction nor by squeezing succeed in replacing the whole of the cæcum; the end of it still remained inverted.

In 1874 Dr Adcock, of Bermondsey, asked Dr Fagge to see a woman who had suffered for a fortnight from paroxysmal attacks of pain in the abdomen. A tumour was readily detected which presented all the characters of an intussusception. She had but little sickness, and no constipation. Inflation with air was at once practised, and the swelling then receded from the left iliac fossa towards the right side of the abdomen, which had been its original seat. This clenched the diagnosis. Mr Howse was then asked to see the patient, and after repeating the injection of air, he performed the operation of abdominal section. There was no difficulty in reducing the invagination, and the patient recovered without a single bad symptom ('Med.-Chir. Trans.,' vol. lix).

The advantage of laparotomy in the case of invagination is that the surgeon knows where the obstruction is, and how to deal with it; the

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disadvantages are the early age of most patients, and the exhaustion they suffer from.

Out of 33 cases collected by Peyrot and Treves in which laparotomy was performed for intussusception there were 24 deaths and 9 recoveries. In 10 of these cases (with 7 successful) the reduction was easily accomplished after the abdomen was opened; in the remaining 23 (with only 2 successful) it was difficult or impossible. (See also Mr Knaggs' cases in the 'Lancet,' June, 1887, pp. 1126 and 1177.)

Of the writer's 24 cases, 10 were not mechanically healed; of these, 7 died and 3 recovered. Inflation was practised in 9 cases, with 4 deaths and 5 recoveries. In the remaining 5 cases, abdominal section was performed after inflation (except in one case) had failed, and all died—3 after successful reduction, 2 after resection.

Of the other forms of intestinal obstruction, some depend on plugging of its calibre, others on contraction of its coats, and others on compression from without. Each of these varieties has a different clinical aspect as they affect the small or the large intestine, each may run an acute or a chronic course, and each may or may not be complicated by strangulation.

(2) *Impaction*, the bowel being blocked by a mass occupying its lumen.

a. In the *small intestine* this scarcely ever occurs, except from the presence of a large gall-stone. Forty-one cases are recorded by Leichtenstern, but they are, nevertheless, of rare occurrence. The seat of impaction is usually in the duodenum or upper jejunum, or else at the ileo-colic valve. This accident is most frequent in women, and after middle life (40—70). The symptoms are not very severe; vomiting is early; there is no distension, but considerable pain. Two cases were published by the writer in the 'Path. Trans.' for 1887, in one of which the calculus was felt (though not recognised) *per rectum*; the patient, a woman of seventy, passed it safely, as did the second patient. One stone weighed 270 grains, the other 323. It is curious how such large masses can reach the gut. In some cases they pass directly into it from the gall-bladder by ulceration, but in others very large calculi enter it by gradual dilatation of the duct.

In idiots the ileum has sometimes been found obstructed by balls made up of fibrous materials that had been swallowed. Dr Langdon Down, in 1866, showed to the Pathological Society a mass of interlaced cocoa-nut fibres the size of a hen's egg, which caused death by occluding this part of the intestine; the patient, a boy aged sixteen, had a habit of putting a shred of cocoa-nut fibre into his mouth, and playing with it between his teeth. A similar case occurred to the late Dr Mackenzie Bacon.

Obstruction of the smaller gut by concretions of magnesia is extremely rare. One example is preserved in the museum of St Thomas's Hospital.

It very rarely happens that ordinary articles of food, in passing along the healthy bowel, cause anything approaching to obstruction. An instance is related by Brinton in which an abdominal tumour, the size of a pullet's egg, was believed to be formed of a mass of half-chewed filberts. Having been first detected in the right hypochondrium, it in two days moved almost entirely downwards into the left iliac fossa, and then disappeared; a few hours afterwards the bowels acted for the first time.

β. In the *large intestine* obstruction has occasionally been caused by concretions. Of these one variety (less rare than in the ileum) consists

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chiefly of magnesia. A case in point was brought before the Pathological Society in 1855 by Mr Hutchinson. It was that of a lady who had the rectum blocked by a rough hard body, at least fifteen inches in circumference, which had to be broken down before it could be removed. It was made up partly of strawberry and other seeds, partly of concentric layers of what looked like a red stone, but was found to be a mixture of iron and magnesia. The patient had formerly taken sesquioxide of iron and also carbonate of magnesia in large doses, but not for twelve years before the detection of the mass in her rectum.

In persons who eat largely of oatmeal another kind of concretion is sometimes met with, consisting of a felted mass of hairs derived from the grain. Such masses have a soft, velvety feel. Several specimens of them are preserved in the museum of the College of Surgeons; their nature was first suspected by Mr. Clift, and afterwards demonstrated by Dr Wollaston. As might be expected, they have been found in Scotland more often than elsewhere. They have not generally caused insuperable obstruction of the bowels, but have been passed after giving rise to more or less distress. In one case, that of a man named Gordon, quoted by Sir Thomas Watson, no less than thirty-two such concretions, varying from a hen's egg to a filbert in size, were voided at different times.

Fæcal matter in the large intestine sometimes accumulates to such an extent as to cause symptoms of obstruction of the bowels. A remarkable case of this kind occurred to Dr Peacock, and was recorded by him in the 'Pathological Transactions' for 1872. A man aged twenty-eight died in St Thomas's Hospital after an illness of six weeks, during which he had had obstinate constipation. The bowel, from the cæcum to the rectum, was found loaded with semi-solid greenish fæces, to the amount of fifteen quarts; it measured from six to eight inches in circumference. The patient had been subject to constipation from childhood, and for twelve years before his death his bowels had never acted without an enema, aperients having ceased to produce any effect. Another case which seemed to be of this kind was remarkable, because the patient, a woman aged twenty-two, was attacked with abdominal pain aud other symptoms of obstruction twenty-four days before her death, and yet at the autopsy no cause could be discovered for her illness, unless it were the presence of scybala in considerable quantity in the sigmoid flexure and rectum. This was not all that had accumulated, for much had been removed during life by an enema.

(3) *Compression from outside.*—This may be by a solid tumour, an abscess, an aneurysm, or a cyst. It naturally is a rare cause of obstruction in the looser parts of the bowel, and is by far most common in the *rectum*, confined within the true pelvis. The symptoms are those of chronic obstruction; they are ingravescent and often intermittent. In two cases under the writer's care the compressing tumour was a pregnant retroverted uterus, which was redressed, with removal of the obstruction; in another it was an ovarian cyst, which had fallen into the pelvis, and was tapped *per rectum* with relief to the symptoms.

(4) *Contraction.*—Obstruction may be caused by external traction and constriction, whereby its lumen is narrowed without the circulation being seriously impeded—obstruction without strangulation.

This condition Dr Fagge, in the 'Guy's Hospital Reports,' ser. 3, vol. xiv,

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described and distinguished by the name of "*contraction.*" Dr Bristowe has since called it "compression and traction." It includes cases otherwise described as "obstruction by adhesions," causing traction, compression, shrinking or bending of the gut. The morbid appearances are much less striking in this than in other forms of obstruction of the bowels, and they are not easily illustrated by drawings or preserved in pathological museums. Consequently it had remained almost unrecognised before the publication of Dr Fagge's paper, although it is by no means of infrequent occurrence.

a. The cases of contraction there recorded affecting the *small intestine* were twelve in number. In four of them the affection was the consequence of chronic peritonitis, by which more or less of the small intestine was bound down to some part of the abdominal wall, or by which its coils were made to adhere among themselves. In two it resulted from the puckering caused by cancer affecting the serous covering of the bowel, and from adhesions which had formed. In one instance a somewhat similar condition of the intestine arose as a result of tubercular peritonitis; and in three it was due to contraction associated with chronic disease of glands in the mesentery. The remaining two cases were of a less definite nature.

The peculiarity in this form of obstruction is that, instead of there being one particular spot beyond which the contents of the bowel cannot pass, the impediment is generally continued through a considerable length of the gut. The whole of the small intestine may be matted up, so that one is unable to say that one point more than another was the seat of the obstruction. Or there may be one or more sharp bends or twists or "kinks;" and sometimes it is clear how the part of the bowel above a bend, becoming distended, pressed on that below, and occluded it. Or, one portion of the intestine being fixed by adhesions, that above it may be stretched by accumulated fæcal matter, and hang down into the pelvis, so as to drag on the attached portion, and prevent anything passing through it. More than one striking instance of the latter kind have occurred in Guy's Hospital since 1868. One of the former kind came under the observation of Dr Bristowe, and has been recorded by him in the 'Pathological Transactions' (vol. xxi, p. 185). In it the intestinal coils from the middle of the ileum to within a foot of the cæcum were adherent to one another and to the brim of the pelvis by bands and filaments of false membrane, and were so entangled that their direction was traceable with difficulty; but there was no part of the bowel through which the finger failed to pass.

The origin of the adhesion and contraction is often the presence of an old hernial sac, or the injury inflicted by a strangulation which was relieved by herniotomy. Often it is pelvic cellulitis, with adhesions of the gut to the ovary, uterus, or broad ligament; often former suppuration of mesenteric glands, past typhlitis, and chronic tubercular or cancerous peritonitis. As Mr. Treves remarks, the fact that femoral hernia, pelvic peritonitis, and perhaps malignant peritonitis are more common in women than in men explains the greater frequency of female cases of this form of obstruction.

β. In the *large intestine*, obstruction sometimes arises from its being bound down or adherent to the adjacent structures; in other words, there is an affection analogous to "contraction" of the small intestine. Three such cases were related in Dr Fagge's paper; in one each end of the loop formed by the sigmoid flexure was bound down to the spine by firm fibrous

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tissue; in another the impediment resulted from adhesion of the transverse colon to the neck of an umbilical hernia; and in a third its cause was that the same part of the bowel had been dragged down and fixed to the mesentery over the lumbar vertebræ.

(5) *Stricture, i. e.* narrowing of the gut owing to thickening of its walls.

a. In the *small intestine* we seldom meet with anything comparable to a stricture of the urethra or œsophagus. In one instance, after an operation for strangulated hernia, the patient suffered from continual vomiting, and died in two months; about an inch and a half of the small intestine was found to be narrowed, its coats thickened by hard, white cicatricial tissue, and its mucous membrane almost devoid of villi; evidently this was the part which had been in the hernial sac. Dr Moxon saw two cases in which narrowing of the small bowel was believed to have resulted from the sloughing of an *intussusception*, and similar instances have occurred to other observers.

In the 'Pathological Transactions' for 1869 a remarkable case is related by Dr Wickham Legg, in which the opening from the ileum into the cæcum was only just large enough to admit a catheter; and this writer refers to similar instances recorded. He supposed that in his case the stricture was *congenital*. May it have resulted from the shedding of an intussusception? The patient, a woman aged thirty-two, had nearly all her life been liable to attacks of what was termed colic, and six years before her death she was in University College Hospital under Dr Walshe. It was then noticed that manipulation of the abdomen produced a peculiar crackling, which could be both felt and heard. That this was due to the presence of cherry-stones in the intestine was evident, for on one occasion she passed some. After death the intestine was found to contain almost enough fruit-stones to fill a pint measure; most of them lay in the jejunum or ileum at a distance from the strictured valve, but a few in a dilated pouch measuring seven inches in circumference, which was situated immediately above it.

Most writers state that the cicatrisation of tuberculous ulcers is a not infrequent cause of stricture of the small intestine; and, by way of contrast, that such a condition never follows the healing of the ulcers which occur in enteric fever. But no case of either kind is recorded in the 'Pathological Transactions,' and none has been met with at Guy's Hospital within the last twenty-five years. The nearest approach to it is a case which occurred at the hospital in 1858. A child died of phthisis after an illness of three or four months' duration, of which diarrhœa had been a principal symptom. There were numerous large tuberculous ulcers in the bowel, some of them extending all round it. A remarkable degree of narrowing, apparently from contraction of the peritoneal coat of the intestine, was found at several parts, and through them the blade of an enterotome could hardly be passed. In this instance, however, the symptoms of intestinal obstruction seem to have been altogether absent. Mr Treves figures one case of double stricture of the jejunum from tubercular ulcers, and quotes a unique case, reported by Klob, of stenosis after typhoid ulceration. He remarks that stricture in the small bowel is often double or multiple. Of 26 cases collected by him from various sources 10 were cancerous, and the rest were cicatricial; 10 after ulceration, 4 after hernia, and 2 after injury.

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β. In the *large intestine* stricture is the most common of all the lesions that give rise to intestinal obstruction. Its most frequent seat is the rectum; next the sigmoid flexure. Out of 100 cases, collected from Guy's Hospital, from the list of Mr Treves and from that of Dr Coupland and Mr Morris (excluding the rectum), 58 were in the sigmoid flexure; while, of the remainder, 11 were in the descending colon, 8 in the splenic flexure, 7 in the transverse colon, 10 in the hepatic flexure, 2 in the ascending colon, and 4 in the cæcum. Thus it may almost be said that the liability of the large intestine to stricture increases regularly from its upper to its lower end. Cases of stricture of the rectum, of precisely the same pathological characters, are excluded, because many, perhaps most of them, are admitted into surgical wards. There is no doubt that the rectum is more frequently the seat of stricture generally, and of cancer in particular, than any other part of the bowels.

The affections which cause stricture of the large intestine vary greatly in their nature in different cases. Occasionally the disease consists of dense fibrous tissue, contracting the bowel and puckering its muscular coats; the result of injury in parturition or of operations for piles or fistula. More often it can be traced to the cicatrisation of a *dysenteric* ulcer. Frequently the stricture (particularly in women) is *syphilitic* in origin, usually rectal in locality, and annular in shape. But by far the most common form of stricture is some form of *malignant* growth. This often has more or less distinctly a villous character. In some cases it forms a raised ring of a bright crimson colour, and projecting with a smooth velvety surface into the cavity of the bowel. In other cases it is excavated by ulceration.

Histologically it may occasionally be an ordinary glandiform carcinoma, but it is far more frequently a cylinder-epithelioma. Indeed, Mr Harrison Cripps finds all reported cases of scirrhus or encephaloid cancer of the rectum to be on examination cylindroma; and so M. Hausmann (1882) and Mr Treves (1884). Not infrequently the growth is found to have undergone extensive colloid degeneration. Secondary tumours in the lymph-glands or in other viscera are often absent; one reason being, no doubt (as in the cases of cancer of the stomach and of the uterus), that the duration of the disease is not protracted.

(6) *Strangulation.*—The remaining forms of intestinal obstruction differ from those last considered anatomically, in the fact that the constricting agent has no structural connection with the coats of the affected part of the bowel, and is invested with a distinct peritoneal covering; and clinically by addition of the symptoms of obstructed circulation to those of obstruction.

a. In the *small intestine* cases of internal strangulation form a considerable proportion among all cases of intestinal obstruction. Thus in Guy's Hospital, between the years 1854 and 1869, there were thirteen among fifty-one cases of all kinds of intestinal obstruction. The exact nature of the constricting agent varies.

In many it is a *fibrous band* of greater or less length, attached at each end, but free in the rest of its course. This was described by Mr Gay as the "solitary band," on account of there being usually only a single one present. Such a band may be attached to any part of the mesentery or intestine, or may pass from the mesentery or intestine to the neck of an old hernial sac, or to the uterus, ovary, or Fallopian tube. Or it may consist of a process derived from the omentum, and pass to any of the structures that

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have been mentioned. Its mode of origin is not always to be ascertained, but some observations have been made which show that it may be the direct result of the process by which injury of the intestine is repaired. Mr Gay records a case in which the transverse colon was punctured by a trocar in the operation of paracentesis abdominis. The patient recovered, but died many months afterwards of fever. A firm and thick band, two inches long, was then found, passing from the seat of the wound to the parietal peritoneum. And this writer quotes a case of Jobert de Lamballe's in which a man who had been stabbed in the abdomen by a stiletto died some time afterwards from strangulation of the intestine by a band, which extended from the abdominal wall to the spot in the bowel that had been injured.

In many cases the constricting agent is not a mere fibrous band, but a cord connected with a *diverticulum ilei*. The latter, indeed, is the cause of fatal obstruction of the bowels in a proportion of cases which is remarkable, if we consider how seldom it is found in those who die from other causes. Among fifteen cases of internal strangulation at Guy's Hospital five resulted from the presence of a diverticulum. This is always situated on the side of the bowel furthest from the mesentery, and near the lower end of the ileum. According to Wilkinson King it is never more than from ten to twenty inches above the cæcum; but since his time a specimen has been placed in the museum of Guy's Hospital which was fifty-four inches from the valve. As Meckel long ago showed, this form of diverticulum is a relic of a fœtal structure, the omphalo-mesenteric duct, which passes in the embryo from the umbilical vesicle to the ileum. Before birth it ought to waste away entirely; but its intestinal end may remain pervious and acquire adhesion to the mesentery or to some other part. It is remarkable that this abnormality scarcely ever occurs except in males. Out of ten cases in which a diverticulum was found at Guy's Hospital, only one occurred in a female subject.

Strangulation by a cord attached to the end of the *vermiform appendix* appears to be much less common. Duchaussoy states that females are more liable to it than males; but Leichtenstern's cases were 27 men to 13 women, while those of ileus from a diverticulum were 52 men to 14 women.

A portion of the small intestine may pass through an *aperture* in the mesentery or in the omentum, and then swell and become irreducible, exactly as it would under the femoral arch. Two cases of mesocolic hernia were shown at one of the early meetings of the Pathological Society by Dr Peacock in 1849.

Strangulation may be effected by the pedicle of an ovarian tumour, or by the edge of the mesentery of another coil of small intestine, which is hanging down into the pelvis.

Internal hernia is another cause of strangulation of the small intestine. A remarkable and interesting form of this was first described by Treitz under the name of "retro-peritoneal" hernia, and a case was published by the writer in the 'Guy's Hosp. Rep.' for 1871. A pouch is formed at the back of the abdomen, a little to the left of the spine, passing backwards and downwards behind the curve formed by the inferior mesenteric artery and its left colic branch. The gut is sometimes incarcerated, sometimes strangulated. It is probable that the same jejunal retro-peritoneal hernia was present in another case where Hilton performed an exploratory operation, and drew out a coil of intestine from an opening, apparently in the mesen-

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tery, just at the point where the jejunum became free from the spine. Subcæcal and intersigmoid post-peritoneal pouches have been also observed as the seats of hernia.

It must be borne in mind that in any case of what appears to be internal strangulation the cause may be an external hernia, too small to be discovered by manipulation. Hilton once opened the abdomen during life, and found an obturator hernia, the presence of which could not even then be detected in the thigh; and in a case at Guy's Hospital, in which an exploratory operation was performed, a very small knuckle of intestine was discovered in one femoral ring. Several of us had previously examined the groins most carefully, but had failed to detect any hernia.

A case of internal hernia was observed many years ago by Mr Cooper Forster, in which a knuckle of intestine became strangulated in a sac close to the upper border of the obturator membrane without passing through it.

β. The *large intestine* scarcely ever becomes constricted by a band, or strangulated in any of the ways just described. There are, however, two cases recorded in which the sigmoid flexure was compressed by the mesentery of a coil of small intestine hanging into the pelvis, one in which the ascending colon is said to have been strangulated by a similar cause, one in which the same part of the bowel is stated to have been constricted by the vermiform appendix, and a fifth in which the cæcum was strangulated by a diverticulum.

(7) *Volvulus.*—*a.* Not infrequently, when a knuckle of *small intestine* is strangulated by a band, or is "kinked" by adhesions, the condition is further complicated by the gut becoming twisted on its axis; or, when a loop has passed through an aperture or into a hernial sac, it may twist round the long diameter of the loop. Either condition may be called volvulus.

A primary "twist" of any kind is very rare in the small intestine. If it complicates strangulation by bands or internal pouches, it occurs on their proximal side. Occasionally a loose cæcum or sigmoid is found twisted round a coil of small intestine.

β. The more loosely attached parts of the *large intestine*—the cæcum and the sigmoid flexure—are most liable to primary volvulus. This usually consists in the twisting of the loop formed by the affected part of the bowel, so that each of its ends may be said to be strangulated by the other one being wound round it. The ascending and descending parts of the loop are, in fact, screwed up into a narrow cord where they cross, their calibre is obstructed, and the circulation of blood in the walls of the bowel is arrested. In such cases the affected portion of the intestine is always enormously distended. Thus we have more than once found the cæcum filling nearly half the abdomen, and reaching up into the right hypochondrium; while in another instance the sigmoid flexure extended upwards in a similar way, so as to come into contact with the diaphragm, and push it and the thoracic viscera upwards. When an attempt is made to untwist the coil after death it sometimes springs back into its abnormal position with considerable force.

There is some difficulty in understanding how volvulus is brought about. Both the cæcum and the sigmoid flexure are not uncommonly found floating freely and considerably enlarged in persons who have passed middle age, and perhaps such a condition is a necessary antecedent to the formation of a volvulus. When it has been the cause of death the loop is always found

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full of fluid and intensely inflamed. Much of the contents are doubtless the product of secretion from its mucous surface, and this must evidently have been poured out at an early period of the case, before the arrest of the circulation through the vessels of the affected part. Dr Bristowe has suggested that enteritis is in fact the primary condition, and that the twisting occurs secondarily. He supposes that the portion of intestine becomes first inflamed and paralysed, and that, being heavy with accumulated contents, it is then pushed aside by the pressure of the active parts around it. But this explanation is insufficient to explain the way in which the neck of the volvulus is screwed up; and it is disproved by the exact limitation of the inflammation to the part of the bowel which is twisted.

Volvulus is most frequent in adult males; it scarcely occurs under puberty. The strangulation is early; the pain, vomiting, and other symptoms severe and acute. If seen before obstruction has led to general tympanites a circumscribed tympanitic tumour may be recognised.

A typical case occurred at Guy's Hospital in 1886. A young man, aged twenty-three, was admitted under the writer's care with a history of previous attacks of the same kind, and with recent symptoms of acute obstruction. The pain was intense, the vomiting severe, and it soon became stercoraceous; the meteorismus was enormous. The abdomen was opened on the fifth day, and the distended colon was reached, but the source of the obstruction could neither be reached nor redressed. After death the sigmoid flexure almost filled the abdomen, twisted on itself, and turned up, so that the returning end of the loop touched the liver. Even when the other viscera had been removed it was found impossible to return it into its natural position.

General anatomy of the obstructed gut.—Whatever the cause of obstruction may be, the bowel below the seat of disease is pale, empty, and contracted; that above it is distended, often in the most extreme degree. The jejunum or ileum may become dilated until it is at least as big as the healthy colon; while the colon may reach a size which can only be described as enormous. Dr Moxon met with a case in which, after removal with its contents, the large intestine weighed nearly eight pounds; and some fæcal matter had previously escaped. One effect of the distension is that in the small intestine the mucous membrane becomes forced out between the layers of the mesentery, forming rounded pouches (as in two specimens shown to the Pathological Society in 1875), for which the author proposed the name of "distension-diverticula." In the small intestine the dilatation of the gut diminishes more or less rapidly as one passes upwards above the seat of disease; but the large intestine may be distended pretty uniformly in its whole length; or there may be a great accumulation of fæcal matter in the cæcum, even when the obstruction is situated far below the arch of the colon, this being evidently the result of reversed peristaltic movements on the part of the bowel, by which its contents are forced backwards upon the ileo-cæcal valve.

In other respects the state of the bowel above the obstruction differs in different forms of the disease. In those in which the course is chronic the muscular coat becomes greatly hypertrophied, forming a translucent grey layer, which gradually increases in thickness towards the affected spot. In the acute forms this is wanting, but all the coats may be swollen and injected, so that the bowel feels unnaturally thick and massive. The mucous membrane is very apt to be *ulcerated*, especially in chronic cases, so that nearly the whole lining of the colon is sometimes found to be destroyed. Perfora-

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tion is a not uncommon consequence of this; and we have seen the greater part of the large intestine to be in a sloughing state, so that its contents were escaping from every part. Peritonitis is the necessary result of such a condition, unless the patient dies before it has time to develop. But inflammation of the serous covering of the bowel is also apt to occur at an earlier stage in the more acute forms of the disease, independently of rupture.

General symptoms.—In their clinical history cases of intestinal obstruction divide themselves into two groups, in which the symptoms are to a great extent different, and of which the treatment has to be regulated by distinct considerations. These are respectively *acute* and *chronic* in their course. The most convenient plan will be to take in succession all the points which concern one of these groups before entering upon those which belong to the other. But first there are three symptoms common to both, which require a brief notice, namely, constipation, pain, and vomiting.

Constipation may be said to be the fundamental symptom of obstruction of the bowels. It is generally absolute and immoveable, whether by purgatives or injections, so long as the disease remains unrelieved.

A first enema may bring away fæces which had lain in the part of the bowel below the seat of the disease, but subsequent ones almost invariably return uncoloured; and even below the obstruction the bowel is as a rule paralysed. One would have supposed that mere obstruction high up would have allowed of the colon furnishing several stools before obstruction became manifest; but this is very seldom the case. Often not even flatus can be passed *per anum*. In this, as in many respects, invagination is peculiar, for constipation, as we saw, is not always the first or even a constant symptom of that condition.

It must, however, be remembered that liquid fæces may pass through a stricture which is capable of causing great distension, ulceration, and perforation of the part above it. In a case in which the writer had diagnosed annular stricture of the ascending colon, and had arranged an operation, the passage of a large liquid motion of healthy fæces made us postpone interference. The patient afterwards died, and the cæcum was found enormously distended, ulcerated, and perforated.

Pain is perhaps always present, and it is often of extreme severity. Brinton distinguished two kinds, of which one is a constant pain, corresponding more or less closely with the position of the obstructed part of the bowel, and often referred to the right iliac fossa. There may be some tenderness with it, but this is not usually marked. Probably this pain is always traceable either to disorder of the circulation in the part of the intestine immediately affected, or to paralytic distension of that which lies above the obstruction; in the latter case it may spread over the whole abdomen.

The other kind of pain comes on in paroxysms. It evidently results from spasmodic contraction of some part of the bowel above the seat of disease, but, according to Brinton, only indirectly so; he thinks that its immediate cause is the increase of pressure in the distended coils of intestine close to the point of obstruction, towards which the waves of peristalsis set. This pain corresponds with that of strangulated hernia, which is usually referred to the umbilicus.

Vomiting occurs sooner or later in all forms of intestinal obstruction. Its severity partly depends upon the seat and nature of the affection, but is

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greatly increased if the patient takes much liquid into his stomach. Brinton found that in animals in which he ligatured the intestine, the quantity of fluid which they drank had more influence than anything else in determining not only the amount of sickness, but also the rapidity with which death ensued.

The vomit consists first of the gastric contents, then of bilious fluid from the duodenum, and afterwards of matters derived from the small intestine down to the obstructed part, and possibly sometimes from the colon. These almost always constitute a thin yellow liquid, which, if the seat of disease be high in the jejunum, may have merely a disgusting mawkish odour, but when the obstruction is lower the smell becomes decidedly fæcal at last. The stench is often so powerful as to fill the room in which the patient lies, and to be almost insupportable by those about him. The cause of this "stercoraceous vomiting" has been matter of some discussion. At first it was universally believed to result from a reversal of the peristaltic movements of the intestine, the waves of which were supposed to travel upwards instead of downwards when obstruction existed. By Brinton, however, the occurrence of antiperistalsis was denied; he showed that even though the muscular coats of the bowel should continue to contract in the ordinary way, there would be a tendency to the formation of a double current in its interior, one downwards in the outer part of its channel, and another upwards in its central part, and he conceived that by this the contents of the whole alimentary canal above the seat of disease must soon be so completely mixed up that the fluid in the duodenum and even the stomach would acquire fæcal characters. Since that time, however, Engelmann has demonstrated the occurrence of reverse contractions in the intestines of animals, in which he had opened the peritoneal cavity; it may frequently be observed in the physiological laboratory. In all probability antiperistalsis occurs in the human subject also, under conditions of disease. Indeed, it is difficult to account in any other way for the excessive distension of the cæcum in cases of stricture of the lower part of the large intestine.

It is, however, certain, that the direction of peristalsis is not, as a rule, reversed: for, if it were, the part immediately above the seat of obstruction would not become distended; and mercury or castor oil, when given by the mouth shortly before the patient's death, would not be found to have passed down to the seat of obstruction.

One result of stercoraceous vomiting is that, during the distressing efforts which accompany it, some of the fæcal matter may be sucked into the air-passages. This occurrence has been more than once noticed in the *post-mortem* room; when pressure was made towards the cut surfaces of the bronchial tubes, these gave exit to little yellow cylinders which certainly must have entered them during life; and in one case of strangulated hernia, in which death had arisen from peritonitis after relief of the obstruction, the lungs contained patches of gangrenous pneumonia, which had a yellow colour in the centre, due, probably, to fæcal staining.

So much for the symptoms which are common to both acute and chronic cases of obstruction of the bowels. We now enter upon other points on which these two groups of cases present wide differences; and it will be convenient to deal first with the chronic.

Chronic obstruction of the bowels—of which rectal stricture is the type—

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is characterised by the slow or imperfect development of the symptoms already mentioned.

Constipation is sometimes incomplete, scanty fæcal evacuations occurring from time to time. Indeed, for several weeks or even months before obstruction definitely sets in the patient often has considerable and increasing difficulty in procuring an action of the bowels; or he may have repeated attacks of partial obstruction before the one which at length completely closes the intestine. Even when the constipation is absolute, it is wonderful how life is sometimes prolonged. The late Mr Cooper Forster recorded an instance in which there was not any action of the bowels for eighty-eight days.

Pain is a variable symptom in chronic obstruction. It is seldom or never absent, but often comes on very late in the progress of the case, and when earlier present it is not constant but paroxysmal in character.

Vomiting is often absent in cases of this kind for some days or even weeks after the cessation of fæcal evacuations.

Distension of the abdomen is often extreme: this depends partly on the absence of vomiting, but chiefly on the fact that chronic obstruction in most cases affects the large intestine, and the lower rather than the proximal part of this, so that the cæcum (and often more or less of the colon) as well as the small intestines is full of retained fæces and gas.

Visible peristalsis is almost decisive of the obstruction being chronic. If in such a case the abdomen be examined during a paroxysm of pain, the writhing movements of the intestine can be seen through the parietes; irregular elevations arise here and there, and are succeeded by depressions, or appear to travel from one part of the surface to another. For the production of these appearances to any marked extent it seems to be essential that the coats of the bowel should have undergone hypertrophy; at any rate, it is seldom or never observed in cases of recent obstruction. Peristaltic movements are much more often seen in the small than in the large intestine, but they may undoubtedly occur in the latter. Even during the intervals between the paroxysms of pain the position of the different parts of the bowel is often distinctly visible through the abdominal parietes; and it is to be particularly noted that the transverse colon, when distended, does not continue to lie horizontally across the upper part of the abdomen, but bends downwards, so as to form a broad loop, lying vertically and (with the dilated ascending and descending colon) filling the whole front of the abdomen. This position is, indeed, forced on it by its distension, which makes it too long to occupy its normal position. The coils of ileum, under similar circumstances, are generally arranged transversely. Now, as these coils are often quite as broad as the transverse colon would be, at least under normal conditions, the uppermost one, lying horizontally just below the ribs, may be mistaken for large intestine; and the error has been repeatedly committed. On the other hand, in the case of volvulus of the sigmoid flexure referred to above, we mistook the huge parallel and vertical limbs of the C for the loop of the transverse colon just mentioned.

Course.—The general symptoms presented by a patient suffering from chronic obstruction of the bowels are sometimes exceedingly slight, particularly when the treatment is judiciously managed. His pulse may be quite natural; there may be no fever; he may sleep well at night. The tongue may be clean, and food may be relished.

Such a patient, however, is always on the brink of a precipice. At any

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moment acute symptoms may supervene which may destroy life in a day or two, or still more rapidly. Probably in many cases these symptoms depend upon the occurrence of paralytic distension in the part of the bowel above the obstruction; for this condition is sufficient to account for them. It must also be remembered that ulceration is very apt to take place in the same part of the bowel; and this without symptoms; so that one can never say when perforation may take place.*

Thus it would be difficult to fix an average duration for cases of chronic obstruction, and if one could be fixed it would be of no practical value.

Diagnosis.—The only question of recognising chronic obstruction is caused by its resemblance to impaction of the large bowel with indurated masses of fæces. Or it is better to say that remediable obstruction by scybala with a torpid state of the colon is one form of obstruction by impaction. At first we can form no positive diagnosis, and must be guided by the effect of treatment and the further development of the case. But in all cases of obstinate constipation we must remember the possibility of the cause being a mechanical one, which cannot be overcome, but may easily be aggravated, by provoking increased peristalsis.

Secondly, the chronic obstruction may be due not to plugging of the gut, but to its gradual compression from without by an abscess, aneurysm or tumour, a pregnant uterus, or an ovarian cyst.

Next come two forms of obstruction affecting the coats of the intestines —contraction and stricture—which run a chronic course. We have seen that contractions occur chiefly in the small, and strictures in the large intestine; the distinction between them is therefore to be based mainly upon the points already mentioned as respectively indicating distension of the small or of the large bowel. There is also a difference in the shape of the abdomen when distended, according as the arch of the colon is below or above the seat of obstruction. In the former case the belly is rounded, projecting well forwards, but with comparatively little fulness of the lateral and lumbar regions. In the latter case it is more broad, and if the hand be placed on the patient's loins as he lies in bed, a feeling of resistance is experienced which is wanting when the small intestine is alone distended.

In some instances a tumour can be discovered on palpation; and this, or the fact that the pain is referred definitely to one particular spot, may suggest the exact seat of the mischief. Moreover, all the signs which show that disease of the large bowel is the cause of intestinal obstruction in reality indicate more than this, and point to the conclusion that the affected spot is situated below, or to the left of, the hepatic flexure of the colon. So far as these signs are concerned, chronic obstruction of the cæcum or even of the ascending colon is undistinguishable clinically from that of the lower part of the small intestine; for the transverse colon does not in either case become

* It once happened to me to send up from my out-patient room into the medical ward a woman who had cancer of the lower part of the sigmoid flexure. She had been ill for three months. I went up to see her later in the afternoon, and she appeared to be perfectly comfortable, had a quiet pulse, and presented no urgent symptoms whatever. I therefore thought that the question of colotomy might be safely deferred until the following day. In the evening the nurse was turning her over to give her an enema when she suddenly expired. About a pint of liquid fæces was found in the abdominal cavity, which had escaped from an opening in the sigmoid flexure. There were also several large sloughing patches in the peritoneal covering of the cæcum, as well as its mucous lining.—C. H. F.

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distended. We have seen, however, that the right side of the colon is but very little liable to disease in comparison with the descending colon, sigmoid flexure, and rectum.

With a view to treatment, it is of great importance to make out whether the seat of obstruction is below the middle of the descending colon or above it. In some cases one may perhaps determine this by placing each hand under one of the patient's loins, and by raising them so as to poise the two sides of the abdomen. If a fulness is felt in the right loin which is wanting in the left, it may perhaps be inferred that the ascending colon is distended. Another procedure which may throw light upon the question at issue is the slow injection of a large quantity of fluid into the rectum. Brinton believed that this was capable of yielding trustworthy conclusions as to the seat of the disease. According to this writer, when a pint is the most that can be thrown up, the obstruction is at the upper part of the rectum; a pint and a half, two pints, three pints, correspond respectively with different segments of the sigmoid flexure. The descending and transverse colon can be made to receive larger but more irregular quantities. In one case, in which it was evident that a stricture occupied the upper part of the ascending colon, nine pints of fluid were always found to be the most that could be injected. But it is to be borne in mind that a stricture may be pervious to fluid injected from below, although fæcal matter may be unable to pass through it from above. Thus, in one of the cases recorded in the 'Guy's Hospital Reports' (ser. 3, vol. xiv), in which there was a mass of disease in the sigmoid flexure, just above the pelvis, four pints of warm water were injected, of which only a small portion returned, the rest having doubtless passed upwards through the affected part of the bowel.

It is well known that often in cases of cancer of the lower part of the rectum the fæces are narrow; and one patient with cancer of the upper part of the sigmoid flexure declared that for three months he had noticed his motions to be smaller than natural. But even if this sign is constantly present when the fæces are hard, one must not forget that under conditions of temporary irritation of the colon, formed evacuations of soft consistence may be much narrower than usual without there being any permanent affection of the bowel. Moreover, there must be a point above which disease of the colon would not give rise to any change in the size of the fæces, for they would be moulded into shape lower down.

Digital examination of the rectum is not to be omitted in any case of chronic (nor indeed of acute) obstruction; and if there is reason to believe that the seat of mischief is higher than can thus be reached, it may be well, when the finger fails to reach the diseased spot, to pass the whole hand into the bowel, the patient being under the influence of chloroform.*

All appear to agree that introducing a long tube into the rectum is useless for purposes of diagnosis; it may catch against a fold of mucous membrane, or against the seat of obstruction, and in either case may bend upon itself so as to appear to pass up much higher than it really does.

Inflation of the rectum and colon by air, and particularly hydrogen gas, has lately been much used in America as a means of diagnosing the seat of an obstruction, and apparently with good effect. The plan is in principle like that of distending the stomach referred to above (p. 199).

* This procedure was introduced by the late Mr Maunder, and advocated by Prof. Simon, of Heidelberg. It needs a small hand and much care and patience, but is sometimes an aid to diagnosis in this and other diseases of the abdomen.

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Beside the determination of the seat of obstruction, the pathological character of the disease is also a question for diagnosis, and one which it would in some cases be extremely important to settle, if only this were possible. We have seen that both strictures and "contractions" may be either simple or cancerous, and sometimes the discovery of a definite tumour shows that the latter is the case in a particular instance. But it is doubtful whether there is any other way of proving it. Cancer affecting the bowel is by no means confined to persons of advanced age. Among nineteen cases collected for the paper above quoted in the 'Guy's Hospital Reports,' six occurred in patients less than thirty-five years old. Moreover, malignant disease may be present in a person who looks well and has a florid countenance. Cancer of the bowel perhaps destroys life at too early a period of its growth to give rise to a proper cachexia. On the other hand, if the patient's health is broken down, one can seldom say that this malignant look may not have been the result of pain and sickness.

Treatment.—In cases of chronic obstruction the patient has often been taking powerful purgatives for a considerable time before he seeks medical advice. When this is the case it is generally advisable to abstain from the use of such drugs, and to employ enemata only. The latter are frequently of great service. In almost every instance there is at first a possibility that the disease may be merely an impaction of the contents of the intestine; and even when organic disease of the coats of the bowel is present, the systematic administration of enemata, with or without the use of gentle laxatives, not infrequently removes all the symptoms for a time. The constipation is, indeed, almost certain to return after a shorter or longer interval; and even if it should once more yield to similar treatment, the period at length arrives when the bowels remain occluded in spite of all that can be done. Then, if not before, the patient must cease to take purgative medicines. It is wrong to prescribe them when peristaltic movements can be felt or seen, when the abdomen is becoming rapidly distended, or when symptoms of collapse or of severe constitutional disturbance appear. But small doses of olive oil, as in our Mistura Olei,* are admissible when ordinary aperient drugs ought not to be given. Enemata may be continued somewhat longer still, but at last they too must cease to be administered.

The remedy which should now be prescribed is opium, and that not sparingly; from half a grain to a grain may be given every four hours, or even every two hours, according to circumstances.

If the patient should suffer greatly from flatulence, we may venture perhaps to puncture the intestine with a fine trocar. The late Mr Stocker long ago performed this operation, which is, indeed, only the application to human patients of a very common and apparently harmless operation in the case of cattle with a distended paunch. The introduction of a sharp instrument into the bowel appears to act as a powerful stimulus, for fæcal evacuations have often been passed soon afterwards. That this is not due to the escape of the gas is evident from a remarkable case of the writer's in which puncturing the abdomen in five places with a grooved needle, although neither fluid nor gas was withdrawn, led to complete subsidence of all the symptoms of intestinal obstruction, and to the prolongation of the patient's life for several months. In chronic cases there is little fear of the escape

* ℞ Ol. Olivæ ʒj, Liq. Potassæ mlv, Aq. dest. vel Menth. Pip. ad ℥j. Misce.

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of the contents of the bowel into the peritoneal cavity, for the mucous membrane protrudes into the minute aperture so as to close it.

As soon as it is evident that a case of chronic obstruction will not yield to medical treatment, the question arises whether the bowel should be opened at some point above the obstruction, so as to establish a fæcal fistula. If we can clearly make out that the seat of disease is below the descending colon, the splenic flexure should be selected for the operation. On the other hand, if there be a doubt whether the obstacle lies below the descending colon, but none that it lies below the ascending colon, the hepatic flexure must of course be opened. In either case the operation of colotomy is, as a rule, successful; the peritoneum need not be wounded, and the patient's life is often prolonged for months or even for two or three years. This operation should not be delayed for many days after the administration of purgatives and enemata has been discontinued.

As a remarkable instance of colotomy may be mentioned a case of Hilton's recorded in the 'Guy's Hospital Reports,' 1868, p. 219. There had been absolute constipation for twenty-eight days, but four days after the operation fæces began to pass through the rectum, and in a short time the wound in the loin closed. The symptoms, however, afterwards returned, and it was necessary to reopen the colon. After this a dilator was introduced twice a day with the object of keeping open the fistulous passage; but, in spite of this, it again became occluded. The patient, however, who was himself a medical man, began to regain his strength, and resumed his professional duties, being able to visit thirty families a day without too great fatigue. Ultimately he died of abscess in the left iliac fossa, which communicated with the interior of the hip-joint. The cause of the obstruction appeared, *post mortem*, to be a simple puckering of the coats of the sigmoid flexure at one spot.

In August, 1885, the writer saw an elderly lady with obstruction of the bowels and enormous tympanites, on whom an eminent surgeon had refused to operate. There was evidence of the seat of obstruction being above the rectum and below the splenic flexure of the colon; it was probably an annular stricture. There being no evidence of disease elsewhere, left lumbar colotomy was advised and submitted to. The patient has been frequently seen since, and is now (November, 1890) still living and able to go about.

Mr Bryant brought before the International Medical Congress of Copenhagen, in 1884, a remarkable series of 82 cases of colotomy performed by himself. Of these, 60 were for cancerous and 19 for non-cancerous stricture, 2 for external obstruction, and 1 for volvulus of the sigmoid flexure. As the result, 26 of the patients operated on died within a month; but for the rest the operation gave marked relief, and prolonged life—in 16 patients for between one and six months, in 8 between six and twelve months, in 12 from one to five years, and in 5 for longer periods. The remaining 8 were Guy's Hospital patients, who left the wards convalescent some weeks after the operation, but were not traced subsequently. At the same meeting Mr Henry Morris contributed 23 cases, of whom 14 recovered from the operation, and Professor Studsgaard 20, of whom 17 survived for periods varying from a month to several years.

Whether lumbar or inguinal colotomy is the preferable operation has still to be settled by the results of more extended experience. Having seen ex-

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cellent results from the lumbar operation, and some unfortunate cases of inguinal colotomy, the present writer feels more confidence in the former. But in some very uncertain cases (always the least satisfactory in results) the inguinal operation is almost compulsory, and in this, as in other cases, we must remember that whatever special method of operation a skilful surgeon adopts is sure in his practised hands to be followed by better results than another to which he is less accustomed.*

When chronic obstruction by stricture or contraction affects the small intestine, or even the cæcum or ascending colon, the only feasible operation is to open one of the distended coils in the groin, as advised by Nélaton. The peritoneum must then be wounded; and the chance of establishing adhesions and forming a fistulous opening must be small, unless the serous space should have been closed by previous inflammation. This procedure has occasionally succeeded, but is seldom justifiable.

The symptoms of acute obstruction of the bowels—of which strangulated hernia is the type—must now be considered. They differ to a considerable extent from those which belong to the chronic forms of the disease. The *constipation* is always absolute; any small fæcal masses that may be brought away by an enema come from below the occluded part of the intestine, and even flatus is no longer passed. Severe *pain* is seldom absent, and the *vomiting* is early and severe. The abdomen rapidly becomes distended, but its form presents little that is distinctive of one form of obstruction rather than another. Peristaltic movements are rarely, if ever, to be seen, but the form of the intestinal coils may be plainly visible.

The most characteristic feature of cases of acute obstruction is the early development of *collapse*. The face becomes sunken, with pinched cheeks and dark circles round the eyes; the extremities are covered with a cold sweat; the pulse is very rapid and small, the voice is high pitched and feeble or whispering. The patient, however, often retains perfect consciousness, and is able to lift himself up in bed until just before his death. So close may be the resemblance between the general condition of a man suffering from this form of obstruction and that which occurs in Asiatic cholera, that during one epidemic a case at Guy's Hospital was actually supposed to be one of cholera with retention of the rice-water evacuations, until on *post-mortem* examination the disease proved to be strangulation of the intestine.

In acute obstruction of the bowels *the patient passes little urine*, and the secretion may be altogether suppressed. The late Dr G. H. Barlow, who first noticed this symptom, thought it was an indication that the seat of disease was high up in the jejunum, supposing that it depended upon diminution of the area for absorption of fluid. Subsequently Dr Habershon attributed it rather to the urgency of the vomiting which occurs when the upper part of the gut is strangulated; and Dr Brinton argued that the mucous membrane above the seat of obstruction becomes a secretory rather than an absorbing surface. Both these writers admitted the fact that when the urine is suppressed the disease is high up in the bowel; but, as stated

* The reader interested in the subject will find the case for lumbar colotomy well set forth by Mr Bryant in his Bradshaw lecture before the College of Surgeons, and that for the inguinal operation in Mr Harrison Cripps's work on 'Diseases of the Rectum and Anus.' See also the debate at the meeting of the British Medical Association at Birmingham reported in the Association journal for October 11th, 1890.

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in the chapter on cholera (vol. i, p. 231), there is reason to believe that this symptom is really one of the phenomena of collapse, and due to temporary anæmia of the kidneys. It occurs in all forms of intestinal obstruction, whatever their seat, in which collapse is present.

Ætiology.—In the great majority of cases no directly exciting cause for the attack can be discovered, but sometimes the patient has shortly before eaten something which may have set up excessive peristaltis. In other cases a fall or blow upon the abdomen seems to have been the starting-point of the disease; as in a boy who died in Guy's Hospital of strangulation of the ileum by a diverticulum, and who had a bruise in the right iliac fossa, the result of his having fallen upon some large stones.

Diagnosis.—As with the chronic form of the disorder, we must first ask if there is any other condition which simulates acute obstruction of the bowels.

Strangulated *hernia* is the same pathological condition, whether it occur within or without the abdominal cavity. The only question is whether symptoms of acute obstruction can be explained by the presence of a hernial tumour within reach.

There is no doubt that *peritonitis*, though very different pathologically and demanding different treatment, is often undistinguishable from acute intestinal obstruction. The main symptoms—pain and tenderness, obstinate constipation, and tympanitic distension of the abdomen, together with more or less close approach to collapse—are common to both diseases. Acute obstruction naturally ends in general peritonitis, and acute peritonitis produces obstruction by paralysing the intestinal peristalsis.

Of all kinds of peritonitis, that which results from *typhlitis*, as above described (p. 221), is the most close in its simulation of primary intestinal obstruction. In fact, it must be admitted that there is no single constant and trustworthy diagnostic mark between the two conditions. If seen from the first there need rarely be much practical hesitation; but when peritonitis is fully developed we can only form a judgment as to whether it is primary or secondary to obstruction by weighing probabilities derived from the age, sex, and previous medical history of the patient, and from the onset and course of the present attack.

The only other disease which could be mistaken for acute intestinal obstruction is the sudden impaction of a *gall-stone* for the first time, so that there would be no history of a previous attack. The pain and vomiting so caused is often accompanied by constipation, and it might be several hours before icterus supervened.

Supposing we have satisfied ourselves as to the presence of acute mechanical obstruction, we have next to ask how far we can discriminate between the various conditions to which it may be due.

In the first place, it must be remembered that there is no form of obstruction which may not present itself with acute symptoms. Even in cases of stricture of the large intestine, constipation sometimes sets in suddenly, and quickly leads to vomiting and collapse. The explanation appears to be that, in such cases, the bowel is occluded, not directly by the disease of its walls, but indirectly by œdema, by muscular spasm, or by the bending over of the portion of intestine above, when it has gradually become overloaded with its contents. The proof of this is that, as in Hilton's case mentioned above (p. 267), when colotomy is performed, fæces soon begin again to pass by the

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natural passage. At the *post-mortem* examination, too, one can often pass the finger through a stricture which had caused obstinate constipation during life. We must therefore remember that acute obstruction is the most frequent termination of chronic obstruction.

No doubt, when acute symptoms are present in a case of stricture or contraction, they have been preceded by slighter ones for some days or even weeks. But in hospital practice it may be impossible to elicit this fact when the patient is admitted at an advanced stage of the disease. Thus there may be scarcely anything to distinguish the case from one of primary acute obstruction. The point of most importance, indicating the latter diagnosis, is the absolute suddenness of the symptoms, the patient having been well until the moment when he was attacked.

The pathological conditions, which may be regarded as the causes of primary acute obstruction of the bowels, fall under four principal heads:

1. *Constriction*, or *internal strangulation*, affecting most often the ileum.
2. *Volvulus*, affecting the ileum, cæcum, or sigmoid flexure.
3. *Impaction of a gall-stone*, affecting the small intestine only.
4. *Intussusception*, affecting the ileum, and most often its lower end.

Unfortunately little can be said as regards clinical differences between these several affections.

We may, however, remember that obstruction by a band connected with a diverticulum scarcely ever occurs except in males, and chiefly in patients under twenty years of age.

In the case of volvulus there is little delay in the occurrence of sickness, as in the other forms of obstruction of the large intestine—on the contrary, all the symptoms develop themselves with peculiar rapidity; the abdomen becomes quickly distended in the greatest possible degree, and death may occur within three or four days. Previous constipation is the rule. Volvulus is rare in women and in children.

Obstruction from traction by adhesions is most common in adult women as the result of pelvic inflammation.

Impaction of a gall-stone in the small intestine is to be distinguished, if at all, by the fact that it occurs chiefly in elderly women. In one case of acute obstruction, which proved to be due to this cause, the writer felt the calculus *per rectum* as a smooth round tumour, very moveable, and only just touched by the finger. It was, of course, felt through two thicknesses of gut.

We can in most cases recognise invagination by the hæmorrhage and the presence of a characteristic tumour, either felt through the abdominal walls or reached by the rectum. Without these symptoms we should probably overlook it in an adult, though in a child its probability would remain in the absence of characteristic symptoms.

With respect to the *seat* of acute obstruction, it is in the majority of cases in the small rather than the large intestine, and in the lower rather than the higher part of the small intestine. Hence the utmost attention should be directed, both in exploration under chloroform and after laparotomy, to the right iliac region.

We have seen that diminution in the amount of urine passed is probably the effect of excessive vomiting or of collapse rather than of diminution of the area of absorption, and therefore is no trustworthy indication of the seat of obstruction being high up. But in early cases, which are free from

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peritonitis, if considerable increase of *indican* in the urine is ascertained, it is probable, according to Jaffé's observations, that the obstruction is in the small rather than the large intestine, the explanation being that the indol formed in pancreatic digestion is reabsorbed and secreted as indican when the contents of the intestine are not rapidly passed on. The writer has, however, noticed increase of indican in the urine in cases of cancer of the colon and even in obstinate constipation: so that its excess can be regarded as only an evidence of checked peristalsis.

Prognosis.—The *duration* of acute obstruction of the bowels is subject to considerable variations, which (so far as their cause can be traced) appear to depend on the length of bowel which has its circulation arrested, or is affected with paralytic distension. Mr Phillips has recorded one case in which death occurred within thirty-three hours. But such cases as this are very rare, life being almost always prolonged for three or four days, and generally beyond the first week. Indeed, one may sometimes avail oneself of this for the purposes of diagnosis; a case of obscure abdominal disease which terminates fatally within two days is much more likely to be perforation of the stomach or intestine than obstruction of the bowels.

Treatment.—*a.* In the treatment of acute obstruction, the first point to be considered is whether one should recommend a surgical operation, that of opening the abdomen (*laparotomy*) and searching for the band or other constricting agent, with the object of mechanically releasing the bowel. The analogy of herniotomy is all in favour of such a course if only one can be sure of the nature of the disease; and every pathologist has met with cases in which as soon as the intestines were exposed a band was seen, which could have been divided without the slightest difficulty. Moreover, there are cases in which this operation has undoubtedly saved the patient's life.

But before we can decide as to the advisability of laparotomy, two or three questions must be answered. First, have we the means of distinguishing those forms of intestinal obstruction in which it would be of service from those in which it must necessarily fail? The answer to this cannot be regarded as satisfactory. By carefully selecting for abdominal section cases which presented in the most typical form the symptoms of acute obstruction, one could probably make it a matter almost of certainty that the operation should not be undertaken in a case of stricture or even of contraction. Volvuli and the different varieties of internal strangulation would all be fair objects for operation, although no doubt some of them would be far more favourable than others.*

But a second question must also be answered, namely, whether the cases that would thus be selected for an exploratory operation are sure, if left to themselves, to terminate fatally; or rather whether the risk to life would be greater than if the operation were performed. Dr Fagge once searched the records of *post-mortem* examinations at Guy's Hospital very carefully to find any case of internal strangulation of the intestine in which recovery

* One of the most brilliant operations on record is one performed for volvulus by Dr Senn, of Milwaukee, U.S.A., in October, 1889. The patient was an elderly man who had suffered from intestinal obstruction for a week. The sigmoid flexure was found twisted round its mesentery and distended with liquid fæces. It was twisted into its right position, opened and emptied, sewn up again, and its mesentery "taken up" by a few stitches, so as to shorten it and prevent future volvuli. The wound was closed antiseptically and the patient made a good recovery ('Philadelphia Medical News,' Nov. 30, 1889).

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had taken place and the patient had subsequently died of some other disease. The only instance discovered was that of a man who had been admitted with constipation and stercoraceous vomiting under the care of Dr Addison, and who got well and afterwards died of phthisis. But in that case, although there were two loose bridles, either of which might have strangulated the bowel, there was also adhesion of a coil of small intestine, and the appendix cæci was firmly bound down. Anatomically, therefore, the case probably belonged to the class of "contractions" rather than to that of internal strangulation. Thus, so far as our experience goes, it lends no support to the opinion that where there is actual mechanical constriction of a part of the bowel, this is ever released by natural processes. One can, indeed, well conceive that such a thing might occur. It is evidently possible for the affected portion of intestine to be disengaged by the peristaltic movements of the portion above, or for the constricting band, which is often very thin, to give way. When the cause of strangulation is a band attached to a diverticulum of the ileum, this is commonly softened at the time of the patient's death; but unfortunately its tendency is to give way where it joins the bowel, so that the result would be a fatal extravasation of fæces.

It is, however, undeniable that recovery sometimes takes place in cases which have presented all the symptoms of internal strangulation. As an example may be cited the case of a medical student who was under the author's care in 1874. He was first seized with abdominal pain one Sunday morning about 7 a.m., and soon afterwards had slight vomiting. There was absolute constipation, although he took several doses of purgatives. On the Tuesday his abdomen became distended, the coils of intestine being visible through the parietes. On Wednesday, when seen for the first time, his face was shrunken and his extremities were cold. The sickness was severe; and on Thursday afternoon he rejected a large quantity of brownish liquid which evidently came in part from the intestine, though it did not actually possess a fæcal odour. That night, however, he passed an offensive stool containing numerous scybala; his urine at once became copious, and all his threatening symptoms quickly passed off. As he got better, one could feel an indurated mass in the region of the cæcum, and he had a relapse of short duration, in which there was an increase of tenderness in this part of the abdomen. There is little doubt that the case was really one of typhlitis, and not of mechanical obstruction.* (See on this point Mr Bryant's lecture, 'Brit. Med. Journ.,' 1884, ii, p. 1182.)

* For my own part, I am inclined to believe that in all probability whenever recovery takes place after symptoms of internal strangulation of the intestine, the disease has really been not mechanical obstruction at all, but inflammation of some part of the bowel affecting mainly its serous surface; or, in other words, a local peritonitis. I have twice seen an exploratory operation performed when the cause of the symptoms has proved to be suppurative peritonitis starting from ulceration of the appendix vermiformis. A critical review of the history of each of these cases reveals some differences between their symptoms and those of internal strangulation, but it is easy to be wise after the event.

I was myself at one time strongly disposed to advocate the performance of laparotomy, but I have seen several cases in which it has been done, and almost every one has terminated fatally. I believe, indeed, that in each of them death would have occurred within a few hours if abdominal section had not been attempted; but this only shows shows that if the operation is to have a chance of success it must be undertaken at an earlier period of the disease, before the patient becomes collapsed or is worn out by pain and suffering. This, in fact, is what Mr Howse recommends, and he has given several valuable directions in regard to the employment of the antiseptic method in such cases; but I fear that if such a course were adopted the disease would sometimes be found to be typhlitis.

I am inclined to think that but few successes are likely to be attained by the operation

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b. If we do not feel justified in recommending operation, there can be no doubt of the advantage derived from the free use of *opium*—a dose of one grain given every four, three, or two hours, according to circumstances. It often affords marvellous relief; sickness and pain may entirely pass off for the time: at the worst the patient's death is freed from the suffering which would otherwise have attended it; and if spontaneous subsidence of the disease be possible, this chance is greatly increased.

Under no circumstances should a single dose of purgative medicine be prescribed, even at the very onset of what may prove to be a case of acute intestinal obstruction.

Much relief is afforded by Prof. Kussmaul's plan of emptying the stomach by a siphon-tube and feeding the patient by the rectum. The extremities should be kept warm, and the patient should lie on his back with the pelvis raised, while pain and peristalsis is prevented by subcutaneous injection of morphia.

c. A course which may be regarded as intermediate between opening the abdomen and trusting entirely to the efforts of nature is what Mr Hutchinson has termed *abdominal taxis, i. e.* inverting and shaking the patient, injecting large enemata from a height, and kneading the bowels, in the hope of mechanically reducing the displacement. This is undoubtedly sometimes followed by immediate recovery, as in a remarkable case in which the present writer called in Mr Hutchinson's skill for the purpose of applying taxis (see his 'Archives of Surgery,' vol. i, p. 8, Case II). Sir Thomas Watson relates the case of a lady who observed that the hands of two other medical men who were seeing her with him in consultation were heavy as they manipulated her abdomen; she fancied that their pressure had displaced something within, and almost directly afterwards she passed a liquid motion. Dr Fagge records that the procedure of kneading the abdominal parietes was adopted in a case which he had seen in consultation a few days before, and within a very short time the bowels acted. But striking as such cases appear, it is possible recovery would have ensued if no manipulation had been attempted; and, except in recent cases, it is obvious that the treatment may do harm instead of good. The same remark applies to faradisation of the abdomen, and to the almost forgotten treatment by making the patient swallow metallic mercury, which has been recently revived in Germany. This last method is surely irrational.

Summary.—It is, no doubt, theoretically possible to treat every case of intestinal obstruction by abdominal section, followed by operative release of the gut, or, when this is impossible, by resection of the strangulated or invaginated portion or of the volvulus, or by making a permanent fistula between the gut above and below the obstruction, or at worst an artificial anus. But practically the difficulties would be enormous, and in most cases insuperable. We must remember that the actual mortality after laparotomy is much higher than is indicated by the published cases, and that the unavoidable delays in the course of opening the abdomen and searching for the seat of disease are far greater than might be supposed beforehand. Though delay is dangerous, it is often less dangerous than action which leads to death or cure.

of abdominal section in cases of internal strangulation, and there would be at least an equal number of cases which would have done well without it, and in which, instead of increasing the chance of the patient's recovery, it would rather augment his risk. The question, however, can only be settled by a wider experience.—C. H. F., 1882.

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The appropriate treatment of typhlitis, of invagination, of impaction, and of stricture of the rectum or colon, is, we have seen, tolerably clear, and on the whole successful. In cases of internal obstruction, the seat and nature of which can be diagnosed with some approach to certainty, the results of an exploratory operation with a defined object are likely to justify the risk. But where, as unfortunately is most often the case, we have no such approach to certainty, it is very doubtful whether the expectant treatment—enemata followed by starvation and the full exhibition of opium—does not afford the patient a better chance of life than laparotomy.

It is, however, fair to remember that the want of power on the physician's part to form a diagnosis frequently leads to so long delay, that it is vain to expect a good result from the most skilful surgical treatment. Here, as elsewhere, improved practice must wait upon improved diagnosis.

Meantime, as a practical guide to those who have not seen many cases of these deeply interesting but most difficult and responsible cases, the following remarks are submitted as being in accordance with our present knowledge and experience.

(1) In some cases, perhaps in most, we are able to make a more or less clear diagnosis by careful study of the symptoms, of the previous history (when obtainable and trustworthy), and of the probabilities of the case which result from the natural history of the several forms of obstruction, their frequency and incidence on certain periods of life.

We are then justified in adopting a definite plan of treatment in accordance with our belief. We treat invagination by inflation, opium, and support; in adults abdominal section is indicated should these measures fail. We treat impaction by belladonna and enemata, impaction of a gall-stone by patience, or possibly by laparotomy. We treat stricture of the colon by colotomy,* and internal or partially internal strangulated hernia by operation.

(2) There remains a large number of cases in which constipation, pain, distension, and vomiting prove the existence of mechanical obstruction, but leave quite uncertain its nature, sometimes even its seat and whether it is originally acute or chronic.

In these difficult cases the present writer believes that most lives would be saved by putting the patient under chloroform, manipulating again with a view to diagnosis, shaking and inverting the trunk (supposing the case to be recent), giving large and repeated clysters, and, if this fails, keeping him fully under opium, with the pelvis elevated, and as little food and drink taken as possible.

If, however, it is decided to perform an exploratory operation under these anxious circumstances, it is important that it should be done as early as possible, before tympanites has become marked, and before the patient has been exhausted by pain and abstinence, or the symptoms obscured by opium. The regions of hernia, the sigmoid flexure, and, above all, the right iliac fossa and groin are the parts to be carefully examined; and if nothing

* The following case illustrates the uncertainty of even what seems to be a tolerably certain diagnosis:—A patient under the writer's care in Philip Ward (1889) had every symptom of stricture of the sigmoid flexure. Colotomy was advised, but refused until, after several days' obstruction, he seemed at the point of death. He then consented to the operation, which was performed by Mr Golding Bird with complete success. The patient recovered his strength and returned to his work. Several months afterwards he again came into the hospital with chronic Bright's disease, the bowels acting regularly, sometimes through the rectum, more often through the splenic flexure of the colon. He died of uræmia, and *post-mortem* there was not a trace of stricture, cicatrix, adhesions, twist, or contraction in the whole length of the intestines.

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can be found wrong by the exploring finger, it is probably best to close the wound and trust to treatment by opium.

If there is reason to fear or be sure that peritonitis is already present, that does not appear to be a reason in itself for rejecting an operation.

The subjoined statistics form a practical illustration of the preceding remarks.

a. The following are the results of eighteen recent and consecutive cases of internal strangulation or other severe and *acute obstruction* (excluding herniæ, strictures, and impactions) collected by our medical registrar, Dr Shaw, from the clinical and *post-mortem* records of Guy's Hospital.

Eight patients out of the eighteen recovered, after periods of complete constipation varying from five to eight days in six cases, extending to twelve and fourteen days in the other two. In all these vomiting and other serious symptoms were present, the cases were acute in course, and were believed to have their seat in the small intestine. All were treated by opium or by opium and belladonna with the frequent use of enemata, and without an operation.

Five patients died without operation, under the same general treatment as was used in the preceding cases. In all these cases the causes of obstruction were found in the small intestine, namely, a kink of the gut, fatal on the thirteenth day; strangulation by a band in two cases, one fatal on the eighth day, the other protracted for four weeks; volvulus of the small intestine in two cases, fatal on the eighth and ninth days.

Five of the patients died after an operation. In all the bowel was found strangulated, in three by a fibrous band and in one by the appendix cæci. In three the constricting band was divided and the gut liberated; in two a fistulous opening was made in the bowel above the constriction. The operation took place on the seventh, eighth, ninth, or eleventh day; later, no doubt, than the surgeon would have wished.

b. The following results refer to cases of obstruction (acute and chronic) which have been under the writer's personal observation down to the end of the year 1889. Of the total number of patients (forty-nine), thirty-six were male and thirteen female.

The two youngest patients (18 months and 5 years) suffered from invagination, and there were eight cases of obstruction due to this cause, six in children under 7, and two in adults. Nineteen cases of strangulation (or contraction with adhesions) occurred at various ages, from 5½ years to 46; two of volvulus at 23 and 31; two of impacted gall-stone in women at 60 and 78; four of fæcal impaction in men at or above 50; and fourteen of cancerous stricture of the colon or rectum at 24, 38, 41, 44, and between 60 and 78.

In the cases of stricture of the large intestine the treatment adopted was colotomy, with relief in every case but one, which was fatal on the second day. The five cases of impaction with distension and visible peristalsis recovered under belladonna and enemata: both cases of volvulus died, one after unsuccessful laparotomy; both cases of gall-stone recovered without operation; and one case of invagination died after laparotomy.

The remaining nineteen cases were acute obstruction due to bands, internal hernia, adhesions, and contractions: of these, one recovered after abdominal taxis, and four under expectant treatment by starvation and opium; six died under the latter treatment, and five after laparotomy; while three recovered from obstruction by traction (the result in each case of a precedent strangulated hernia), after an exploratory operation from the groin.

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INTESTINAL WORMS

(CESTOID AND NEMATOID ENTOZOA)

Αἱ καλούμεναι ἕλμινθες· ἔστι δ' αὐτῶν γένη τρία, ἥ τε ὀνομαζομένη πλατεῖα καὶ αἱ στρογγύλαι καὶ τρίται αἱ ἀσκαρίδες.—ARISTOTLE.

Tapeworms—Anatomy and transformations—Tænia solium—its distinctive characters and distribution—its relation to Cysticercus cellulosæ of pork—Tænia mediocanellata—its specific distinctions—its relation to the cysticercus of beef—its geographical distribution—Bothriocephalus latus—its characters and distribution—its probable origin—Symptoms of the presence of tapeworms generally—Preventive and curative treatment.

Round-worms—Ascaris lumbricoides—its anatomy—natural history—distribution—symptoms, diagnosis, and treatment—Thread-worms—anatomy—symptoms and treatment—Trichocephalus dispar—Eustrongylus gigas.

Sclerostomum duodenale—its discovery—anatomy—habits—Anæmia caused by its presence—Treatment.

Trichina spiralis—its discovery—anatomy, transmigrations, and encapsulation—Trichiniasis—its symptoms, diagnosis, treatment, and prevention.

Filaria sanguinis hominis—its discovery in the blood—its transmigrations—its relations to chyluria and elephas.

Dracunculus—Distomum—Bilharzia hæmatobia—its habitat—relation to hæmaturia—Echinorhynchus.

THE human alimentary canal, like that of other animals, affords shelter and food to several species of animals, which are commonly known as intestinal worms. By zoologists these are placed in two separate and widely different groups, Cestoidea and Nematoidea.

There are also other vermiform parasites inhabiting the muscles and the blood, which may also be described in this chapter, particularly as it is probable that they all inhabit the intestines at some period of their lives. On the other hand, the hydatid stage of Cestoidea will on clinical grounds be deferred to the chapter which deals with the diseases of the liver. The most common human *tænia* has been known from classical times, as were also the *lumbrici intestinales*, and the thread-worms, or *ascarides*. Hydatids were regarded as pathological cystic formations, not as parasites, until after the middle of the present century. Trichocephalus is described by Baillie. The other parasitic worms were only discovered within the last thirty years.

The Cestoidea, or "tapeworms," are flat, riband-like creatures, made up of a number of joints, which are arranged in a line from one end to the other. They are really compound organisms, colonies made up of the parent head, and of lineally coherent products of gemmation. There is no alimentary canal. Each "joint" has a double sexual apparatus, both male and female, and is capable of maintaining an independent existence after detachment from the colony.

The Nematoidea, or "round-worms," have long cylindrical bodies;

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they have an alimentary canal, the sexes are distinct, and they undergo but slight changes of form after leaving the egg.

CESTOID WORMS.*—It has been a matter for debate whether the "individual" is the whole colony, to which the popular use of the word applies it, or each separate joint of the tapeworm. There is a so-called "head," provided with suckers, and often with a circle of hooks which fastens itself to the intestinal wall. This at first sight suggests that the entire tapeworm is the individual, and, as Leuckart admits, this view is supported by the fact that the movements of the creature take place by waves transmitted from one joint to another, large portions of it shortening or lengthening at the same time, as though the joints were all under a common directing impulse. But if we deny to each segment of a tapeworm the rank of an "individual" (as we deny it to the components of a compound salpa or of a polyp colony) it is impossible to extend the term from such compound organisms as a sponge or a tree to the *unconnected* products of gemmation, some living and some dead. Yet this is logically required by adopting the definition of an individual as "the total product of a single fertilised ovum until fertilisation is repeated." The fact is that the conception of an individual was originally derived from human consciousness, and is inapplicable to many of the lower classes of animals and to most plants.

By zoologists the tapeworm as a whole is called a *strobila;* the "head" before budding was a *scolex* or budding larva, and the segments are known as *proglottides.*

In the course of their development the Cestoidea pass through an extraordinary series of changes, which bring them within the scope of pathology at various points, and it will be convenient to give a general account of these before describing individual species of tapeworms.

For a reason which will presently appear, the Cestoidea very rarely occur in the alimentary canal of any but carnivorous animals. Let us, then, suppose that a tapeworm is present in the intestine of a man, or of a dog or cat. Its joints or proglottides are by no means all alike. Those nearest the head are very small, and appear almost structureless; for the development of new and imperfect proglottides is constantly going on at this part of the strobila, and these, as they are formed, separate the scolex further and further from those which preceded them. Thus the greater the distance from the head the older and more developed are the joints, and towards the distal end of the tapeworm they are fully developed, and possess elaborate hermaphrodite sexual apparatus. Here the ova are formed and impregnated. In each is presently formed an embryo, which is a globular body, provided at one part of its circumference with six curved hooks arranged in pairs; it is enclosed in a thick shell.

The ova are not discharged from the proglottis through the genital canal or "vagina;" they are, indeed, too large to pass through it; they remain *in situ*, until the proglottis is ruptured and a way is made for their escape. In the meantime the proglottis enters upon a more or less prolonged series of adventures. We have seen that the tapeworm is constantly forming new joints near its head. At its other end the mature joints are as constantly being cast off. Thus every proglottis in turn is pushed on, until by the time that its ova with their embryos are fully developed it

* *Synonyms.*—Tæniada—Tapeworms, including Flat-worms and Bladder-worms or Hydatids.—*Fr.* Vers cestoïdes, kystes hydatiques.—*Germ.* Bandwürmer und Blasenwürmer.

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reaches the distal extremity of the tapeworm, and in its turn becomes detached. When this has happened, it is either discharged with the fæces of its host, or wanders out of the rectum by its own movements; or perhaps it may be ruptured while still within the intestine, in which case its ova are expelled with the fæces.

Having reached the external world, the proglottides creep about for a time. If warmth and moisture favour them, they remain alive and active for some days. Leuckart supposes that they may crawl up the stalk of a plant or a blade of grass, and with this be swallowed by some herbivorous animal. Probably it more often happens that they die and become disintegrated, or that, as Cobbold suggests, the growth of the multitude of ova within them causes them to burst. In either case the ova escape and become scattered in all directions. Some perhaps are carried into streams and ponds, others on to the stems or leaves of plants, where they retain life for several days under favourable circumstances. The immense majority of them no doubt perish, but from time to time one of them meets with the condition which is necessary for its further development.

This consists in its being swallowed by some particular species of animal, generally herbivorous, which may swallow the tapeworm ovum either in the water which it drinks, or in the food which it eats. As soon as the now ripe egg reaches the stomach of this animal, in future to be its "host," its shell is dissolved by the action of the gastric juice, and the six-hooked embryo (larva or *scolex* or *proscolex*) is thus set free. It immediately starts upon an active migration. By means of its hooks it bores through the walls of the stomach or intestine of its host. In this way it is very likely to enter some radicle of the portal vein; and, being washed away by the current of the circulation, to be carried to the liver. In other instances it perhaps continues its active movements through the tissues, until it has reached some other organ far from its starting-point. However this may be, the migration ultimately ceases, and it comes to rest in some part of the body of its host, and there undergoes an entirely new phase in its development. In the first place it begins to grow, and loses its six hooks. It becomes surrounded by a layer of granular matter, which is an exudation from the tissues of its host. Within four or five days from the time when a rabbit was made to swallow the ova of a tapeworm, Leuckart found on killing it, that its liver and lungs were studded with minute white grains, exactly like miliary tubercles, but each having in its centre a tapeworm embryo.

The embryo still goes on increasing in size, and when it has reached a diameter of 0·6 to 0·8 mm., it becomes hollow in the centre, the cavity being filled with a transparent watery fluid. From this time it presents the character of a more or less globular vesicle or bladder; and as it was recognised in this condition long before its relation to its tapeworm parent was understood, it was formerly known as a *bladder-worm*, or hydatid. The *echinococcus* of the human liver, the *cysticercus* of "measly" pork, and the *cœnurus* found in the brain of sheep, are all examples of the hydatid or cystic stage of a tapeworm. All of these are surrounded by capsules of fibrous tissue derived from the tissues of their host, in which they lie free and unattached, but which grow as they grow. It is from the blood-vessels of the capsule that they derive their nourishment.

After a time the growing bladder-worm begins to show a projection from one part of its inner surface, and this gradually increases in size and becomes

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pear-shaped. Soon four suckers make their appearance in the interior of this body, and a circle of minute hooks; it thus acquires a striking resemblance to the head of the parent tapeworm, and after a time a kind of neck becomes developed, by which it is suspended in the interior of the hydatid. In strictness it should be added that the likeness is not exactly to a tapeworm head as one is accustomed to see it, but as it would appear if it were withdrawn into its body, like the finger of a glove turned inside out.

In this condition the bladder-worm may remain quiescent for a lengthened period, embedded in the tissues of its host. It may die there, and its remains shrivel up until only a small cheesy or calcareous relic is left. But if its host should die first it may be set free, and then its transformations begin again. The condition required for its further development is that it should be swallowed by a carnivorous animal, with or without the tissues in which it is embedded. Thus the cysticercus or measle of pigs is swallowed by men or dogs; and the echinococcus cyst of sheep or men by dogs or wolves.

Having thus reached the alimentary canal of a new host, the bladder-worm enters upon a series of changes, which end by its conversion into a tapeworm. In the first place, the parts which have been described as resembling a tapeworm's head and neck, inverted like the finger of a glove, now turn themselves inside out. Thus, instead of being suspended in the interior of the bladder-worm, the head and neck come to project from its exterior, so that some writers now call the original sac the "caudal vesicle." This, however, has but a brief existence. It is speedily dissolved by the gastric juice, except a small remnant, which for a time may be observed attached to the neck. The head and neck resist the solvent action, and pass on into the intestine. There they become attached and begin an active process of growth. Within a few days transverse lines show themselves on the neck, and the segments thus formed increase in size and multiply by gemmation, until in the course of some weeks a jointed tapeworm or *strobila* is developed. The cycle of changes undergone by the parasite is thus completed. We have arrived at the point from which we started.

It must be added that in individual species some of the steps in this marvellous series of transformations deviate slightly from the account just given. But all the Cestoidea, without exception, require two different hosts for the completion of their existence. The one host in which the entozoon assumes the form of a bladder-worm may be either herbivorous or carnivorous, but is most often the former. The other, in which the parasite becomes a tapeworm, must always be carnivorous, since it is only when swallowed with animal tissues that the bladder-worm is likely to enter the alimentary canal of its host.

As may well be supposed, the manner in which tapeworms are developed and their relations to their respective bladder-worms, have only been discovered by patient investigations continued through many years. More than a century ago, in 1769, Pallas noted the close resemblance between common tapeworms and the *Cysticercus tenuicollis* from the abdomen of ruminants. But it was not until the year 1845 that the first definite hypothesis as to the nature of bladder-worms was propounded. And even then the idea was not that they constituted a regular stage in the development of tapeworms, but rather that they were tapeworms which had "strayed" into a wrong animal, and had consequently become dropsical and degenerated. Very soon, however, this was shown to be a mistake, and in

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1851 Küchenmeister administered the *Cysticercus pisiformis* of the rabbit to dogs and succeeded in rearing in their intestines the *Tænia serrata*; he also gave the *Cysticercus fasciolaris* of the rat or mouse to cats, and found that it became developed into the *Tænia crassicollis*. In 1853 the first experiments of the converse kind were performed by the same observer; proglottides of the *Tænia cœnurus* of the dog were given to sheep and lambs with the result that bladder-worms (cœnuri) were found in their brains, the symptoms of "staggers" being also present, which are well known to be caused by this parasite. Since that time similar investigations have been prosecuted with many different species, and the result is that we have now complete experimental proof of the relations and mode of development of many of the Cestoidea.

The number of species of tapeworm which have been known to occur in the human alimentary canal amounts to seven or eight, but of these only three are common enough to require description from the point of view of human pathology.

1. *Tænia solium* (Linn.) was until recently believed to be the most common human tapeworm. When fully developed it measures from seven to ten feet in length, or possibly more.

Its "head" is as big as that of a pin. This is provided with four suckers, and with a proboscis on which is a circle of about twenty-six hooks, ranged with their points outwards. They are of two sizes, and are large and small alternately. The head is often black from the presence of pigment. The "neck" measures an inch in length. The joints are at first very small and broader than they are long. They gradually increase in breadth and still more in length, so that at about a yard from the head they are square, and towards the distal end of the strobila their length is considerably greater than their breadth. There the ripe segments measure about half an inch long by a quarter of an inch in breadth. They have often been compared to melon-seeds, and are, in fact, not unlike them.* The "genital pores" or orifices of the sexual apparatus are placed in a little papilla, which is easily recognised on one of the free edges, more or less regularly on the alternate sides of each successive joint. The "uterus" consists of a central passage, running in the length of the proglottis, and giving off at right angles from seven to ten branches on each side, which again have complex secondary branches. A good way of observing their characters is to compress a tapeworm-joint slightly between two plates of glass, and hold it up to the light.

The eggs are nearly globular, and measure 0·03 mm. in diameter. They have a shell or capsule of considerable thickness, composed of a number of rod-shaped projections, which closely cover its surface, and give it the aspect of being marked with minute radiating lines.

The bladder-worm which forms a stage in the development of the *Tænia solium* is called the *Cysticercus cellulosæ* (*sc. telæ*). It is found chiefly in the pig, occasionally in the monkey, the dog, and some other animals, including man himself. In the pig it occurs in the liver or the brain, but more often in the connective tissue between the fasciculi of the voluntary muscles.

* Hence they were called *cucurbitæ* and the worm *Tænia cucurbitina*. According to Küchenmeister, the Arabs call the complaint "Chabb-al-kar," *i. e.* pumpkin-seed. Aristotle makes the same comparison: The flat mawworm produces bodies like colocynth seeds, by which physicians detect its presence.—'Hist. An.,' lib. v, cap. xix.

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Pork so affected is said to be "measly," *i. e.* spotted). Its relation to the tænia would be rendered probable by the identity of the scolex which it contains with the head of that creature. But this point has been conclusively demonstrated by experiments of both kinds. Van Beneden, Leuckart, and others have administered proglottides of the tapeworm to pigs, and the result has repeatedly been that the flesh of the animal has become full of cysticerci, the size of which has corresponded with the length of time that may have been allowed to pass before it was killed. Two months and a half are required for the full development of the cysticercus. From observations made by Stich it is probable that its life within the tissues (at least in man) is limited to from three to five years; he found that at the end of such a period cysticerci in the subcutaneous tissues, which had been plainly felt through the integument, became flaccid and shrank away until their presence could no longer be discovered.

The converse experiment to that of feeding pigs with the proglottides of the tænia of course consists in the administration of cysticerci to human beings. This has been occasionally done, the victims being sometimes criminals condemned to death; sometimes persons who volunteered for the purpose. Perhaps the most striking instance is one of Küchenmeister's. He gave to a criminal twenty cysticerci on each of two occasions, one of which was four months, the other two months and a half before his execution; nineteen tapeworms were afterwards found in his intestines. A young man who of his own accord swallowed four cysticerci in Leuckart's presence began, for the first time in his life, to pass proglottides in his fæces three months and a half afterwards, and a month later took a dose of kousso, with the result that he passed two tapeworms, each about two yards long.

The name *Tænia solium*, given to this parasite by Linnæus, was meant to imply that it occurred singly in the intestine;* and the same notion is expressed by the French title, *ver solitaire*. But this is a mistake. Two or three are not uncommonly present in the same individual, and cases are recorded in which twenty-five have been passed by a single patient.

This parasite is more common in adults than in children, and it has been often found in butchers and in cooks; these facts are of course just what might be expected, since it is derived from measly pork. Out of Europe it is said to have been observed only in India, Algiers, and North America. The duration of life of this tapeworm is estimated by Leuckart at from ten to twelve years. Cobbold mentions the case of a patient who was infested with it for sixteen years. It is said to have been present for as long as thirty-five years; but Leuckart thinks it probable that in such instances the worm was not the *T. solium*, but the *T. mediocanellata*, which will be presently described.

The cystic form.—Cysticercus cellulosæ is also sometimes found in the human subject, and this is the only known instance in which man is liable to both the larval and mature forms of a cestode entozoon. As a bladder-worm, the parasite is observed most commonly in the eye and in the brain; but it is very likely that it is really most frequently present in the muscles and subcutaneous tissue, where, however, it is very apt to escape notice. It

* It should have been written *T. sola*. The same notion was expressed by the specific name *T. solitaria* (Bradley). Of other synonyms *T. cucurbitina* (Pallas) referred to the proglottides, *T. dentata* (Gmelin) and *T. armata* (Brera) to the hooklets. It is often called the "armed tapeworm," the mawworm, or the "pork tapeworm."

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appears to be found from time to time in the German dissecting-rooms. It is often solitary or present only in small numbers; but in Stich's case at Berlin more than three hundred could be felt through the skin. The writer only once saw cysticercus of the subcutaneous tissue; the cysts were very numerous, and were found after death in the body of an old man.

A person who has a tapeworm in the intestine cannot derive cysticerci directly from its ova; they must first pass through the stomach, where their shells are removed by the action of the gastric juice. Still it is remarkable that such patients do not more commonly become affected with bladder-worms. For the ova are very apt to hang about the anus, and must frequently be carried thence, particularly at night-time, and finally might reach the alimentary canal. Moreover, long-continued retching might bring the worm itself or some of its joints into the stomach. As a matter of fact, however, very few of those who have a tapeworm become affected with cysticerci; although von Graefe found that among thirteen patients with cysticercus in the eye five had tapeworms.

The ova closely resemble those of the pork-tapeworm in their minute size, shape, and thickness of the capsule.

2. *Tænia mediocanellata** (Küchenmeister) was long confounded with *T. solium*. The head in *T. mediocanellata* is flattened, and has neither proboscis nor circle of hooks; there is commonly, but not always, much pigment deposited round the four suckers. The strobila is much broader than that of *T. solium*, so that each segment has a square form; and its water-vascular system is more simple in its arrangement. It is often called the "unarmed" tapeworm, to distinguish it from *T. solium*, which is armed with its circle of hooks.

The strobila also presents peculiarities which a physician must be acquainted with, as it is often desirable to determine the species before the head can be obtained. It is considerably longer than *T. solium*. Leuckart says that it may reach four yards in length. It is also firmer in texture and flatter, and of a darker colour towards its distal extremity. Its joints are more numerous. The sexual organs attain their full development, as in *T. solium*, about the 450th joint from the head; but whereas in that species the uterus is full of ova at about the 200th joint further on, in *T. mediocanellata* this is not the case before the 360th or 400th joint.

The ripe proglottides are larger than those of *T. solium*, measuring three quarters of an inch in length and a quarter to one third of an inch in breadth. They are more apt than those of *T. solium* to creep out of the patient's anus independently of defæcation. They also more generally rupture and discharge their ova while in the intestine, so that those which are passed *per anum* are often shrivelled and empty.

But the most important peculiarity of the proglottides of the beef tapeworm is in the form of the uterus. This has from twenty-five to thirty branches on each side of its longitudinal channel (*T. solium* having only from seven to ten); they are, therefore, packed much more closely; they are simply forked over and over again, and terminate in round, club-shaped ends, not in the broad, notched, or leaf-like pouches which are seen in the pork-tapeworm. The eggs are not globular, but slightly oval in form.

Another peculiarity of *T. mediocanellata* is its liability to malformations.

* *Syn.*—*Tænia inermis*—*T. saginata*. Küchenmeister gave it the specific name of "mediocanellata," believing that it had a median water-vessel in addition to the two lateral ones; but this was apparently an accidental peculiarity of a malformed specimen.

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Sometimes there are two or three genital pores in a single proglottis, each corresponding with a separate double sexual apparatus; sometimes the segmentation is incomplete; sometimes a supernumerary proglottis projects by the side of the continuous line of joints. The most remarkable malformation is one in which there are two distinct chains, united in their whole length by one edge at an acute angle, so as to constitute a "double monster."

This tapeworm has only been recognised as a distinct species since Küchenmeister's original account of it was published in 1852. Bremser, indeed, had previously noticed that the tæniæ which he obtained from human beings in Vienna had no hooks; but he thought that they had dropped off in consequence of the age of the worms. Other observers adopted this view, although it obviously could not account for the fact that all the tapeworms in a particular district were unarmed.

For some time longer the source of *Tænia mediocanellata* remained undetermined. It had, however, been observed that the tapeworm which was known to be common in Abyssinia belonged to this species, and that the people there ate, not raw pork, but raw beef and mutton. It was also noticed that infants to whom raw beef grated fine was given under medical advice got a tapeworm, which at least in one instance was unarmed. Küchenmeister related the case of a patient who had harboured this parasite ever since a particular period when he had fed several times on raw beefsteaks. Putting these facts together, Leuckart came to the conclusion that the bladder-worm corresponding with this tænia probably occurred in horned cattle. He therefore in 1861 gave part of a strobila on two occasions to a young calf. Twenty-five days after taking the first portion of tapeworm the calf unexpectedly died. The muscles (including the heart), the lymphatic glands, and other parts, were full of minute round or oval vesicles, embedded in an opaque, whitish substance, which made them much more conspicuous objects than they would otherwise have been. They looked very like tubercles, and, indeed, the affection has sometimes since been spoken of as "acute cestode tuberculosis." The experiment has since been repeated with the same result. It has also been shown that this form of bladder-worm has but a brief existence; if its host is allowed to remain alive it perishes and calcifies in about eight months.

The frequency of *T. mediocanellata* as compared with *T. solium* varies, as might be expected, in different countries, according as the people live more on beef or on pork. It is stated that in Bavaria and Würtemberg the armed tapeworm is never met with, whereas in North Germany it occurs almost to the exclusion of the unarmed species. In England, Cobbold found that *T. solium* is more common among people of the lower class who eat much pork, whereas *T. mediocanellata* occurs in those who are better off, and eat more veal or beef. But he was gradually led to the conclusion that the latter, on the whole, is the tapeworm which is most prevalent in this country.*

3. The only other species of tapeworm which is common in man is *Bothriocephalus latus* (Bremser), or the "broad tapeworm." It is said to have been originally distinguished in the seventeenth century by Felix Plater,

* A third species of the same genus, *T. nana*, was once discovered by Bilharz in large numbers in the intestine of a boy at Cairo. Others have been described in isolated cases, from Iceland, North America, and the West Indies. *T. echinococcus* has never been found as a strobila in man.

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who called it *Tænia prima*, to distinguish it from the worm after named *T. solium* by Linnæus, which he called *T. secunda*. It is larger in every dimension than any other human parasite. It measures eight to twenty or even twenty-six feet in length, and has from three to four thousand joints. These segments are broader than they are long. In the middle of the strobila they are nearly half an inch broad by one seventh of an inch in length. Towards the distal end they increase in length and diminish in breadth until at last their form is almost square.

This tapeworm has a longitudinal projecting ridge traversing its whole length. Its head is unarmed; it is club shaped, and has two deeply-grooved longitudinal suckers, one on each side, whence it takes its generic name of "pit-headed." The reproductive organs differ altogether in appearance from those of the tæniæ. The genital pore lies in the middle of each segment, opening upon its ventral surface. The uterus is an unbranched tube, which is bent on itself four or five times each way. When distended with ova its loops are flattened against one another, so that it resembles a rosette.

The eggs are larger than those of the tæniæ; they measure 0·07 mm. in length, and are oval in form, with an operculum or lid at one end which allows the escape of the embryo. The capsule is comparatively thin.

A peculiarity of this tapeworm is that its joints do not come away singly, but that portions of the strobila from two to four feet in length are expelled with the fæces.

Like the Tæniæ, the broad tapeworm is usually single, but like them, two specimens are occasionally found in the same patient. In the 'Pathological Transactions' for 1890, Dr Montague Murray records the occurrence of no less than seven tapeworms of this species, which were found unattached, in an entangled mass, in the cæcum of a man who died of tubercular meningitis without abdominal symptoms. He was an Englishman by birth, but had lived several years in Sweden, Finland, and Russia.

The *Bothriocephalus latus* is almost limited to the inhabitants of certain countries of Europe. The locality in which it is best known is the western part of Switzerland; in Geneva one person in every four is said to harbour it. It also occurs in the north-west of Russia, in East Prussia, in Sweden (where the whole population of one province is said to be infested with it), in Poland, Holland, and Belgium. Leuckart speaks of its having been observed in persons living in London, and implies that this has sometimes been the case in those who could not have obtained the parasite from abroad; but this is doubtful. It is not impossible that *Tænia mediocanellata* may have been sometimes mistaken for it; for Leuckart remarks that muscular contraction may shorten the joints of that species, and anyone accustomed to the comparatively small *T. solium* might assume that a worm so much larger was the so-called *T. lata*.

The source from which this parasite enters the human body has not yet been certainly determined. The observation has long been made that the districts in which it is met with are low-lying regions, situated either near the sea, or at least near some large lake or river, and it has been suspected that the corresponding bladder-worm inhabits some kind of fish, or possibly a fresh-water mollusc. Salmon, trout, and bleak have especially been mentioned as likely to prove to be the resting place of the immature form of the bothriocephalus. Such a view derives some support from the fact, first discovered by Schubart, but more fully made known by Knoch in 1862,

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that by keeping the ova several months in water, each of them gives out an embryo posessing the usual six hooks, but enclosed in a membrane which is completely covered with beautiful long delicate cilia. These enable it to keep up a constant rotatory movement, like that of a volvox. After four to six days, it escapes from the ciliated membrane and becomes free. Its further fate has as yet eluded observation. Knoch, indeed, thought that he had proved that the administration of proglottides of bothriocephalus to puppies led to the direct development of the tapeworm in their intestine, but the validity of his experiments is disputed by Leuckart.*

Symptoms of tapeworms generally.—The effects of the presence of a cestoid worm in the human intestine are somewhat vague. Adults in robust health, and even healthy children, experience as a rule no discomfort whatever; it is only when proglottides or portions of their strobila are evacuated, that a suspicion arises that they are not perfectly well. When this has once happened, the patient often begins for the first time to complain of pains and other symptoms, of which nothing had before been heard, although the parasite must have been present for several months. The sensations which are said to have been directly caused by it are described as an "uncomfortable feeling in the abdomen," "a colicky pain," a "gnawing pain at the epigastrium, especially when the stomach is empty or after certain kinds of food." Sometimes the patient is convinced that he can feel the movements of the worm; and, in reference to this, it is to be said that the sluggish contractions of the strobila outside the body give no idea of its activity while under the influence of the warmth of the intestine. Leuckart particularly speaks of the vigorous motions of its segmented body, the continual play of its suckers, and the bendings of its neck. It usually hangs at length, but sometimes it is bent on itself, or rolled up.

Leuckart mentions that in the dog he has sometimes observed injection of the mucous membrane, separation of the epithelium and even ulceration, produced by tapeworms; but it is doubtful whether such changes in the human intestine, even if they occur, would go further than the mere presence of the parasite towards explaining any symptoms that might be observed. Foulness of the breath, and irregular and craving appetite, constipation, or very rarely, diarrhœa, are said to be caused by the existence of a tapeworm in the human subject.

Morbid sensations are also sometimes produced at distant parts,—itching of the anus, itching of the nose, so that the patient is always picking it, headache, giddiness, lassitude, and faintness. As might be expected, such symptoms are observed chiefly in persons of nervous temperament. Grinding the teeth at night is another symptom, and patients have been known to have hysterical fits, epileptic fits, and even maniacal attacks, which have been cured by the expulsion of the worm. Dr Graves relates the case of a young lady, who was attacked with what were regarded as alarming symptoms of bronchitis. She had a dry, hollow cough, which was repeated every five or six seconds, night and day, whether she was asleep or awake. Bleeding, tartar emetic, blisters, antispasmodics, were tried in turn, but without result, until Dr Graves gave up the case in despair. At last she

* Another species, *B. cordatus*, has been more than once observed in human beings in Greenland, and a third, *B. cristatus*, twice in France.

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had a sudden attack of colic, for which an old servant of the family gave her a full dose of oil of turpentine with castor-oil. She passed a large piece of tapeworm, and from that moment every symptom of pulmonary irritation disappeared.

We are not justified in prescribing anthelmintics indiscriminately for all cases of spasmodic nervous affections of which we are unable to find the cause, but we should at least not forget to inquire as to the presence of worms in such cases.

The bothriocephalus is said to give rise to more marked symptoms than the tæniæ, but even it may be altogether latent. Bremser mentions the case of a Swiss, who had been eleven years away from his native country before he discovered that he was the host of this parasite.

Prophylaxis.—To prevent the development of tapeworms in the human intestine two measures may be taken which, however, do not apply to the bothriocephalus, since the seat of the corresponding bladder-worm has as yet only been guessed at. In the first place, meat which is observed to contain cysticerci should not be eaten at all; and in the second place, all meat should be so well cooked before being eaten, as to destroy any cysticerci that may chance to be present.

Measly pork may often be easily recognised; the bladders are of considerable size and may be present in very large numbers. But it is remarkable that in the flesh of horned cattle cysticerci have never yet been seen, except after the experimental administration of proglottides of *T. mediocanellata* to the animals. The ox or heifer is a more cleanly feeder than a pig, and so its only chance of being infected with the cysticercus is by swallowing stray ova on the leaves of the plants which it eats or in the water which it drinks.

The second precaution against tapeworms consists in eating only meat which is thoroughly well cooked. The cysticercus cannot survive the temperature of boiling water. For more reasons than one, people should take especial care not to eat sausages which are underdone in the middle. It is said that pork or ham which has been thoroughly smoked or salted may be safely eaten, even though it has not been cooked.

Several physicians in this country have recorded instances of tapeworms infecting persons who have been addicted to eating meat raw. But the most striking instance is that given by Kaschin, of the Bürater of the Baikal. These people live almost exclusively upon flesh, which they neither properly clean nor thoroughly cook; and they eat from tables that are never washed, and that are also used for cutting up the meat. Even when stationed as Cossacks at Irkutsk, so that many of them had been away from their native country for years, they were infested with tapeworms to such an extent that in 130 autopsies only two bodies were found to be free from the presence of the parasite; often there were several, and once as many as fifteen, in the intestines of the same individual.

The curative treatment.—This consists in the administration of some substance which has the power of killing the creature without injuring its host. At the present time, no drug is used so largely for this purpose as the liquid extract (or "oil") of male fern. The dose is generally said to be from fifteen to thirty minims, but at Guy's Hospital we have been in the habit of giving a drachm or a drachm and a half. Sir William Gull, many years ago, published in the 'Guy's Hospital Reports' (3rd series, vol. i, 1855) a series of 200 cases thus treated with much success. It never does

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any serious harm, but Cobbold speaks of it as causing irregular effects on the nervous system if its dose is too large.

Another useful drug, derived from Abyssinia, is kousso, which consists of the dried flowers of the *Brayera anthelmintica*; from a quarter to half an ounce of this is infused in boiling water, and swallowed, powder and all. Oil of turpentine, again, is often effectual, of which from half an ounce to two ounces may be taken for this purpose; a single large dose is less apt than repeated small doses to cause the strangury which sometimes results from its administration. A decoction of the bark of the pomegranate root is another valuable anthelmintic; the direction is that three or four doses of from one to two ounces each should be given at intervals of about half an hour; it often causes faintness and giddiness.

Whatever medicine may be chosen, it is advisable for the patient to fast for several hours before taking it, although Cobbold objected to this. The intention is that, the alimentary canal being empty, the drug may with more certainty come into contact with the tapeworm. And for the same reason a dose of castor-oil is sometimes given three or four hours before the anthelmintic.

The administration of one of the remedies above mentioned almost always brings away a large portion of the tapeworm, if there be a fully developed strobila in the patient's intestine. But, unfortunately, it very commonly breaks at the neck. The head then remains behind; and as it still retains its vitality, it at once begins to form fresh segments. Now, if the parasite belong to either species of tænia, it is remarkable that after such an accident, an interval of three months (Cobbold says thirteen weeks) is almost invariably found to elapse before proglottides again begin to be passed. The author repeatedly saw this occur on almost the very day which had been predicted. This period of three months corresponds exactly with the length of time which is required for the full development of the tapeworm from a cysticercus; it therefore follows not only that under the influence of anthelmintics the line of fracture is constantly at one part of the worm, but that it is quite close to the head. It no doubt sometimes happens that the creature breaks in the middle, particularly if the dose of the anthelmintic is inadequate. But this, at any rate, may be said,—that if a portion of tapeworm be brought away, in which part of the narrow neck is recognised, and if the patient should in much less than three months begin again to pass proglottis *per anum*, it is certain that more than one tænia is present.

The patient must always be told to look very carefully in his evacuations for the head, the appearance of which should be described to him. An enthusiastic practitioner may himself search for it. Cobbold recommends that the whole mass of fæces should be passed through a sieve. If the head be not discovered, the patient may either wait for three months to learn whether the treatment has been effectual, or he may take a second dose. One would have thought it doubtful whether drugs would act satisfactorily upon a tapeworm of which nothing but the head is left; and it would be obviously very difficult to obtain evidence as to the frequency with which a cure is effected under such circumstances. But Cobbold relates one instance in which, having brought away almost the whole of a tapeworm with one dose of extract of male fern, he gave another dose the next day and actually succeeded in finding the head with its four suckers in the patient's fæces.

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The remaining species of intestinal worms belong to the Nematoidea.*

1. The common round-worm (*Ascaris lumbricoides*), is an almost universal parasite in the human intestine all over the world, and most animals are infested by allied species and genera. In fact the distinction between what is natural and pathological fails here as in so many other particulars. Parasitical animals and plants exist everywhere and by as good a right as their hosts. It is as "natural" for a man to have worms in his bowels as pediculi in his hair, and both entozoa and epizoa are found in quadrumana and all other mammals. The cleanliness of the most civilised communities is only maintained by constant effort, and freedom from external and internal parasites is as "artificial" as freedom of a well-kept garden from weeds.

As its specific name implies, the ascaris is not unlike the common earthworm (*Lumbricus terrestris*) in appearance. When alive it is of a reddish-brown colour with a tinge of yellow; but after its death this colour slightly fades, and it becomes greyish. It has a disagreeable smell, which cannot be removed by washing, and which, according to Leuckart, is due to an odorous principle having its seat in the deeper muscular layers of the body.

Dr Bastian and some others have suffered from irritation of the eyes, sneezing, and other symptoms like those of hay-catarrh from dissecting this worm.

The female is fifteen inches long; the male, which is comparatively seldom met with, measures only ten inches; its circumference is also much less than that of the female. They are both cylindrical in form, tapering at each end, but rather more gradually towards the head than the tail.

The ova are elliptical in form, much larger than those of the tænia, and not unlike the eggs of bothriocephalus in size and shape.

The life-history of this parasite has not yet been completely ascertained. The female discharges ova which certainly do not undergo any development while they are in the human body. After their escape with the fæces, however, an embryo slowly appears in each egg if it be kept in water or in moist earth. Davaine and others formerly supposed that the ova were swallowed in this condition, either in drinking-water or upon uncooked vegetables or fruit, and that their shells having been removed in the stomach, the embryos gradually became developed into full-grown worms. But experiments made for the purpose of confirming this hypothesis have uniformly failed; several German investigators have deliberately swallowed large numbers of ova, and have given them to chidren, but no specimen of the ascaris has hitherto been obtained in this way. Another possibility is that the embryos escape from the ova and enjoy independent existence for a time before they enter the human body. Thus Dr Paterson, of Leith, is quoted by Aitken as having observed that certain families who drank the water of a particular well were very subject to the parasite, whereas towards the other end of the same street families who drank the pure water supplied to the town of Edinburgh were free from it. The well-water came from a dirty pond in the vicinity, and in it numerous minute vermiform animalculæ existed, which perhaps were larval ascarides. But Leuckart lays stress on the fact that the embryos of *Ascaris lumbricoides* show little or no tendency to escape from the ova, and that their organisation is not like that of embryos which are destined to maintain an independent existence. He therefore thinks it

* Sometimes spelt "Nematoda." The word is derived regularly from νῆμα, a thread, and εἶδος, appearance. The group nearly corresponds with the Cœlelminthia, *vers entozoaires cavitaires* of Cuvier.

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most probable that the ova are swallowed by some intermediate host—perhaps a worm or the larva of some insect—and that within the body of this animal the embryos pass through such further changes as may prepare them to undergo their complete development on being afterwards transferred to the intestine of a host.

The *Ascaris lumbricoides* (Linn.) is rare in infants under a year old, although Leuckart refers to one instance in which it occurred in a child of eleven weeks. Children between three and ten years of age afford the most numerous specimens of the round-worm, as was observed by Hippocrates. It is more common in rural districts than in towns, and particularly in low and damp localities. It is met with more often in the autumn than at any other season; this Leuckart connects with the hypothesis that eating summer fruits has in some way a share in introducing it into the human body. Persons who are poor and dirty are more subject to it than those in better circumstances. In the insane it is very common; among thirty lunatics of dirty habits in the Hofheim Asylum there was not one who was free from this parasite. In the Southern States of America, the West Indian islands, Cayenne, and Brazil, the negroes at all ages are, with scarcely an exception, infested with round-worms. It is also much more common in some parts of Europe than in others, particularly so in Finland and Holland.

The *Ascaris lumbricoides* inhabits the small intestine. It may be solitary, or there may be two, three, or any number of them. When numerous they often cohere together in knots, and they have sometimes been found filling almost the whole of the intestines. Children have been known to pass some hundreds of them in the course of a few weeks. Cruveilhier found more than a thousand in the intestine of an idiot.

It is probable that each individual worm remains only a few months within the body of its host. If they pass down into the large intestine they are voided from the anus, either alone or with the fæces. If they make their way upwards into the stomach they are generally vomited. Sometimes one is discharged through the nose; and it has even been known to enter the larynx, and cause death by suffocation. A curious point, to which Cobbold drew attention, is that the *Ascaris lumbricoides* is very apt to insinuate itself into any kind of small ring that may be swallowed by its host, such as the "eye" of a lady's dress or the shank of a button. A single worm has been found with two buttons thus attached to it. This peculiarity may explain the fact that the parasite sometimes makes its way into the bile-duct or gall-bladder, setting up jaundice, or even suppuration in the liver. In other cases it has escaped into the peritoneal cavity through the floor of an intestinal ulcer; in yet others it has been found in an abscess generally situated either at the umbilicus or in the groin. There has been much discussion whether the inflammation has in such instances been originally set up by the presence of the worm; a strong point in favour of this view is that after its escape the abscess has generally been found to heal, and thus to end much more favourably than an ordinary fistulous opening from the bowel.

The *symptoms* produced by these worms vary according to the number of them which are present and the irritability of their host. It is only in a very delicate subject that a single *Ascaris lumbricoides*, or even two or three, would cause any appreciable discomfort. When symptoms arise they are generally such as indicate irritation of the intestinal mucous mem-

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brane, pain in the abdomen (especially in the umbilical region), nausea, foulness of breath, irregularity of appetite, tumidity of the abdomen, and the presence of mucus in the stools. It is true that in the bodies of those who have died from other causes, and in whom worms are found, the intestine does not generally present any morbid appearances that can be attributed to their presence; but Barthez and Rilliet say that they have seen the mucous membrane reddened by vascular injection at points occupied by several round-worms, and not anywhere else.

More serious cases have been recorded of fatal obstruction caused by masses of the parasites rolled up together; some of these are probably not genuine; but Dr Beaven Rake has reported a recent and undoubted case which occurred in a negro child in Trinidad ('Guy's Hospital Gazette,' March 8th, 1890). It is possible that the presence of worms may lead to invagination.

In certain patients the *Ascaris lumbricoides* has given rise to reflex symptoms similar to those which have been described as effects of the presence of tapeworms: dilatation of the pupils, swelling of the eyelids, squinting, irritation of the nostrils, grinding of the teeth during sleep.

Diagnosis.—There are two conditions under which one has to deal with this parasite:—either where a patient, generally a child, presents some of the symptoms just enumerated, which, in the absence of any more obvious cause, may possibly be due to its presence; or, where one ascaris has been vomited or passed *per anum*, and there is a question whether there are still others in the intestine. In the second class of cases one has sometimes to bear in mind that impostors have been known to bring earthworms with them, which they pretend to have passed from the bowels. The true lumbricus, however, is readily distinguished from an ascaris; it is much redder, it tapers less at its extremities, and it has rows of small bristles, which aid it in locomotion; its mouth is a short fissure on the under surface of its rounded head, whereas the mouth of the *Ascaris lumbricoides* is a triangular aperture at the more pointed end of the animal, surrounded by three tubercles or lips.

When the presence of this parasite in the intestines is suspected, a common practice is to give a dose of medicine, on the chance that it may bring away an ascaris. But Leuckart and others have pointed out that the question may very readily be answered by a microscopical examination of the patient's fæces, which, if the worm is there, are sure to be full of its ova. It has been calculated by Eschricht that the female ascaris produces about sixty millions of eggs, and if these were a year in being discharged from its genital passages, the patient's evacuations would contain 160,000 of them every day.* No wonder, therefore, that a single microscopical specimen often places the presence of the parasite beyond dispute. The ova are elliptical in form, measuring $\frac{1}{340}$ of an inch by $\frac{1}{440}$ of an inch; they are of a dirty brownish colour and nodulated on the surface, from the presence of a thick layer of an albuminous substance deposited outside their proper shell. In illustration of the value of this method of diagnosis Dr Ransom gives (in his article on the entozoa in

* The round-worm is estimated to produce in the year some 64,000,000 eggs. Though the egg of an ascaris is extremely minute, its diameter being a twentieth of a millimetre, the total weight of the yearly produce of the eggs comes, according to Leuckart, to a mass 1740 times that of the parent worm. The queen bee only produces 130 times her weight in the form of eggs. . . . To equal the fertility of the ascaris the human female would have to produce about 25,000 children in the year.

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'Reynolds' System of Medicine,' vol. iii, p. 197) the case of a child who was admitted into hospital for abdominal pains and disordered digestion, and because she had passed two round-worms previously. The evacuations contained the eggs of the parasite. Medicines on several occasions brought away one or more specimens of the ascaris, and her symptoms entirely disappeared. But ova were still detected in the stools, and therefore she was kept under treatment three months and a half longer, until seventeen worms in all had been passed. No more of the ova could then be discovered, and she was accordingly sent out of the hospital.

Treatment.—The drug which appears to possess more power than any other in effecting the expulsion of the *Ascaris lumbricoides* is santonine. The dose, for an adult, is from three to six grains twice daily, and for a child one to three grains. While it is being taken, an occasional purgative should also be prescribed. An inconvenience sometimes produced by santonine is a curious temporary disturbance of vision, objects appearing of a yellow, green or blue colour. The urine may also be reddened, but that is of no consequence. Cobbold says that santonine sometimes produces tenesmus, spasms, and even hæmorrhage from the bowels, so that it should not be too long continued. Dr Ransom speaks of *Dolichos pruriens* and oil of turpentine as being also worthy of trial.

As to preventing the ascaris from entering the body, all that can be said is that one should be careful to drink only pure water and to have all solid food thoroughly cooked.

The allied species *Ascaris mystax* has occasionally been observed in the human intestine both by German and British helminthologists.

The *Oxyuris vermicularis* (Bremser), or "thread-worm" (*Germ.* Madenwurm), is very much smaller than the round-worm just described. It was formerly called *Ascaris vermicularis* and in England this name is not quite obsolete, for thread-worms are still commonly called "ascarides." Since *A. lumbricoides* is seldom present in large numbers in a living patient, there is not much risk of confusion in this use of the term, but it is incorrect according to modern zoological classification, and is liable to mislead the student.*

The oxyuris may best be compared to a small piece of white thread. The female measures four tenths of an inch in length, the male one sixth of an inch. The latter is much less often seen and was formerly supposed to be very rare, but now it is supposed that there is about one male to every nine females. They taper towards the tail, as their generic name denotes.

Thread-worms occur only in the large intestine. They live on its contents, and yellow bile pigment can plainly be recognised in their bodies. They are often present in vast numbers, and are found either singly in the mucus lining the interior of the bowel, or matted together with this mucus into little balls.

The eggs of oxyuris are oval in form, and flattened on one side, with a smooth surface and thin shell. They measure $\frac{1}{1100}$ of an inch by $\frac{1}{490}$ of an inch. Unlike those of the *Ascaris lumbricoides*, they contain embryos at the time of their liberation from the parent worm. It might therefore be supposed that no impediment existed to the multiplication of the oxyuris within the human intestine for an indefinite period. But Leuckart and

* Etymologically the word ἀσκαρίς (from ἀσκαρίζειν, to leap) is applicable rather to the thread-worm, which performs brisk movements, than to the comparatively sluggish round-worm.

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other modern observers believe that the ova are incapable of undergoing development until they have passed into the external world, and been swallowed by the same or by another individual. One is at first startled when one is told that every single thread-worm in a child's intestine represents an ovum which the child must have taken into its mouth. But this theory is supported by the analogy of all other parasitic worms, none of which are capable of reproducing themselves indefinitely *in situ*. Indeed, these creatures produce such immense quantities of ova that there would be no limit to their numbers, were not their development subject to some such limitation. Leuckart observes that one never finds *young* oxyurides in numbers bearing any proportion to those of the ova, whereas, if they were developed directly from the eggs, they ought to be far more abundant. He tested the point, as far as he could, by swallowing a few ova and giving some to those of his pupils who volunteered to share in the experiment; at the end of the second week, three out of the four persons experimented on began to pass thread-worms.

This question is one of considerable importance in reference to the measures which should be adopted for preventing the occurrence of oxyuris. This parasite emerges from the rectum of its host, especially at night; it creeps about the anus, and in women often passes into the vagina. It is therefore quite conceivable that it might be conveyed to the anus of another person. Küchenmeister supposed that this was the way in which the worm obtained access to a fresh host.

But modern helminthologists explain the emigration of thread-worms from the rectum as follows. The worms and their ova often become adherent to the skin and hair in the neighbourhood of the anus; they dry up, and ultimately break down into dust, containing enormous numbers of ova still capable of springing into life if brought under suitable conditions. Thus every opportunity is afforded for what may be termed "self-reinfection."

Again, every fæcal evacuation of a person infested with thread-worms probably contains hundreds of thousands of their ova. These must be carried into drinking-water, taken up by flies, deposited upon vegetables and fruits, and so in countless ways gain access to the human alimentary canal.

The *symptoms* produced by thread-worms are not like those caused by other entozoa. They are due to the irritation which they produce by creeping about the anus of their host. Chief among them is a sense of heat and tingling or itching at the fundament. This comes on at a particular time, generally soon after the patient is in bed, but sometimes before he has retired to rest. Marchand quotes the account which a man gave of his own sufferings, as follows:—"Every evening about 5 or 6 o'clock, when I first feel the worms, I become pale and troubled, and sometimes I have even shivered; my companions often notice it; I am restless and obliged to walk about; even if I am at a place of entertainment, I leave instantly and hasten to employ a cold enema; this does not always give me relief, and I am then in torture; I tear my perinæum and scrotum. I am obliged to micturate every instant." Irritability of the bladder is well known to be sometimes caused by the presence of thread-worms, and there is reason to believe that they sometimes excite priapism or nymphomania, and thus lead to the practice of masturbation. Another symptom may be tenesmus; and the fæces may contain a large excess of mucus. Cruveilhier recorded the case of a child who was awakened every night at the same hour by an agonising pain in the anal region, so that he screamed

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and writhed about in bed. The periodicity of the attacks led to the administration of quinine, but with no success, until the part was looked at, when the cause was at once discovered.

The *treatment* is a less easy matter than might be expected; and there is still some uncertainty as to the best way of ridding a patient of thread-worms. Until lately it has been taught that the rectum and sigmoid flexure were the parts of the intestine chiefly infested by this parasite; it was known that it might be found as high as the cæcum, but this was regarded as exceptional. The older writers, therefore, recommend enemata for their removal. Sir Thomas Watson says that he has often relieved patients of thread-worms by prescribing infusion of quassia as an injection. Lime-water, solutions of chloride of sodium, of perchloride of iron, and many other substances have been recommended for the same purpose. The rule has generally been that an enema should be given every third or fourth day for two or three weeks.

Of late, however, the opinion has been gaining ground that the oxyuris occurs in the cæcum and in the upper part of the colon more generally than had been supposed. Cobbold believes that the cæcum is its head-quarters; he therefore recommends active saline cathartics, repeated for several days in succession, and large draughts of infusion of gentian; also an Indian remedy, *Aristolochia bracteata*. He mentions that the introduction of a little mercurial ointment within the verge of the anus, as the patient retires to rest, will effectually prevent thread-worms from creeping out of the rectum, but this plan must require some caution, lest salivation should follow. Relief is sometimes afforded to the itching by the application of oil or of dilute red oxide ointment.

Children, particularly about five or six years old, are much more commonly infested with thread-worms than grown persons, but children are much more easily freed from their presence than adults.

If the modern view of the life-history of the oxyuris be correct, it is most important that scrupulous cleanliness should be maintained in all the surroundings of a person infested with it.

The *Trichocephalus dispar** (Rudolphi), another nematode worm, has its seat in the cæcum. It is remarkable for its very long and thread-like neck, which forms about two thirds of its whole length of one and a half to two inches. It was formerly called *Trichurus* from a mistake between the head and the tail. This parasite appears to give rise to no symptoms; and it has scarcely ever been discovered in the evacuations. It therefore possesses no clinical interest.

As its ova may be found in the fæces, it is well to mention that they are bluntly spindle-shaped, with transparent ends, and that they measure 0·023 mm. in breadth by 0·051 mm. in length, somewhat smaller than those of oxyuris.

The *Sclerostomum duodenale*† (Cobbold) is of far greater importance, although happily it is not found in this country. It is occasionally met with in Italy, where it was discovered in 1838 at Milan by Dubini; and occurs very commonly in Egypt and in Brazil. At a meeting of the Pathological Society

* *Ascaris trichiura* (Linn.), Trichurus (Buttner).—*Germ.* Peitschenwurm.

† Also known as *Ancylostomum duodenale* (Dubini), as *Strongylus* or *Dochmius duodenalis* (Leuckart, Diesing), and as *Str. quadridentatus* (von Siebold).

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in 1867 some specimens of it from the latter country were exhibited by Dr Hermann Weber. (See his paper, with figures, 'Path. Trans.,' xviii, p. 274.)

In Cairo, Bilharz found it in almost every dead body which he examined. It inhabits chiefly the jejunum, generally lying between the valvulæ conniventes, with its mouth firmly fixed in the mucous membrane by means of its four conical chitinous teeth. The female occurs in larger numbers than the male.

The sclerostomum is a small round worm with its head bent nearly at right angles. The male measures nearly half an inch in length; the female seven tenths of an inch. It may be present in enormous numbers, as many as 1250 having been counted in a single patient. It feeds, not upon the intestinal juices like other worms, but upon blood, which fills its digestive canal, and gives its body a red colour. It fixes itself firmly by means of two pairs of teeth (of which the ventral pair is the larger) into the mucous membrane of the duodenum or jejunum; and within its mouth there are two moveable blades, which doubtless serve to incise the tissues.

The spot to which it is attached is indicated by an ecchymosis; and Leuckart thinks that it shifts its position from time to time, and that the punctures which it leaves may go on bleeding. The cavity of the bowel is sometimes found full of blood after the patient's death, although hæmorrhage *per anum* seldom, if ever, occurs during life. The body of the worm commonly hangs free within the gut, protected more or less by the ridges of the mucous membrane; but sometimes it is rolled up in a hollow space in immediate contact with the muscular coat.

The female sclerostomum throws off numerous eggs, which are oval bodies, with a thin, transparent shell, of nearly the same size as those of the oxyuris, but less elongated, measuring only $\frac{1}{20}$ mm. in length. They also differ in having no operculum, and in their yolk being undivided or only just segmented at the time of their expulsion in the fæces, whereas the eggs of the oxyuris already contain embryos; according to Cobbold, however, it is sometimes viviparous.

The life-history of the sclerostomum has not been directly traced, but it is believed to be the same as that of the allied *Dochmius trigonocephalus* of the dog. If this be correct the ova become hatched when they pass into mud or water, and produce slender worms which exhibit active movements. These require no intermediate host, but develop into sexually mature animals when they are swallowed and reach the human alimentary canal. It is not surprising that the parasite should be met with chiefly in hot climates, where men are often compelled to drink water from dirty pools exposed to contamination in every way.

As hundreds and even thousands of these parasites are sometimes present in the same individual, it is not surprising that they should give rise to grave anæmia.

It was Griesinger who showed, in 1854, that the form of anæmia known as Egyptian chlorosis was due to the presence of this parasite ('Arch. f. phys. Heilkunde,' xiii, 557). Wucherer, of Bahia, found in 1886 that the parasite gives rise to a similar complaint in Brazil. It occurs in the Comoro Islands, and Dr Strachan has reported cases from Jamaica ('Brit. Med. Journ.,' June 27th, 1885). One would have expected that the disease would sometimes have been seen in England in persons recently arrived from Egypt, just as a somewhat analogous affection, due to the bilharzia, is often brought to this country from South Africa.

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The sclerostomum was the cause of severe epidemic anæmia among the workmen in the St Gotthard Tunnel in 1880 (see a paper by Dr Bugnion, 'Brit. Med. Journ.,' March, 1881, p. 882). One of these cases came under the care of Prof. Bäumler, of Freiburg.

The resulting anæmia may prove fatal in a few weeks, or it may run on for years, unless death occurs by dysentery. At first the patient appears well nourished, but at length he becomes wasted and dropsical. The only special symptoms mentioned are disorders of digestion, and a cutting pain in the abdomen.

The other symptoms of this "tropical anæmia" appear to be identical with those of other forms of anæmia. They may last for years, until the patient is at length carried off by diarrhœa or pneumonia, or some other accidental malady. Much more rarely the chlorosis proves directly fatal after causing dropsy.

Microscopically the eggs may be recognised by diluting the fæces with water, stirring, and allowing them to settle. They are, however, much like those of oxyuris—rather more slender in shape. The worms always remain fixed in the intestine.

Discovery of the ova is the only way of distinguishing anæmia thus produced from other kinds of symptomatic anæmia, and from the severest form described by Addison as idiopathic.

The drugs which have been used in the *treatment* of this disease are chiefly oil of male fern, santonine, oil of turpentine, assafœtida, aloes, and iron; but Dr Weber says that none of them have proved to be permanently successful. If the patient can be removed to another climate, and placed under favourable conditions, recovery may take place.

Strongylus (*Eustrongylus*) *gigas** is a very large nematoid worm inhabiting the pelvis of the kidney in certain Carnivora and other mammals. It is not very uncommon in dogs and wolves, in the coatimundi (*Nasua*), racoon, otter, and seal, and has also, it is said, been met with in horses and oxen. It is excessively rare in man. Most recorded cases are spurious, and refer to ascaris or to fibrinous clots passed *per urethram*. Küchenmeister quotes fourteen cases, but only those of Grotius (1595), Ruysch, and Blasius, and a more recent one of Moublet, seem to be authentic. Diesing can only adduce three clear cases. A specimen in the Hunterian Museum is said to be taken from the human kidney. This species is the largest nematoid entozoon known. The male grows to a length of ten inches, and the female to thrice that length. One huge strongylus of this species was found free in the abdominal cavity of one of the Esquimaux dogs which McClintock took on his arctic expedition in search of Sir John Franklin. It was sent from Greenland to Steenstrup at Copenhagen, and by him given to Leuckart. The account of the strongylus given by this anatomist is based on that and only two other specimens, one from a coati, and the other from an American mink (*Mustela vison*).

An allied species (*Strongylus armatus*) produces aneurysms in the mesenteric arteries after it has wandered from the cæcum or colon, in horses and asses.

Trichina spiralis † (Owen).—This nematode is truly an intestinal worm,

* Strongylus (στρογγύλος), round.
† Hair-worm—θρίξ, τριχός, a hair.

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although until recently it was only known as being now and then found unexpectedly in the muscles of the human body. How it found its way into the muscles was for a long time a puzzle which exercised the minds of the ablest zoologists; but, as we shall presently see, its life-history is not altogether unlike that of the other nematodes which infest the human intestine. The principal difference is that, instead of its ova being discharged through the rectum of the host, they develop within the parent worm into young trichinæ, which, as soon as they are born, begin an active migration among the tissues of their host.

Tiedemann, in the year 1822, was the earliest observer who took note of the presence of a number of white stony concretions in the human muscles, but he did not describe them further, and Leuckart doubts whether these were trichina capsules. In this country Hilton was the first, in 1832, to record the fact that he had met with them.* He could not determine their precise nature, but thought that they were probably very small cysticerci. Next Sir James Paget, then a student of St Bartholomew's, independently described them. Two years later Owen showed that they were hollow capsules, and that each of them had coiled up within it a minute nematoid worm, to which he gave the name of *Trichina spiralis*, and fully described its anatomy.

The capsules themselves are just visible to the naked eye; they measure $\frac{1}{78}$ inch in length, and $\frac{1}{180}$ inch in breadth. They are lemon-shaped, and their long diameter is always in a line with the muscular fibres among which they lie. They occur in all striped muscles, even in the tensor tympani, but are most numerous in the diaphragm and the muscles of the trunk. The heart is scarcely ever affected, and in non-striated muscles they are never met with, so that the œsophagus contains them only in its upper part. They feel gritty when touched with a knife; this is due to the deposition of calcareous matter, which sometimes renders them altogether opaque, but is usually present only in their extremities, leaving the middle transparent, so that under the microscope the little worm in the interior is at once visible. There is an excellent description of the trichina with figures by Dr Bristowe and the late Mr Rainey, in the 'Pathological Transactions' for 1854; and good plates are given in Cobbold's 'Entozoa.'

Not only rats and swine (the usual bearers of the disease to man), but cats and dogs, hedgehogs and moles, and herbivorous animals, as horses, calves, rabbits, guinea-pigs, may be infected. The muscles of birds remain free from invasion though the worms multiply in their intestines; and trichinæ do not breed in cold-blooded animals.

The earliest experiments which threw light upon the way in which this remarkable entozoon gains access to the human body were those of Herbst, who in 1851 showed that when the flesh of animals containing trichinæ was given to other animals their flesh in turn became infested with the parasite. He did not trace the intermediate steps by which this result is brought about, and afterwards observers were for a time led astray by Küchenmeister's hypothesis that the trichina was a stage in the development of *Trichocephalus dispar*. In the spring of 1860 Virchow and Leuckart showed that this was not the case.† They fed animals with trichinous

* 'London Medical Gazette,' 1833, p. 605. Owen's paper is in the 'Zool. Trans.,' vol. i, p. 315. The oldest preparation of trichinæ is one of the sterno-hyoid muscle in the Guy's museum, No. 1361[30]. It was put up by Mr H. Peacock in 1828.

† Leuckart ('Die menschlichen Parasiten,' vol. ii, p. 525) recalls the early observation

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meat, and found that the worms at once escaped from their capsules and developed into sexually mature entozoa of a kind that had never before been recognised.

The experiment has since been repeated by many observers, and with uniform results.

For example, an animal to which trichinous flesh has been given is killed at the end of forty-eight hours; the mucus lining its alimentary canal is found to contain numbers of minute living worms already sexually mature. They are not visible to the naked eye, but they are easily recognised under the microscope. The majority of them are females; these measure one twelfth to one ninth of an inch in length (2—3 mm.). The males are smaller, being only one eighteenth to one fourteenth of an inch long (1·2—1·6 mm.); they are further distinguished from the females by having two conical projections from the caudal extremity. If the animal be left until the sixth day before it is killed the female worms contain free embryos, which are bent and closely packed side by side in the uterus. These may even be watched under the microscope as they become extruded from the vagina and afterwards move about the field. It is estimated that at least 150 young worms are produced by each female trichina.

It is therefore clear that from the sixth or seventh day after the ingestion of trichinous flesh by man or any other animal, living trichina-embryos are poured in enormous numbers into the alimentary canal. These at once begin to bore their way through the coats of the intestine. They enter the peritoneal and other serous cavities, the lymph-glands, the viscera, and above all the muscles. Observers are not yet altogether agreed as to the way in which the trichina-embryos reach distant parts of the body. Leuckart and Virchow suppose that they make their way along the connective-tissue spaces. But the rapid migration of the parasite to the most distant parts of the body is strong evidence that the embryos enter the blood-stream, and ultimately reach the tissues through the walls of the systemic capillaries. However this may be, it is certain that the voluntary muscles are the only parts in which they find the conditions necessary for their further development. If they enter other tissues they either perish or migrate again until they reach their proper seat. Even when they have reached the muscles they seem at first to move on in the course of the fibres, for they are found in larger numbers towards their tendinous insertions than elsewhere, as though these formed obstacles arresting their further progress. They have sometimes been distinctly seen within the sarcolemma of the primitive fibres, and Leuckart asserts that this is always their position, but others maintain that they more commonly lie between the fibres. When once lodged in the muscles they rapidly increase in size, and, though at first structureless and undistinguishable from minute filariæ of different species, they gradually acquire a distinct alimentary canal, and rudimentary sexual organs. At this time they roll themselves up, and round each of them a capsule is developed. This is first to be recognised about the fourth week after their immigration. It consists originally of a nucleated transparent material, the product of the irritation of the tissues which their presence causes. After a time calcification begins in it. Dr Thudichum says that in rabbits he has seen the capsules perfectly opaque within ten weeks. But in the human subject a high degree of calcification

of trichina in a case of "pneumonia and pericarditis with muscular rheumatism," *i. e.* of trichiniasis, at Bristol, by Dr Wood, in 1835 ('Lond. Med. Gaz.,' p. 190).

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does not occur in less than a year, and Rupprecht found capsules still transparent in the muscles of a man who had had an attack of trichiniasis two years before.

Calcification of the capsule does not necessarily interfere with the life of the trichina within. The parasite remains quiescent, waiting for the death of its host to call it into activity. But in course of time it may itself perish; it then becomes converted into an almost structureless mass, which under slight pressure breaks up into fragments.

The number of trichinæ which may be contained in the muscles of the human body is enormous. From data obtained in experiments on animals it has been estimated at from twenty to thirty millions. This, however, applies to cases which would perhaps always terminate fatally before the worms would have time to become encapsuled.

The entrance of trichina-embryos into a muscle produces certain changes in its fibres which have yet to be mentioned. They lose their striation, become brittle and homogeneous, and show numerous minute fissures. To the naked eye the muscular substance appears of a pale reddish-grey colour.

Trichiniasis.—At the time when Leuckart and Virchow were working out experimentally the life-history of the trichina, Zenker had just observed a case in which the parasite caused a fatal illness in the human subject. On January 12th, 1860, a girl was admitted into the Dresden Hospital suffering from what at first appeared to be fever. She died, and on *post-mortem* examination the characteristic lesions of enteric fever were absent, but the muscles were full of living trichinæ as yet unencapsuled. The girl had been in the service of a butcher, who had killed a pig about a week before her illness commenced. She had been employed in making sausages of the pork, and had very likely eaten some of it in an uncooked state. The sausages and a ham from the pig were examined and found to contain trichinæ. It was also ascertained that the butcher and two other persons had been taken ill about the same time, but had recovered.

Symptoms.—Since the publication of Zenker's case several others have been recorded, so that the clinical aspect of trichiniasis is now well known. At first it is undistinguishable from those of other febrile diseases. The patient complains of loss of appetite, sleeplessness, and a sense of extreme lassitude and depression, and it may be of nausea and vomiting, but at the end of a week, or a little later, the arms and legs begin to be stiff and painful. The elbows and knees become flexed, and great pain is produced by any attempt to straighten them. After a time the limbs are sometimes rigidly extended, and the body is as though affected with opisthotonos. The muscles are tender to the touch; when grasped they feel hard and swollen, and as if they were distending the fasciæ in which they are enclosed. The jaws are often closed for several weeks, after which the muscles may become suddenly relaxed again, with an audible crack. Movements of the eyes are painful, no doubt from the presence of trichinæ in the recti and obliqui, but the power of accommodation is lost at the same time, and this is less easy of explanation. The breathing become shallow and hurried from implication of the thoracic muscles; and coughing, sneezing, and yawning may be almost impossible.

About the end of the second week the eyelids are observed to be œdematous, and sometimes the rest of the face and even the neck. Afterwards the legs and the parts round the joints become swollen.

The fever is not generally high in trichiniasis; the temperature seldom

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rises above 102° Fahr. There is often profuse sweating, and a miliary eruption may develop itself. The pulse is sometimes very rapid. The tongue is red, slightly furred, and rather dry. The bowels are sometimes constipated; but diarrhœa may occur, especially in severe cases.

Fatal cases usually terminate in the fourth or fifth week, but sometimes much earlier. The immediate cause of death may be exhaustion, or pneumonia, or ulceration of the colon. If the patient should recover, convalescence is tardy, lasting three or four months.

Diagnosis.—Well-marked cases are not difficult to recognise; for the symptoms, taken together, are unlike those of any other disease. The diagnosis has several times been established by the microscopical examination of a minute portion of muscle removed during the patient's life. Another way of verifying it is to search the fæces for adult worms. Rupprecht found numbers of them in the stools of patients to whom large doses of calomel had been given.

Often the discovery of the disease is facilitated by the fact that it occurs epidemically. Thus, at Plauen, in 1862, thirty persons were attacked about the same time. At Hettstädt, near the Hartz Mountains, four separate outbreaks of trichiniasis occurred between September, 1861, and March, 1864; in the most important of these 158 persons were attacked, of whom twenty-eight died. Other epidemics have been observed at Stassfurt, Dessau, Leipzig, and elsewhere. Of all these, details may be found in a paper by Dr Thudichum which appeared in 1864 in the seventh Report of the Medical Officer to the Privy Council.

In England the only cases of trichiniasis which have been detected during life, or attended with symptoms (since Wood's in 1835) are those recorded in 1871 by Mr Dickinson, of Workington, in Cumberland. The patients were a farmer's wife, his daughter, and a serving man. They had all been eating sausages and pork from one of the farmer's home-fed pigs, the flesh of which was subsequently found by Dr Cobbold to be full of trichinæ. It has been supposed that cases in which symptoms are produced by the migration of trichina-embryos into the muscles do really occur from time to time in this country, but are overlooked.* Indeed, the fact that encapsuled trichinæ are now and then found in the bodies of those who have died of other complaints, shows that the conditions which are necessary for the entrance of this parasite into the human body are not entirely absent from English life. But there is reason to believe that even in this form the parasite is rarely met with here as compared with its frequency in Germany. In Dresden, Zenker detected it in four out of 136 *post-mortem* examinations; and in Berlin Virchow found it six times in the course of a single year. These observers mention that often a very small number of trichinæ only were present, so that they might easily have been overlooked. Probably in such cases the symptoms, if any, would be very slight, merely such as might be attributed to a "rheumatic affection." It is tolerably certain that, as a severe or fatal disease, trichiniasis has not yet occurred in any London hospital—at least since the publication of Zenker's case. Even on the Continent the disease is far more common in Northern Germany than anywhere else, the most common of all in Saxony. In districts where pork is not eaten raw it is rare.

* The outbreak of a febrile disease among the boys on the "Cornwall," related by Mr Power ('Lancet,' vol. i, 1880), was at first ascribed to trichiniasis, but this was afterwards found to be absent.

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The severity of a case of trichiniasis appears to depend upon the number of embryos which penetrate into the muscles. Thus the most important guide to *prognosis* is believed to be the state of the patient's limbs, as regards mobility and pain on movement.

Treatment.—The first indication is to expel as many of the trichinæ as possible from the alimentary canal. For this purpose castor oil is recommended, or calomel in twenty-grain doses, repeated at intervals. Experience is said to have shown that the latter medicine gives relief to the symptoms. Friedreich at one time proposed the picrate of potash; he gave it in one case which terminated in recovery; but live trichinæ were afterwards found in this patient's muscles. Glycerine is said to kill the trichinæ when directly applied (probably by abstracting water), and it has therefore been administered in cases of trichiniasis in the United States, and, it is reported, with success.

The *prevention* of trichiniasis resolves itself into two distinct questions. First, how can animals intended for human food be preserved from the parasite? The answer to this is that they must be kept out of the way of trichinous flesh. It is indeed conceivable that adult living trichinæ (a few of which are known to be sometimes passed in the fæces of animals in whose intestines the worm is undergoing development) might enter the alimentary canal of another animal, and that their young might migrate into its muscles. But such an explanation goes a very little way towards accounting for the numerous encapsuled trichinæ which are often found in pigs; and they doubtless derive them from the filth which they devour, particularly perhaps when rats infest the sty, for rats are the most frequent trichinæ-bearers of all animals. The muscles of hedgehogs, moles, and pigeons are known to be often infested with the parasite; and it has been shown to retain its vitality even after the flesh containing it has become putrid.

Secondly, if flesh containing trichinæ should by accident be used for human food, all danger is obviated if the meat is thoroughly cooked.

FILARIA SANGUINIS.*—In 1866 Wucherer detected, in cases of chylous urine occurring in Brazil, certain minute living organisms, evidently the embryos of a nematoid worm. Six years later, in 1872, the late Dr T. R. Lewis, in India, discovered similar embryos in the blood. It was very soon found that this was no isolated occurrence, and that the hæmatozoon (as it was called) was by no means limited to persons affected with chyluria. In South China, for instance, among 1000 natives taken at random, about 100 are said to be infested with this parasite. In that country similar larval entozoa (as of *Filaria immitis*) are very commonly seen in the blood of dogs and in many species of birds, so that their presence in man excited the less surprise in the minds of experienced helminthologists. Among those persons who harbour the *Filaria sanguinis hominis* (as Lewis termed it) some appear to be in perfect health, but others are affected with one or more of a limited number of diseases, of which the chief are lymphangitis with varicosity of the lymph-channels in the inguinal glands, lymph-scrotum, elephantiasis of the scrotum or of the leg, and chyluria.

The next step was the discovery of the parent worm from which the embryos found in the blood are derived. This was effected in 1876 by Dr Bancroft, of Brisbane, in Australia. He first obtained a dead specimen from

* The thread-worm (*filum*, a thread).

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a lymphatic abscess in the arm, and afterwards four living ones from a hydrocele of the spermatic cord. These he sent to England to Dr Cobbold, who gave to the entozoon the name of *Filaria Bancrofti*. They were all females, and as yet no perfect specimen of a male seems to have been found. The length of the female is from three to three and a half inches; its breadth from $\frac{1}{100}''$ to $\frac{1}{90}''$. It has a circular mouth, destitute of papillæ, a narrow neck, and a bluntly pointed tail. Its body is smooth, and of an opaline appearance, and it has been described by Dr Manson, of Amoy, in China, as looking "like a delicate thread of catgut, animated and wriggling." This observer, in 1880, while operating on a case of lymph-scrotum, removed at the same time a portion of a living worm, and showed that it lay in the interior of a dilated lymphatic. He also proved that the parasite is naturally viviparous, for he saw fully formed embryos, exactly like those which are found in the blood, escaping from the animal's vagina. Strictly speaking, indeed, the accuracy of this last statement is open to question. For it has been known from the first that the embryo in the blood is always enclosed in a delicate sac or sheath, which fits it accurately, except that a collapsed or unoccupied part is seen projecting behind either the head or the tail, according to the direction in which the worm happens to be moving; and it seems now to be certain that this sheath is nothing else than the envelope or shell of the ovum, which, as the embryo develops, yields before it, and so continues to be stretched out over the skin. Still, the fact remains—and it is one of which we shall presently see the importance—that the parent filaria, instead of throwing off ordinary oval and motionless eggs, gives birth to active organisms, capable from the first of vigorous spontaneous movements. The size of these embryos is such as not at all to interfere with their traversing the lymph-paths through any glands that may come in their way, and so passing on from the lymphatic vessel in which the parent worm lies into the thoracic duct, and beyond this into the blood-vessels. Their diameter, indeed, is only about $\frac{1}{8200}''$, not more than that of the leucocytes which circulate through the lymph-glands; their length is about $\frac{1}{90}''$.

So long as the embryos of the filaria remain in the blood they continue to be of the same size, and show no indication of undergoing further structural development. This fact is of itself sufficient to suggest to anyone acquainted with helminthology the idea that they are waiting to be transferred to some other host; and it seems to have occurred both to Dr Bancroft at Brisbane, and to Dr Manson at Amoy, independently of one another, that this host might probably be some species of mosquito which feeds on human blood. Dr Manson remarks that the limitation of the parasite to certain parts of the earth's surface was almost sufficient to exclude from his consideration many blood-sucking animals, such as fleas, lice, bugs, and leeches, which are found pretty well everywhere. He therefore came to the conclusion, in 1877, that it was likely to be either a mosquito or the sand-fly that took the embryo filaria from its human host, and supplied to it the conditions requisite for its development into a more mature form. Had he been at that time aware of another extraordinary fact that he afterwards discovered, he might safely have set aside the sand-fly. This fact is that, instead of the young filariæ being found in the blood throughout the whole twenty-four hours, none of them can generally be detected in it during the day, even when they are abundantly present in it during the night. At about 6 or 8 p.m. they begin to make their appearance; by midnight their

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numbers reach the maximum; as morning approaches they become fewer and fewer; by 8 or 9 a.m. they cease to be discoverable. What becomes of them in the interval is not at present known, but it must be borne in mind that all that has yet been proved is that the capillary blood-vessels of the integument contain them at night, but not in the daytime. There seems to be no necessity for supposing that they circulate with the blood like its normal constituents, the red discs and leucocytes. Possibly during one part of the twenty-four hours they may be all collected in the pulmonary capillaries, or in those of the deeper structures generally; but when the patient retires to rest they may betake themselves to the vessels of the skin. In a case which Dr Stephen Mackenzie has recorded in the 'Pathological Transactions' for 1882 observations at intervals of three hours were made for weeks together, and the periodicity was found to be as complete as it possibly could be. Dr Mackenzie also submitted his patient to the experiment of having his habits of life reversed, so that for nearly three weeks he remained out of bed all night, and rested in the daytime, the hours of his meals being arranged accordingly; the result was that during this time the filariæ were found in the blood during the day, but not at all, or only in much smaller numbers, during the night. Obviously, therefore, that which determines their migrations is the resting or moving condition of their human host. Moreover, they were not tempted to come out during the day by even the thickest London fog. But it is nevertheless impossible not to recognise the fact that their usual habit of entering the capillaries of the integument at night-time is precisely adapted to bring them within reach of the proboscis of a nocturnal blood-sucker like the mosquito. In Dr Mackenzie's case the blood seems always to have been taken from the patient's finger; as much of it was examined as would lie beneath a five-eighths inch cover-glass. In this quantity of blood at midnight there were often fifty or sixty, and sometimes eighty or ninety filariæ. Dr Mackenzie therefore calculated that from thirty-six to forty millions of them were probably present in the whole mass of circulating fluid; but it is obvious that this estimate is enormously too high if it is only the capillary vessels of the skin that contain them in abundance.

Dr Manson lost no time in verifying his hypothesis with regard to the mosquito. He persuaded a Chinaman, known to be infested with the filaria, to sleep in a "mosquito house." Next morning the gorged insects were caught and examined. The blood in their stomachs was found to contain filariæ in even larger numbers than that of the man from whom it had been derived. It is supposed that they become entangled by their lashes in the proboscis of the mosquito, and are so removed from the blood-vessels. Having reached their new host, some of them proceed to enter upon a process of development. They lose their sheaths, grow to the length of one thirtieth of an inch, and acquire a distinct alimentary canal, a mouth crowned with three or four nipple-like papillæ, and rudiments of generative organs. Their movements also become extremely active. These changes are completed in from four to six days. During this time the mosquito has been digesting her single meal and maturing her own ova; she now deposits them on the surface of water, after which she dies, and probably falls into the water on which her eggs are laid. The progress of the filaria has not been directly traced further; but there can be little doubt that it makes its way out of the body of the dead insect into the water. And in all probability the next step is that it is swallowed by a human being, from

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whose stomach it bores a passage into the thoracic duct or into some lymphatic vessel; and along this it then works up stream, in obedience to some strange instinct, until it reaches a spot which it takes for its permanent abode. Here we must suppose that it is joined by another parasite of the opposite sex, after which it proceeds to furnish in the lymph-channels and to the blood-current of its host those swarms of larvæ which formed the starting-point of our inquiry as to the life-history of the entozoon. How long the parent worm lives we do not as yet know; but a case observed by Dr Manson shows that it may be at least as long as thirty-two years; he found living filariæ in the blood of a man aged fifty, who had had lymph-scrotum from the age of eighteen. There is some reason for supposing that it may be killed by the occurrence of severe acute disease in the host. At any rate Dr Stephen Mackenzie's patient was attacked with rigors as the result of going out of the hospital on a cold and windy day in October, and after the following day no embryos were ever discoverable in his blood. Pleurisy set in, and an abscess formed near the left collar-bone; and when he died two and a half months later no trace of the worm could be found. Dr Mackenzie supposes that it became dislodged during the rigor, and reaching the termination of the thoracic duct on the left side of the neck, excited both the pectoral abscess and also the pleurisy. However this may be, it seems clearly to have perished from an early period of the man's fatal illness, and its body must be supposed to have undergone disintegration.

It now becomes an important question to determine how it is that the filaria produces injury of the lymphatic vessels. The only hypothesis that can be said to account for this result is one that has been formulated by Dr Manson, in a paper in the 'Pathological Transactions' for 1882. His idea is that so long as the discharge of embryos goes on after the manner above described, the parasite is perfectly innocuous to its host. But from some cause or other it happens in certain cases that instead of the larval filariæ enclosed in their sheaths, ova in a much earlier stage of development, with unstretched shells, are extruded from the maternal vagina. Dr Manson has twice obtained such ova from the lymphatics; and probably they have been found in the urine also. Now, according to Dr Manson, they measure $\frac{1}{150}''$ in breadth by $\frac{1}{500}''$ in length; according to Dr Cobbold, $\frac{1}{160}''$ by $\frac{1}{1000}''$. In either case their transverse diameter is far greater than that of the embryos; and nothing is more likely than that they should fail to pass along channels which the embryos would find no difficulty in traversing. Dr Manson supposes, for instance, that when they are carried by the lymph-stream to a gland they become impacted in the small channels formed by the afferent vessel. The necessary result must be, as anastomosing paths become one after another obstructed, a more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, and in the scrotum.

The pathological effects of the filaria will be described hereafter in the chapter on affections of the urine and in that on elephantiasis.

The Guinea-worm (*Dracunculus medinensis**) is an enormously long nematoid worm of which the female only is known. It inhabits the subcutaneous tissue, usually of the legs, to which it gains access from the

* The specific name refers to their frequent occurrence in Medina and other parts of Arabia (cf. Plutarch, 'Sympos.,' viii, 9).

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water-tanks in India, Egypt, the coast of Guinea, and other hot countries. Its effects are purely those of local irritation.

Two trematode worms have been found in the human body. Of these, the liver-fluke (*Fasciola hepatica* or *Distomum hepaticum**) infests the gall-passages, but it is very rarely found in man, at least in civilised Europe—it is said to be more common in Bosnia. In sheep it causes a common disease, the sheep-rot.

The other (*Bilharzia hæmatobia*) inhabits the pelvic veins of persons living in Egypt and Natal, and produces serious hæmaturia. Its ova are passed out in the urine, and are recognised by a pointed process at one end, or in some cases by a second spike projecting from the side of the egg. The parasite will again be noticed in the chapter on Hæmaturia. It was described by Griesinger, who named it after Bilharz, its discoverer in Cairo in 1855.

An acanthocephalous or thorn-headed worm, *Echinorhynchus*, sp., has only once been certainly discovered in the human intestine by Lambl ('Prager Vierteljahrschrift,' Feb., 1849). A second case, reported from Netley in 1872, is doubtful. It is common among pigs in England.

* *Fasciola*, dim. of *fascia*, band or tape. *Distomum*, a name given under the strange mistake that the two suckers are mouths.

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DISEASES OF THE PERITONEUM

Ἢν δὲ ὕδωρ ἅλις ἐς τὸ περιτόναιον ἐμπεριέχηται, ἐμπλώῃ δὲ τῷ ὑγρῷ τὰ ἔντερα, καλέομεν ἐπίκλησιν ἀσκίτην.

ARETÆUS: *De Morbis Chron.*, lib. II, cap. i.

ACUTE PERITONITIS—*Importance—Clinical symptoms—Origin: secondary to visceral disease—puerperal—uræmic—extreme rarity of idiopathic acute peritonitis—Morbid anatomy—Local peritonitis and circumscribed abscess—Diagnosis—Treatment: medical and operative—Prognosis.*

CHRONIC PERITONITIS—*Peritoneal adhesions—Thickening, general and local—liver, spleen, omentum—Locular and general effusion—Frequency—Causes.*

TUBERCLE OF THE PERITONEUM—*Anatomy—Symptoms—Relation to phthisis and to tabes mesenterica—Diagnosis—Prognosis—Treatment.*

CANCER OF THE PERITONEUM—*Commonly secondary—Anatomy—Symptoms.*

ASCITES—*Its physical signs—its diagnosis—its causes, inflammatory and obstructive—its prognosis—Treatment by drugs and by paracentesis abdominis.*

THE peritoneum is part of the great body-cavity (*cœlom*) formed by the mesoblast splitting into somatopleure and splanchnopleure. It is a huge areolar space or lymph-sac, and its most intimate pathological relations are not with skin or mucous membranes—not even with the joints or the so-called arachnoid space—but with the pleura, pericardium, and tunica vaginalis.

The diseases of these three divisions of the same original cavity are the same: acute inflammation, serous or purulent, traumatic or septic; chronic adhesive inflammation with hypertrophy; chronic irritative effusion, and passive dropsical effusion—hydrothorax, hydropericardium, and ascites. All three serous membranes are liable to be invaded by tubercle, and also by cancer. All three are prone to follow the pathological fate of the viscera which they cover; they are all apt to suffer in the course of Bright's disease; and lastly, they are all affected together often by inflammation or tubercle, and more rarely by cancer.

On the other hand, rheumatism seldom or never affects the peritoneum as it does the pericardium, and peritonitis is seldom or never the result of exposure to cold, as pleurisy undoubtedly is. Although peritonitis is not so constant a companion of inflammation in any abdominal viscus as is pleurisy of pneumonia, yet its most frequent cause is undoubtedly the presence of irritating products of inflammation from one or other of the organs it covers, the effect ranging from the adhesions which slowly form about an irritated ovary to the rapid and violent inflammation which blazes up when pus or fæcal material finds its way into the cavity.

The most important viscera from this point of view are, for men patients, the intestines and the stomach, next the liver and gall-bladder, and then the bladder. In the case of women the ovaries, Fallopian tubes, and uterus set up peritonitis more frequently than any other viscera.

We may divide peritonitis as we see it clinically into the following varieties:

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1. Acute, septic, suppurative, "malignant," affecting the whole cavity of the abdomen: always secondary to perforation or to some septic process, as in puerperal fever after childbirth.

2. Acute, sero-fibrinous, or sero-purulent, but local and circumscribed: traumatic, or secondary to visceral inflammation or to m. Brightii.

3. Chronic, adhesive, local, hypertrophic.

4. Chronic, serous, sometimes latent.

5. Tubercular, with adhesions and effusion, serous or sero-purulent, occasionally hæmorrhagic.

6. Cancerous, with adhesions, thickening, and effusion, usually of blood-stained serum.

ACUTE PERITONITIS.—This disease is exceedingly fatal; cases in which it was the immediate cause of death make up a large proportion of the total mortality from disease and injury, at least in hospital practice. Thus in 1873, taking a year at hazard, of 434 inspections made at Guy's Hospital, in at least 52 death was directly attributable to acute peritonitis, or nearly one in eight.

Symptoms.—These vary greatly in different cases, and they are often combined with and masked by those of other affections, so as to make recognition peculiarly difficult.

If we take the case of an apparently healthy person, who is suddenly seized with peritonitis, we shall find him lying in bed on his back, with his knees drawn up, his features pinched and drawn, his eyes sunken and dark. There is no sweat, and his face is pale, but its watchful, anxious look is like that of a patient with rheumatic fever.

He complains of sharp, sometimes cutting or burning pain in the abdomen. This is constant, but liable to aggravation if he changes his posture, if he coughs or sneezes or strains, and also when there is movement of gas in his intestines. He dreads pressure on the abdomen—sometimes the lightest possible application of the hand. Tenderness may either be diffused equally over the whole surface, or may be most intense at some particular spot, probably the starting-point of the inflammation. The movements of the diaphragm cause so much pain, that in breathing the patient instinctively uses the upper ribs only; the inspirations are therefore shallow, and are repeated forty or fifty times a minute.

An attack of peritonitis often begins with sharp rigors. These are followed by more or less fever. The *temperature* may rise to 104° or to 105°; but it is important to remember that a normal temperature is no guarantee that peritonitis, and even purulent peritonitis, is not present. We found the same exception in cases of pleurisy, and even of empyema. When death is approaching, the temperature falls to normal, or below it, and the hands and feet are icy cold. The *pulse* is frequent, ranging from 100 to 150. At first it is often small, hard, and strong—the wiry pulse. In the later stages it becomes still smaller, feeble, compressible, irregular, or imperceptible—the thready pulse.

In fatal cases death usually occurs by collapse, the mind often remaining clear to the last moment. But there may be great restlessness towards the end, the patient tossing about in delirium for an hour or two before death, and careless of the posture he assumes.

The fact that the inflammation penetrates to the subserous and muscular coats may be one reason why there is *constipation* in peritonitis. The bowels

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can, however, be moved either by purgatives or enemata, if this dangerous mistake is committed; and Dr. Fordyce Barker states that in puerperal peritonitis diarrhœa is more frequent than constipation.

Vomiting is among the earliest symptoms of peritonitis. The *tongue* is small, slightly furred, and dry. When the case is approaching a fatal termination the patient is sometimes tormented by obstinate hiccough; micturition is often painful and difficult, particularly in pelvic peritonitis.

The surface of the abdomen is not only tender to the touch, but also much harder than natural by a conservative reflex action, which interferes with physical examination, but is of great diagnostic import. After a time it is distended, sometimes enormously, with gas which accumulates in the paralysed bowels—a condition called *tympanites* and *meteorismus* by the Greek writers.

The recti and other muscles are rigid, and the semilunar and transverse markings may be plainly discerned through the integuments.

At first the percussion-note is everywhere tympanitic, usually of a higher note than in health, but after a time it may become short and tympanitically dull from extreme distension. Sometimes we can detect one or more circumscribed regions of dulness, which points to local effusion, and occasionally is accompanied by fluctuation. A friction-sound like that of pleurisy may be detected by the stethoscope occasionally, but seldom in acute general peritonitis. Fluctuation points to the case becoming chronic, and an audible rub to its being local.

Ætiology.—Acute peritonitis, as above stated, is most commonly caused by extension from a *viscus*—as a rule a hollow viscus. For the cavities in the abdomen, besides being liable to undergo perforation and to discharge their contents into the serous cavity, are also more subject than the solid viscera to those septic forms of inflammation which, when they reach the peritoneal surface, excite the same unhealthy action there. Thus cirrhosis of the liver does not set up acute general peritonitis, nor does chronic inflammation of the ovaries, nor the swollen spleen of ague or of enteric fever; but a ruptured hepatic abscess, a perforated gall-bladder, a sloughing enteric ulcer, a perforated appendix cæci, a ruptured bladder or ovarian cyst, or inflammation of the uterus and Fallopian tubes after delivery or abortion—these are almost certain to produce acute and fatal peritonitis. A sloughing block in the spleen (the result of ulcerative endocarditis) may occasionally set it up; in three instances noted in the *post-mortem* records at Guy's Hospital its starting-point was suppurative inflammation of the kidney; and other exceptional causes are malignant tumours, or, still more rarely, tubercular lymph-glands.

The perforating ulcer of the stomach is a frequent cause of general peritonitis. A perforating intestinal ulcer will do the same; even tuberculous ulcers, which are rightly said to be much less likely to perforate than those of enteric fever, have done so in several cases at Guy's Hospital. Hernia and intestinal strangulation give rise to peritonitis by extension of inflammation; but chronic obstruction (even fæcal impaction) of the bowels may also produce fatal peritonitis by ulceration and perforation of the dilated part of the gut above the seat of obstruction. Lastly must be mentioned surgical operations on the abdominal organs as occasions of peritonitis, although this result is rendered far less likely by strict antiseptic precautions.

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Typhlitis and pelvic suppuration in women are probably the most frequent of all causes of secondary peritonitis.

In some of these varieties of peritonitis the cause is obvious; in others it may be utterly obscure, the patient being apparently in perfect health until he is attacked by acute inflammation of the whole abdomen. This is frequently the case when the starting-point of the disease is a perforating ulcer of the stomach or duodenum; and even when it is a typhoid ulcer of the ileum, the occurrence of peritonitis may be the first indication of illness, for, as we have seen, enteric fever may be entirely latent. The most important of these obscure causes of peritonitis is typhlitis.

In most, if not in all cases, *puerperal peritonitis* starts from an unhealthy inflammation of the lining membrane of the uterus, which reaches the peritoneum either along the Fallopian tubes or through the tissue of the organ, the venous channels in which are often filled with pus. Miscarriages, again, are not rarely followed by peritonitis; and it may also be set up by extra-uterine fœtation, a pelvic hæmatocele, suppuration of a Fallopian tube, or sloughing of an ovary. The most careful vaginal examination should therefore be made in every case of peritonitis the cause of which is not evident.

We cannot always trace acute peritonitis to a primary local disease. Sir Thomas Watson, in common with many of the older writers, gave "exposure to cold" as one of its causes; but the pathology of the dead-house lends no support to such an opinion. Dr Fordyce Barker, indeed, speaks confidently of having seen puerperal peritonitis caused by a chill; and shows clearly that in some cases no inflammation can be detected in the uterus or the neighbouring organs. But it is surely more probable that there is an undiscovered local cause or some specific affection than that these cases of peritonitis differ from all others in causation.

Smallpox, typhoid fever, gout, rheumatic fever, glanders, pyæmia, erysipelas, have all been at one time or another regarded as causes of acute peritonitis. But the evidence is more or less doubtful.*

Of general predisposing causes of acute peritonitis, Bright's disease is the only one well ascertained. Sixteen cases of this kind were observed in Guy's Hospital between the years 1854 and 1872. The inflammation was generally suppurative; and there was often a marked absence of vascular injection of the serous membrane. The kidneys were, as a rule, enlarged, and in a more or less advanced stage of tubal nephritis; but in three cases they were contracted and granular.

During twenty years only two cases of acute peritonitis were recorded at

* Dr Wilks has recorded a case in which this disease occurred as part of an erysipelas that had started from an ulcer in the groin; but probably the inflammation extended through the parietes, as it sometimes does after a severe burn.

As regards pyæmia, the only case that I know of in which it seemed to have given rise directly to peritonitis was that of a man who died in less than an hour with cerebral symptoms, and in whom the only lesions found after death were softness of the spleen, and the presence of about six ounces of pus in the lower part of the peritoneal cavity, with a smaller quantity of pus in the left knee-joint. As no history of the case could be obtained, I had no means of knowing whether he had suffered from any symptoms of illness previously; but it was certain that he had been giving evidence in a court of law just before he was attacked with a fit, which rapidly passed into fatal coma. Instances of ordinary pyæmia are constantly presenting themselves in the *post-mortem* room; and I do not know of one in which acute peritonitis did not start from some local lesion. I believe, therefore, that at the bedside it is needless to think of pyæmia (apart from abdominal abscess) as one of the possible causes of peritonitis.—C. H. F.

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Guy's Hospital which could not be attributed either to renal disease or to extension from an inflamed tubercular or cancerous viscus.

In 1874 several children at a school at Wandsworth were attacked at the same time with acute peritonitis. The late Dr Anstie investigated this epidemic; and the conclusion at which he arrived was that the disease was caused by exposure to the influence of sewer gas. It was in making a *post-mortem* examination in one of the fatal cases that he received the wound in his finger which cost his valuable life. Dr Shirley Murphy has since met with the following case, which appeared to be attributable to a similar cause. A woman, aged thirty-six, died on her way to the Homerton Fever Hospital. The autopsy showed that acute peritonitis was the cause of death. The coils of intestine were matted together by lymph, but the intestines, uterus, and other viscera were healthy, and no local starting-point for the inflammation could be discovered. It was afterwards ascertained that the patient had been living in a house the drain-pipe of which was obstructed, so that for two or three weeks the sewage had been spread over the yard. When first taken ill she shivered, fainted, and vomited; next day she complained of pain in the left iliac fossa, with purging; and two days later she died.

Anatomy.—The changes which occur in the peritoneum under inflammation present considerable variations, but rather of degree than kind. They are essentially the same as in other serous membranes. The surface first becomes reddened, from injection of the minute vessels. This injection is often not uniform, but is especially marked along two longitudinal lines, which run over the bowel, at a little distance from one another, parallel with the attachment of the mesentery. The explanation of this appears to be as follows:—In health atmospheric pressure keeps every part of the serous surface in contact with some other part; the intestines are not (as one is apt to suppose) regularly rounded tubes; they are flattened against one another and the abdominal wall. But the distension caused by peritonitis leads them, by physical necessity, to assume a cylindrical form; and the result is that blood is forcibly drawn into the angular spaces between them. The red lines so produced were therefore called "suction-lines" by Dr Moxon. They are wanting when the intestines fail to become distended, when air has access to the peritoneal cavity, and perhaps also when inflammatory effusion is poured out very early and in large quantity.

The further morbid appearances vary according as the inflammation tends rather to the effusion of lymph or to suppuration. In the former case the membrane becomes dull and lustreless, and very soon it presents shreds and small patches of fibrin; in the latter case it is even more lustrous than in health, and feels greasy to the touch.

The inflammation in some cases does not go beyond the formation of lymph. This forms a layer of greater or less thickness, which may either be limited to certain parts or cover the whole surface of the serous membrane. Microscopically it consists of fibrinous threads which cross one another in all directions, leaving interspaces in which are masses of leucocytes.

When inflammation of moderate intensity extends to a serous membrane from limited areas of disease in a subjacent organ, the corresponding parts of its surface become covered with local patches of fibrin. This is the case even when the affected parts are on the sloping sides of the lungs or liver, from which any fluid must at once gravitate away. The minute observations of Rindfleisch, Ranvier, and Klein prove that some of the cells

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are derived by proliferation from the endothelium; others are exuded leucocytes. Klein, in his 'Anatomy of the Lymphatic System,' describes the lesser omentum and mesentery being œdematous and swollen to five times their normal thickness in animals in which he had set up artificial peritonitis. The occurrence of effusion into the subserous tissues accounts for the well-known fact that after death from peritonitis the serous membrane can be stripped off much more readily than when it is healthy.

Whenever the inflammation goes beyond a certain degree of intensity, fluid is also effused into the peritoneal cavity. This is more or less turbid, and under the microscope exhibits numerous leucocytes. When they are sufficiently abundant to give to the liquid a milky colour, this is said to be purulent. Thus every gradation may occur between the purely serous exudation and pure pus. All depends upon the proportion of leucocytes to serum.

If the peritonitis remains at a lower degree of intensity it leads to adhesion of the opposed surfaces of the serous membrane. The active agents by which this is brought about appear to be the cells embedded in the fibrin. Some pass into spindle-cells, and ultimately form perfect connective tissue; while others develop into blood-vessels, the walls of which are at first exceedingly soft, consisting entirely of opposed cells. These readily give way if the exudation-fibrin is subjected to pressure or traction by the movements of the organ beneath. Spots of hæmorrhage are consequently often seen, and sometimes the amount of blood effused is very great. Writers have described a hæmorrhagic form of peritonitis which appears to arise in this way.

The adhesions resulting from peritonitis may be universal, the cavity being obliterated and the abdominal organs united together by connective tissue, from which they have to be dissected out when a *post-mortem* examination is made. More frequently, perhaps, the opposed surfaces adhere in certain places only. They still move on one another as in health; and thus the tissue which connects them becomes stretched into bands or cords which may acquire a considerable length. These, as we have seen, are frequent causes of intestinal obstruction (p. 257).

Even when the inflammation has gone on to the effusion of a large quantity of fluid, the possibility of its terminating in adhesion is by no means excluded. The fluid may be absorbed, and the two surfaces may then come together and unite. The connecting fibres seem then to be formed from the cells of a layer of granulation tissue, which covers each surface, and is derived from the endothelium of the serous membrane. Even pus may dry up and become converted into a caseous mass embedded in the fibrous tissue of the adhesions.

Circumscribed peritonitis.—In most cases of peritonitis the inflammation starts from some one spot and diffuses itself over the whole abdomen. This process is doubtless much accelerated by the movements of the intestines, so that parts already inflamed are brought into contact with others which had not hitherto been reached by the disease. When the stomach or intestine has been perforated, the extravasated matters may be carried to the most distant parts of the cavity.

But the inflammation does not always thus spread over the whole of the peritoneum. The omentum often seems to check its progress towards the organs situated above it. Even when pus is poured out, it may be limited by the agglutination of the two serous surfaces round the space which it

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occupies. Thus peritonitis starting from the uterus may lead to a circumscribed abscess occupying the pelvis and more or less of the lower part of the abdomen. In one case of this kind the pus was discharged through the bladder during life, and in another through the umbilicus. So also peritonitis arising from ulceration of the intestine often gives rise to localised collections of pus. This is especially apt to occur when the cæcum is the starting-point of the disease. The abscess then forms a swelling in the right iliac fossa. It sometimes points near the crest of the ilium, but not infrequently it passes down below Poupart's ligament and discharges in the groin. In other cases, again, it makes its way backwards towards the loin. Such abscesses are sometimes difficult to distinguish from those caused by diseased bone, and the difficulty is increased by the fact that when the crest of the ilium lies in the way of the pus, part of it sometimes becomes denuded of its periosteum, so as to be within easy reach of a probe.

The bowels sometimes communicate freely with an abscess of this kind, and much fæcal matter may be discharged with the pus. In one case for a considerable time before the patient's death almost all the fæces passed through an opening in the groin; in that instance the abscess was secondary to cancerous disease of the cæcum. The fact, however, that the pus discharged from a circumscribed abscess in the abdomen has a fæcal odour does not prove that there is a communication with the intestine. Matter collected in the neighbourhood of the bowels may acquire such an odour as a result of the diffusion of the intestinal gases. Again, when there is an opening, this does not invariably prove that the abscess started from the ulcer; for the intestine may be perforated from outside by a suppurating gland.

Another form of circumscribed abscess in the peritoneal cavity is limited to the sac of the lesser omentum. In one such case this cavity contained two or three pints of pus, the inflammation having started from disease of the pancreas. Such cases are exceedingly rare. On the other hand, abscesses limited to one or other hypochondrium are by no means uncommon. In the 'Guy's Hospital Reports' for 1873–4 several cases of this kind were recorded by the author. In some of them the abscess started from an ulcer of the stomach, or other disease in the neighbourhood, but in most it resulted from some direct injury, particularly when seated in the right hypochondrium. In cases of the latter kind the suppuration is often preceded by a circumscribed effusion of blood, which may itself form a distinct swelling, analogous to the pelvic affection known as peri-uterine hæmatocele.

In one of the most interesting of these cases the patient had been kicked in the left side, and came in with a large rounded tumour in the hypochondrium. After a time we found that air had entered it, for curious musical sounds, synchronous with the heart's beats, were heard over it, and the percussion-note became tympanitic. Yet there were no symptoms indicative of constitutional disturbance, and the man left the hospital, refusing to believe that anything serious was the matter with him. Some time afterwards he returned, saying that he had vomited a quantity of matter, and that the tumour had disappeared; on examination no trace of it could be discovered. Cases of abscess in the hypochondrium, however, do not often terminate so favourably. This combination of circumscribed peritonitis with fluid and gas was described by Dr G. H. Barlow in the 'Medical Gazette' for 1845, Dr Wilks, then a student, reporting the case. Prof. Leyden, of Berlin, has

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since described it as "subphrenic pyopneumothorax." (See a case by Dr Coupland, 'Brit. Med. Journ.,' March 23, 1889.)

Diagnosis.—This may be considered under three heads.

(1) When a person, previously supposed to be well, is suddenly seized with pain in the abdomen, it may be far from easy to determine whether the attack is peritonitis, or colic or hysteria.

The chief distinction is found in manipulation of the abdomen. In *colic*, pressure and friction give relief; the belly is usually hard and contracted; the pain intermits from time to time, so that the patient has intervals of complete ease; and when his sufferings are at their worst he is restless, and tosses about in search of relief. In *hysteria*, on the other hand, there often appears to be the most extreme tenderness of the surface; but if the patient's attention be diverted no further complaints are made, and after a time considerable pressure is perfectly well borne, while the abdominal walls become soft and supple. The exaggerated susceptibility and sensitiveness to the lightest touch are in such cases the very symptoms that show the absence of serious disease. One must inquire whether the patient has ever had hysterical attacks or amenorrhœa; but we must not forget that acute peritonitis from perforating ulcer of the stomach occurs in anæmic young women who are very likely to have had hysterical symptoms. It is possible that the thermometer may sometimes show a rise of temperature in cases of mere hysteria,; and, on the other hand, pyrexia may be absent in peritonitis.

The probability that the pain is due to colic is of course greater if the patient has eaten any indigestible food, or if the gums present the dotted lead-line.

In all doubtful cases one must remember that to attribute to peritonitis a pain really due to colic or hysteria is an error free from serious consequences, whereas the converse mistake may be fatal to the patient; and a few hours' delay will always solve the question.

The rupture of a concealed aneurysm into the subperitoneal tissues is another possible cause of sudden severe pain that must not be overlooked, particularly if the patient fainted when the attack began, or was pulseless from the first. It is sometimes impossible to distinguish this from perforation of the stomach or intestine, and the same applies to hæmorrhage from the Fallopian tubes.

(2) When it is clear that peritonitis is present or impending, diagnosis is not complete. The *cause* remains to be discovered. Among the very numerous affections that may give rise to inflammation of the peritoneum comparatively few are likely to be latent. Hence, when a person supposed to be healthy is attacked, the range of possible, or at least of probable causes is not very extensive. Perforating ulcer of the stomach or duodenum, perforating typhoid ulcer of the ileum, disease of the cæcal appendix, and pelvic disease in the case of a woman, are the chief. Now, a perforating ulcer of the stomach or small intestine is commonly fatal in a few hours, or in a day or two at the latest; while it may be still more rapid. The 'Pathological Transactions' contain reports of one case in which the patient died within an hour, and of another in which the subject of the disease, an Oxford professor, fell down dead while walking in the streets of London. Hence when peritonitis runs a more protracted course than this in a male patient (or even in a woman, if the ovaries, Fallopian tubes, and uterus can be proved to be healthy) there is a very strong presumption that it started

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from the cæcal appendix; and, if recovery should take place, a hard mass can often be discovered in the position of the cæcum.

(3) There is also a *negative* side of the difficulty in the diagnosis of peritonitis; *e. g.* acute suppurative peritonitis, starting from disease of the vermiform appendix, may run its course without characteristic symptoms. Such instances are doubtless very rare, but under other circumstances it not uncommonly happens that peritonitis remains altogether latent. In enteric fever, for instance, all physicians know that perforation of the intestine is often found in the *post-mortem* room to have taken place some hours or days before death, although there had been neither increased pain nor tenderness in the abdomen, nor, indeed, any marked aggravation of the symptoms, to suggest that the fatal issue was being thus brought about. The cause of this is generally supposed to be that patients suffering from fever have their senses and intelligence stupefied. But the truth is that peritonitis remains latent in persons whose minds are clear to the last. Thus in making a *post-mortem* examination after an operation for hernia, ovariotomy, or the like, we have repeatedly found universal peritonitis when those who had watched the patient most closely had detected no evidence of it during life.

In any case in which symptoms of intestinal obstruction have been present for a few days one can never assert positively that inflammation of the peritoneum has not already set in. But then it is to be observed that the symptoms of ileus scarcely differ in kind from those of peritonitis; the principal distinction is the fact that the constipation is insuperable in the one case but not in the other.

Again, in the majority of cases in which acute peritonitis is set up by Bright's disease, its presence is first discovered in the deadhouse, the patient having at most complained of pain in the abdomen such as might have arisen from some trifling cause.

In latent forms of peritonitis the effused fluid is generally pure pus. In this connection it may be noted that some of the older writers describe the pain in peritonitis as subsiding when free suppuration has taken place; and they add that it is important for the practitioner to be aware of this, lest he should commit the error of supposing that the patient is about to recover when in reality his death is surely approaching. The most trustworthy guides to a prognosis in peritonitis are, they say, the aspect of the patient and the state of the pulse. The more frequent its beats, the greater the danger.

Treatment in acute peritonitis must to a great extent be varied according to the conditions under which the disease arises. When it is the last in a series of morbid changes that tend irresistibly to destroy the patient, there is nothing to be gained by active interference. Often little can be done beyond the relief of pain by the application of light poultices or cloths wrung out of hot water to the abdomen, and the administration of morphia by subcutaneous injection.

The case is very different when the disease is set up by a perforating ulcer of the vermiform appendix, or by any similar local affection. In the whole range of therapeutics there is nothing more important than the treatment of peritonitis of this kind. It is not saying too much to assert that a single error in conducting such a case may at almost any period be the immediate cause of death; and, on the other hand, that skilful and judicious management is often the direct means of saving life. *Physiological rest* to the inflamed parts is the one thing essential. The patient must be kept in bed

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from the moment that the existence of peritonitis is suspected, and must maintain the recumbent position most scrupulously, being forbidden to sit up for any purpose whatever. A pillow may be placed beneath the knees to support the thighs in a flexed position. No purgatives of any kind should be administered or allowed to be given, even though the bowels should remain closed for many days; in most cases not even an enema. The importance of this rule may be made apparent by quoting a remark of Dr Habershon's that in cases of this kind he has at the *post-mortem* examination seen castor oil floating on the contents of the abdominal cavity.

Whether nourishment and medicines should be given by the mouth depends upon the cause to which the peritonitis seems to be attributable. When we suspect that it is due to perforating gastric ulcer the stomach must of course be kept empty. A most striking instance of success from what may be termed the "starvation" plan of treatment was many years ago recorded by Dr Hughes in the 'Guy's Hospital Reports' (2nd series, vol. iv, p. 332). A young woman became collapsed, and was seized with severe pain in the stomach. The last food which she had taken was a little gruel, four hours before; for some days previously she had eaten almost nothing. She sent for the late Mr Ray of Dulwich, who (instead of giving her brandy and castor oil) administered twenty minims of tincture of opium in a little water. She rallied somewhat, and was carefully removed to the hospital. She was there ordered half a grain of opium in a pill every three hours, and to have nothing whatever to drink except two measured teaspoonfuls of toast and water. After two days she complained much of thirst. An enema of five ounces of strong, tepid beef-tea was therefore administered, with five minims of laudanum. This was afterwards repeated three times a day. She was also allowed to suck one teaspoonful of beef-tea jelly, instead of the toast and water. It was not until the ninth day that she was permitted to have two table-spoonfuls of strong mutton broth. She completely recovered, and was discharged from the hospital. Nearly four months afterwards, having been so foolish as to indulge largely in cherries and gooseberries, she was attacked with the same symptoms as before. She had brandy and water given to her, and died in nineteen hours. An ulcer in the stomach was found, which had become torn away from a thick layer of old lymph by which it had before been closed. In its neighbourhood there were old vascular adhesions. It seems almost certain that in this case perforation of the stomach occurred during the first attack as well as the second, and that she would have died on the former occasion under less skilful treatment.

In a case of the same kind it would perhaps be well to allow the patient to have nutrient enemata from the first, so as to assuage the thirst; and to administer the opium by the rectum or subcutaneously as morphia. Perhaps it is right to let the patient suck ice broken up into small pieces (ice *pills*, as the Germans call them), but he must take them very slowly, so as hardly to make more frequent efforts to swallow than he ordinarily makes to get rid of the saliva.

Unless these precautions be adopted, perforation of a gastric ulcer, even if it should happily have taken place at a time when the stomach was empty, terminates fatally in a few hours, or at latest within a day or two. If, therefore, one is called to a case of peritonitis which has already run on for some days, one may, in the absence of direct evidence, commonly conclude that it is due to disease of some other viscus; and very small quantities of

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milk, beef-tea, and the like may be allowed at intervals, as the stomach may be able to bear them.

Opium should be administered freely in all cases of peritonitis. This practice was first introduced by Dr Graves, of Dublin, who, in 1822, ordered it in very large doses, to relieve the agony experienced by a woman in whom inflammation had set in after the operation for tapping. Her case seemed hopeless; but to his great astonishment she recovered. Dr Graves, however, used also to give calomel, which at the present day is generally believed to be unadvisable. Two grains of opium should be given at first, and afterwards one grain every two or three hours, the action of the drug being of course carefully watched. There is great tolerance of this remedy in cases of peritonitis. A lad, who had probably never swallowed a dose of opium before, once took as much as twelve grains daily, without being made sleepy or having a furred tongue, and without his pupils being in any way affected by the drug. When the disease subsides the greatest caution should be exercised in discontinuing the remedy. In one case the bowels began to act regularly every day, while the patient was still taking a grain of opium every two hours throughout the day and night.

In many cases a few leeches may be applied to the most painful part of the abdomen with advantage; the pain is often much relieved by them. Warm fomentations or large poultices should be used constantly, and changed as often as they cool. When there is much meteorismus, great relief is often afforded by a flannel, wrung out of boiling water, and sprinkled with turpentine. A long tube introduced into the rectum, and cautiously pushed upwards, has sometimes been known to afford escape to a large quantity of gas from the colon; but it much more often fails.

If death should seem to be impending from tympanitic distension, it may be necessary to puncture the intestine, with a very fine trocar, through the parietes, but this procedure is attended with more risk than in cases of mere mechanical obstruction, because the coats of the bowel when inflamed lose their elasticity, so that the hole made by the trocar is apt to gape after its removal, and may allow fæcal matter to escape.

When the distended and immoveable abdomen, the small and rapid pulse, and the other symptoms above enumerated show that general and acute peritonitis is already present, the ill-success of all treatment, even that by full doses of opium, to do more than procure an easy death, has led to attempts in other directions; and the experience of surgeons in performing ovariotomy and other operations involving the peritoneum has encouraged the bold procedure of opening the abdomen, washing out the products of inflammation, and putting in a drainage-tube. This plan of treatment has been carried out in several cases, sometimes with temporary relief, sometimes hastening death, but sometimes undoubtedly saving an otherwise forfeited life.* In a case under the writer's care in 1889 of general peritonitis dependent on typhilitis in a youth of twenty, this operation was performed by Mr Jacobson with relief to the symptoms, and but for the recklessness of the patient might probably have saved his life.

When we have reason to believe that the peritonitis is septic and puru-

* See, for example, an important paper by Mr Thos. Smith in the 'St Barth. Hosp. Reports' for 1873 (vol. ix), one by Sir Jos. Lister in the 'Lancet' for 1881 (vol. ii, p. 863), and cases brought before the Royal Medical and Chirurgical Society in 1885, by Mr Howard Marsh and Mr Treves, and before the Clinical Society in 1887, by Dr Knaggs and Dr K. Clarke, of Huddersfield.

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lent, but not due to perforation, it would probably be always good practice to draw off the effusion, wash out the cavity, and drain it.

Prognosis.—It still remains that something should be said with regard to the prognosis of acute peritonitis, which, however, could not precede the consideration of its treatment. The intensely dangerous character of this disease is manifest. Sometimes death is inevitable, particularly when the inflammation is set up by perforation of an ulcer in a stomach containing a considerable quantity of food; and when perforation occurs in enteric fever recovery is doubtless exceedingly rare, although instances of it have been recorded (vol. i, p. 147). But that form of peritonitis which is set up by ulceration of the cæcal appendix is, when properly treated, far less dangerous than is supposed, if only its nature is correctly diagnosed, and if it is treated according to the rules laid down above, with no purgatives or enemata (p. 222). Nor of late years have any fatal cases occurred in Guy's Hospital, except such as died very shortly after admission. Hence one may give a favourable though a guarded prognosis in cases of typhlitis, even when symptoms of diffused peritonitis are present.

Simple Chronic Peritonitis.—*Its anatomy.*—This is very different from the acute affection in its causes, pathology, and results, and the one seldom or never ends in the other. The whole surface of the peritoneum is thickened and opaque. Adhesions often exist between different parts, so that the liver, spleen, and stomach may be united into a single mass by firm connective tissue, and may be closely adherent to the diaphragm and abdominal parietes. The omentum is frequently drawn up, and its folds inextricably blended together; so that, with the fat which it contains, it forms a solid mass, binding the colon to the stomach, and capable of simulating a tumour during life. The intestines may be fixed to the front wall of the abdomen; indeed, the entire peritoneal cavity may be closed by adhesions in cases of this kind. Far more commonly, however, the small intestines, if adherent at all, are so only among themselves, and they are then collected in a more or less rounded mass in front of the spine. Sometimes the membrane which unites the several coils can be stripped off, leaving the intestines still covered with a serous coat. This tendency to the formation of adventitious membranes, looking like thickenings of the peritoneal covering of the viscera, but really laid over them, may be seen in all parts of the abdominal cavity. Such a "reduplication" of the capsule of the liver is one of the most remarkable features of the affection known as perihepatitis; and perihepatitis, besides occurring as an independent disease, forms part of very many cases of chronic peritonitis.

The newly formed membranes may further form adhesions among themselves, dividing the general cavity into a number of *separate chambers*, each containing fluid. A very remarkable instance of this occurred in Guy's Hospital in 1860. A woman, aged forty-four, was sent to the hospital, supposed to be suffering from cystic disease of the ovaries. The physician under whose care she came doubted this, and thought that there was fluid in the peritoneal cavity. After some weeks she died. At the autopsy it appeared at first as though the original diagnosis had been correct. Nothing could be seen but a mass of cysts covering the intestines, the stomach, and the liver. Presently, however, it was seen that these cysts had been formed, not in the ovary, but in the peritoneal cavity. Several of them lay between coils of intestine, and some contained a fluid of milky appearance, from the admixture

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of chyle. Another very similar case came not long ago under the author's care. The abdomen contained a considerable quantity of fluid; and this would have been regarded as passive ascites (caused by the heart disease for which the patient was admitted) had it not been that the physical signs were in some respects anomalous. After death the peritoneal sac was found to be divided into a number of distinct chambers by adhesions; one of them was above the transverse colon, another occupied the middle of the abdomen, a third filled the right loin.

In the great majority of cases of chronic peritonitis, however, the small intestines are not compressed by an adventitious membrane, nor even adherent among themselves. Their coils are still capable of moving on one another, and their mesentery is fan shaped. But the mesentery is remarkably shortened; it may measure not more than about two inches from the spine to the attached edge of the bowel, which is thus closely tethered to the back of the abdomen, instead of floating freely. Moreover, the length of the bowel itself is greatly diminished. It may not be more than a few feet long from the duodenum to the cæcum; so that the mucous membrane of the ileum is thrown into folds, resembling the valvulæ conniventes of the jejunum. Its diameter is no less contracted, so that it may hardly admit the little finger. The muscular coat of the bowel is generally thin, but that of the stomach is sometimes much thickened, so that it resembles an india-rubber bottle more than anything else (cf. p. 202).

In the great majority of cases of chronic peritonitis a more or less transparent straw-coloured fluid is effused into the abdominal cavity. Even when no liquid is found after death, it has probably been present at a former stage of the disease. Sometimes, instead of being pale, the liquid is darkened by the presence of blood; sometimes it contains flakes of lymph or even pus; but the latter, when present, is generally the product of an acute inflammation, supervening upon the chronic disease as a result of paracentesis. The surface of the peritoneum, besides being thickened, is opaque. But opacity is not a proof that chronic peritonitis has existed; in cases of dropsy the peritoneum generally, perhaps always, looks white and opaque. This has been regarded as a cadaveric change, due to imbibition by the dead tissues, but it may also be due to the action of the fluid upon the serous membrane during life. In a large proportion of cases of chronic peritonitis, however, the peritoneum is not white, but blackened, or of a slaty colour, from effused blood. This appearance is often particularly marked over the intestines. When there are no adhesions, such a condition of the peritoneum is exactly like that of passive effusion (ascites) during life, and is only distinguished after death by the thickening of the membrane.

Age.—Simple chronic peritonitis is very far from being a rare disease. In Guy's Hospital there is on an average one case of this kind to two of ascites from cirrhosis of the liver, the most common of the local causes of ascites. Of thirty-four cases, eighteen occured in males, sixteen in females. There was in these cases a very wide range in the age of the patients. Between twenty and thirty there were almost as many cases as between thirty and forty, or between forty and fifty; several patients were more than sixty years old, and one had passed the age of seventy. It appears to be about equally frequent in both sexes.

Origin.—Sometimes we can trace chronic peritonitis to what may be called a subacute attack. The peritoneum fills with serum, with little or no pain or febrile symptoms. In these cases it may be removed by diuretics or

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by tapping, and not return again, although more often it goes on to the ordinary chronic ascites with thickened peritoneum. In other cases the process is insidious from the first.

In a remarkable case we had several years ago in Guy's Hospital, the writer saw the origin of the disease in a healthy country lad of fifteen, and its slow increase, until at last it proved fatal, after nearly two years. Here there was a similar chronic effusion into both pleuræ and into the pericardium, with enormous thickening of all the serous membranes, including the tunica vaginalis, with which an open inguinal canal communicated. There was no trace of tubercle found *post mortem*, and the viscera, including the kidneys, were perfectly healthy. It was an example of a concomitant affection of the peritoneum, pleura, and pericardium, comparable to those of tuberculosis of the serous membranes which will presently be described.

Chronic peritonitis is very seldom distinctly traceable to any one of the subjacent viscera; at least only three such cases are recorded in our *post-mortem* books: in one it was believed to have started from the cæcum; and in two from old pelvic cellulitis, which itself in one of them arose from morbus coxæ. Most frequently, perhaps, perihepatitis is its origin. Like that affection, it commonly occurs in patients who have Bright's disease, which therefore may perhaps be regarded as its principal cause. Many patients affected with chronic peritonitis have been intemperate, some have suffered from plumbism, some have had gout, and others suffer from disease of the heart.

The principal *symptom* of chronic peritonitis is the presence of fluid in the abdominal cavity. This can most conveniently be discussed further on, together with its diagnosis, prognosis, and treatment, under the head of ascites (*infra*, p. 323).

TUBERCULOUS PERITONITIS.—In this disease the peritoneum is covered with minute grains, which, however, are seldom uniformly distributed over its surface, but are much more numerous in some parts than others, especially on the under surface of the diaphragm and in the flanks. The serous surface of the intestines is sometimes comparatively free. The omentum often contains a large quantity of yellow cheesy material, or of recent tubercle, and it is drawn up into a flattened mass, which may be as much as two or three inches thick, lying below the stomach and over the colon. The abdominal cavity is often found to contain a considerable quantity of turbid serum or pus; but more frequently it is closed by adhesions, or there are merely a few scattered collections of liquid here and there between the viscera.

Sometimes the intestines are firmly matted together, and tuberculous ulcers perforate so as to form communications at several points. In one case of peritonitis in a child under the writer's care in 1890 an abscess appeared, which was opened and formed an intestino-cutaneous fistula.

Tuberculous affections of other parts are commonly associated with tuberculous peritonitis. Thus, in women, the Fallopian tubes are almost always affected; they are much enlarged, lined with a thick caseous layer, and very often contain pus. Sometimes the same condition is present also in the cavity of the uterus. Dr Moxon believed that the disease spreads into the open mouths of the tubes from the serous surface; he has observed that the tuberculous change is often limited to the ends furthest from the uterus. In men the epididymis or testis (on one side or both) is sometimes

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the seat of tuberculous mischief. When this can be made out during life it has afforded great help in the diagnosis.

Other serous membranes often become affected in the same way as the peritoneum. Thus one or both of the pleural cavities may contain a considerable quantity of fluid, or they may be covered with tubercles and closed by adhesions; and in at least two cases tuberculous pericarditis existed, attended with the effusion of a large quantity of pus (cf. vol. i, p. 948). The intestines often show tuberculous ulcers. In seven cases out of nine the lungs contained tubercles, but it very seldom happens that pulmonary disease is present in such a form as to be capable of recognition during life.

Symptoms.—Tuberculous peritonitis is sometimes acute, but never so rapid or severe as that which follows perforation or septic poisoning; and it is sometimes chronic, but seldom so free from symptoms and insidious in its course as the ascitic peritonitis just described. Subacute is the adjective which best qualifies its symptoms and its progress. The first symptoms are often very vague and obscure. The patient becomes out of health and loses flesh. He complains of pains in different parts of the abdomen. He may have diarrhœa, particularly if the intestines are ulcerated. The abdomen may be tender and harder than natural, and it may feel hot. Very often it is rather retracted than enlarged, but sometimes it is tumid, and there may even be marked fluctuation, and other indications of the presence of fluid in considerable quantity. Clinically, ascites is more often detected than might be supposed from experience in the deadhouse, for at an early stage of the disease the peritoneum frequently contains fluid, which is absorbed in its further progress.

A well-marked case in a child is recognised at once. The swollen abdomen, more or less resonant but with patches of dulness, and sometimes with lumps to be felt, the diminutive thorax covered only with skin, the wasted limbs, pinched features, and fretful cry, make up a characteristic picture.

Age and sex.—Tubercle of the peritoneum is one of the common diseases of childhood; and when associated, as it often is, with tubercular ulceration of the bowels and secondary tubercle of the mesenteric lymph-glands it assumes the familiar clinical aspect of *tabes mesenterica*. But it would be a great mistake to suppose that tuberculous peritonitis is only a disease of early life. In twenty-eight successive fatal cases at Guy's Hospital, two patients were under ten years of age, six between ten and twenty, eight between twenty and thirty, five between thirty and forty, three between forty and fifty, and four over fifty. The disease is more than twice as common in men as in women. Of the twenty-eight cases only eight were in females; and in all of those above the age of puberty, with one exception, there was coexistent disease of the Fallopian tubes.

Diagnosis.—In the account just given of the symptoms of tubercular peritonitis there is little to distinguish it from other forms of chronic and subacute abdominal disease. Great assistance, therefore, is often afforded by the induration of the omentum, which may be felt as a rounded tumour running more or less obliquely across the abdomen above the umbilicus (p. 316). It has been mistaken for the edge of the liver, depressed and rounded by thickening of its capsule; but a resonant percussion-note can be elicited *above* the mass, where, if it were an hepatic tumour, there must have been absolute dulness. Another sign of tubercular peritonitis is the existence of inflam-

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mation and thickening, and even of erysipelatous redness, round the umbilicus. This may sometimes result from adhesion of the small intestine to the abdominal wall at this spot, for in two cases a fæcal fistula resulted. More commonly, perhaps, it is caused by an extension of the inflammation of the parietal peritoneum to the surface along the track of the obliterated umbilical vessels, just as we shall see that cancerous disease is often propagated. In some of those rare cases of strumous peritonitis in which the abdomen becomes distended with pus the umbilicus gives way, and allows the fluid to escape.

The diagnosis of tubercular peritonitis may be confirmed by the discovery of coincident effusion into one of the pleural cavities, or into the pericardium.

Lastly, whenever we suspect tubercular peritonitis in a female patient, we must not forget how constantly this disease is associated with tubercular disease of the pelvic organs. One patient had amenorrhœa for eighteen months, another had menorrhagia, a third had a miscarriage a month before her abdomen began to enlarge, a fourth had one period which lasted a fortnight, and in which the flow was excessive; she then missed her next period, and from that date her abdomen began to swell and her fatal illness commenced.

Prognosis.—The clinical recognition of tuberculous peritonitis is the more important because the disease has by no means so decided a tendency to terminate fatally as might be supposed. We have had several instances in which there was reason to believe that recovery from it took place, and in one case the diagnosis was afterwards proved to be correct by a *post-mortem* examination. The patient (who had left the hospital apparently well) came in again some months afterwards, and died with tubercles in almost all parts of his body; and it was clear that the peritoneum had been the seat of the same disease before. A remarkable instance of recovery from tuberculous peritonitis is recorded by Sir Spencer Wells. The patient, a female aged twenty-two, was believed to have an ovarian tumour, and had twice been tapped. It was decided that ovariotomy should be performed. But, on the abdomen being opened, the peritoneum was found studded with myriads of tubercles. Some coils of small intestine were floating, but the great mass was bound down with the colon and omentum, all nodulated with tubercles, towards the back and upper part of the abdomen. The fluid was pumped out and the wound closed. The patient went through a sharp attack of peritonitis, but recovered, and she afterwards married; six years later she was stout, hearty, and well.

Treatment.—It appears probable that in children tubercular peritonitis is capable, in the majority of cases, of being cured by the local application of linimentum hydrargyri. This practice has long been carried out in Guy's Hospital, the liniment being spread freely over the surface of a flannel belt, which is stitched tightly round the abdomen. We have more than once seen the greater part of the fluid removed within a few days under such treatment, and the patient has also improved in health and gained strength. It is true that there has been no direct proof of the tuberculous nature of the affection, but the cases in question were such as are commonly regarded as instances of "strumous peritonitis," and many of them were running a chronic course. There is evidence to show that in children all tuberculous affections tend towards a fatal termination less uniformly than in adults. It is no doubt advisable to give cod-liver oil,

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syrupus ferri iodidi, and the like; but in several cases these have failed, and the mercurial application has proved successful.

Opium is often called for by pain, and is useful in checking peristalsis. Many physicians still believe that in these cases it is best given with grey powder or minute doses of calomel.

Encouraged by cases like that of Sir Spencer Wells, many surgeons have of late years opened the abdomen, drawn off the effused serum, washed out the peritoneal cavity, and put in a glass tube or flexible drainage-pipe. The result has been favourable beyond reasonable expectation.*

MALIGNANT PERITONITIS.—This—the third chronic disease to which the peritoneum is liable—is of considerable importance in several respects.

Anatomy.—It consists in the presence of an immense number of roundish or flattened nodules or small tumours, with which the peritoneal surface is studded over, and which are sometimes isolated, sometimes aggregated together. Often each little tumour is distinctly umbilicated, and it may send out processes which show a strong tendency to pucker and drag the neighbouring parts of the serous membrane towards it as a centre. In this way, as we shall presently see, the calibre of one or more of the hollow viscera may be very considerably diminished. It is probably by means of a somewhat similar process of contraction that the omentum becomes drawn up and converted into a solid mass, which lies transversely across the abdomen, below the stomach, just as was described above in the case of simple and of tuberculous peritonitis. This induration of the omentum is present in most cases of malignant disease of the peritoneum. In other respects the distribution of the nodules may vary widely. Sometimes the mesentery is covered with them; in other cases it is comparatively free. Often, as Dr Moxon pointed out, the growth is far more abundant on the peritoneum lining the flanks and the diaphragm than elsewhere.

Origin and course.—Malignant disease of the peritoneum is often spoken of as though it were a primary affection. But, as a rule, some one or other of the subjacent viscera is the seat of a similar growth; and to this in all probability the peritoneal affection is secondary. The organs most frequently concerned are, as Virchow stated, the stomach and ovaries.

Out of forty-five consecutive cases of extensive malignant disease of the peritoneum that occurred at Guy's Hospital, in only six were all the viscera free from the same disease. In nineteen the *ovaries* were affected, and were often converted into large tumours by the growth. In seventeen the *stomach* was diseased in the same way; seven times without, and ten times with malignant disease of the ovaries. In three cases the peritoneal affection appeared to have started from the *uterus*, in two from the *rectum*, in three from the neighbourhood of the *pancreas*. In two there was a hard mass in front of the rectum; and in several cases (including some of those in which the disease seemed to have begun in the ovaries) the uterus and its appendages were matted together and fixed to the adjacent parts by a large diffused growth in the subperitoneal tissue.

In one instance, which occurred in 1861, the affection of the peritoneum seemed to have started from a cancerous growth in the ascending *colon*.

* Among many other cases recorded at home and abroad, three published by Mr. Keetley in the 'Lancet' (Nov. 15th, 1890) may be referred to for the sake of the judicious remarks at the close of the paper.

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The omentum formed a solid mass an inch thick, which was spread over the intestines, and reached down to the pubes.

The way in which malignant disease spreads from the stomach or ovaries over the whole peritoneal surface is a matter of much interest. When the growth reaches the serous surface of an organ, it is well known to be capable of infecting the surface opposed to it, without the formation of adhesions between them. The author once saw an excellent instance of this. The body of the uterus was affected with cancer, which reached its outer surface. The omentum was long and hung down into the pelvis, so as to touch the uterus, and in its extreme lower end there was a hard mass, resembling the uterine cancer exactly in its character. There was no malignant growth in any other part of the peritoneum. It is probable that such local infection of the omentum is really not uncommon, and forms the starting-point of the remarkable change in this structure already mentioned. The infection of the general surface of the serous membrane probably arises in the course of the movements of the contained organs. It is even possible, to use the words of Rindfleisch, that "the mutual friction of the viscera may detach fragments of the nodules, and carry them hither and thither over the smooth surface of the membrane, until they find their way into some fold or recess, when they give rise to the development of fresh nodules."

Histology.—The microscopical characters and real structure of the malignant nodules doubtless vary in different cases. Virchow is disposed to include a considerable number among the sarcomata, but most other writers speak of them as "cancerous" in the strictest sense of the word. However, they generally yield but little juice from their cut surfaces, and their structure is to a large extent fibrous. According to Wilks and Moxon the nodules "often consist of fibres with a few spindle-cells," without any alveolar structure or epithelial elements.

Sex and age.—Unlike tubercular peritonitis, cancer of the peritoneum appears to occur much more frequently in women than in men. Out of the forty-five fatal cases referred to above, only eleven occurred in males.

Under the age of thirty this disease is exceedingly rare. Between thirty and forty it is not very uncommon in women, but is very seldom seen in men. In each sex the most numerous cases occur between the ages of fifty and sixty; it is also common between sixty and seventy, and in one instance it was found in a man who died at the age of eighty-two.

Diagnosis.—Clinically, malignant disease of the peritoneum presents itself in different cases with very different symptoms.

The growth may, by the contraction and puckering which it causes, so narrow the intestine as to interfere with the passage of its contents, and to give rise to well-marked ileus (cf. p. 255).

Most frequently the only marked symptom of the disease is ascites. The serum is usually stained more or less deeply with blood. Another character, which was first pointed out by Sir William Jenner, is the occasional presence of a hard mass in the skin and other tissues round the umbilicus. Probably the growth travels along the connective tissue in the path of the obliterated urachus or umbilical arteries or vein.

In other instances the principal symptom of malignant disease of the peritoneum is an increase in size of the abdomen, without any fluid being present. In these cases the growth is a true carcinoma, which has

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undergone *colloid* degeneration.* All the organs may be enveloped in thick layers of this substance, in the form of round gelatinous masses, many of which are attached only by the most delicate threads, or seem to be free.

ASCITES.†—Apart from chronic inflammatory effusion (whether with or without tubercle or cancer) the peritoneum is also liable to passive dropsical effusion, which is known as ascites. This is sometimes accidentally discovered by the physician; more often the patient finds it out for himself, by the fulness and sense of weight in the belly to which it gives rise. An examination of the abdomen, however, is always required to determine the presence of fluid with certainty. For the patient may experience exactly the same sensations from the accumulation of flatus in the bowels, and of fat in the subserous tissue, so that those who seek advice for abdominal dropsy often have none.

Physical signs.—Palpation and percussion are both useful in revealing the presence of fluid in the peritoneal cavity.

Palpation may be employed in two distinct ways. If any solid organ or tumour lies at a little distance from the anterior wall of the abdomen, separated from it by fluid, one can often, by a sudden movement of the fingers, depress the abdominal wall, and push aside the fluid, so as to feel the solid mass beneath in a way that would be impossible if no fluid were present. Thus one may not only detect an enlarged liver, but also at the same time determine the presence of ascites. This procedure is sometimes spoken of as "dipping for the liver:" it requires a little dexterity, and should be carefully practised by the student.

The other method of discovering by palpation whether there is fluid in the peritoneal cavity is by observing whether *fluctuation* can be felt.

This term, as every student knows, is commonly employed by the surgeon to designate the peculiar elastic sensation which results from manipulation of an abscess or other cavity containing fluid. But the way in which "surgical fluctuation" is detected is not that which proves the presence of ascites. To understand the latter, we must bear in mind the fact that the walls of the cavity in which the fluid lies are everywhere more or less yielding. Hence, when an impulse is given to the wall of the abdomen at one spot, the fluid can transmit it freely in the form of a wave. If, for example, the left hand be placed on one side of the patient's abdomen, and a tap be then given to the other side with the right hand, the left hand receives a distinct shock. When the parietes are thin, and other conditions favourable, the slightest touch may cause a thrill that can be felt all over the belly. There is perhaps no other physical sign which the tyro recognises so easily as this. If, however, the parietes are massive, and very hard, or loaded with fat, the detection of fluctuation may be difficult. The two hands must then be placed near one another; and a smart blow must

* Many years ago, when I was a senior student at the hospital, a medical man in the country asked me, during the vacation, to look at a case in which he was about to tap for ascites. I found that although there was very great enlargement and dulness on percussion over the whole abdomen, yet no fluctuation could anywhere be discovered. I remembered hearing Dr Wilks describe colloid cancer of the peritoneum, and ventured to suggest that the case was of this kind, and that paracentesis would lead to no result. During a subsequent vacation I made the autopsy, and found that I had been right.—C. H. F.

† Ascites (ἀσκίτης sc. νόσος, or rather perhaps ὕδρωψ) from ἀσκός, a wine-sack, was recognised by the Greek physicians, and distinguished from *tympanites* (from τύμπανον, a drum), sometimes called the false or windy dropsy.

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be given with one hand, while attention is closely directed to the reception of the impulse with the other. Sometimes the fat in the abdominal walls gives a sensation that might be supposed to be due to fluctuation. To avoid the possibility of error from this source, one may get an assistant to hold a thick piece of cardboard, with its edge pressed upon the median line of the abdomen while percussion is made.

In some cases we may fail to obtain fluctuation, although a large quantity of fluid is present; probably the walls of the space containing it are too unyielding on every side for a wave to be transmitted.

It is remarkable what small quantities of fluid can often be detected in the way just described. One might have expected that unless it were present in large amount it would all have gravitated into the loins or into the pelvis (according to the position of the patient), where it would have been out of reach. On the contrary, distinct fluctuation can frequently be felt over parts of the abdomen when the intestines can be proved to lie immediately in contact with the parietes.

Percussion is also of service in detecting the presence of ascites, and still more in distinguishing this from some other conditions which resemble it in causing abdominal enlargement. Whenever the amount of fluid is at all considerable, that part of the abdomen which contains it gives a dull note on percussion. But a small quantity, lying among the intestines in the way just described, may fail to affect the natural tympanitic note, and this although it gives rise to distinct fluctuation.

Diagnosis.—For the determination of ascites, however, something more is required than the mere discovery of dulness on percussion, or even of fluctuation. The former might be caused by a solid tumour, and the latter might depend upon a collection of fluid within one of the hollow viscera, or in an adventitious cyst. Cystic disease of the ovary is by far the most important of all the affections that may be confounded with ascites. But there are several other conditions that, as a matter of fact, have been mistaken for it. It is recorded that John Hunter once tapped the bladder in the belief that the patient had abdominal dropsy; and Murchison relates a case in which 480 ounces of urine were drawn off by a trocar introduced midway between the umbilicus and sternum, it having been thought that there was a hydatid tumour. A large and elastic tumour may yield physical signs more or less like those of ascites; so may a renal cyst, and, still more commonly, a pregnant uterus.

In the great majority of cases one can readily distinguish an accumulation of fluid in the peritoneal cavity from all these conditions by noticing which parts of the abdomen are dull and which are resonant on percussion, particularly if one makes the patient assume different positions in turn. In ascites the fluid, being specifically heavier that the intestines, tends, in the main, to sink towards the more dependent part of the peritoneal cavity; while they may be said (as Aretæus put it—see head of chapter) to float in it. Hence, when the patient lies upon the back, the bowels fill the umbilical region, and the percussion-note there is tympanitic, whereas in the flanks it is dull. But if the patient is made to turn upon one side, the position of the intestines at once becomes altered; whichever side is uppermost is now resonant, while the dulness on the other side undergoes a corresponding increase. When the patient stands upright the fluid gravitates towards the lower part of the abdomen, which, up to a certain level, becomes uniformly dull. Again, when in ascites the border of

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the dull region is percussed firmly, the left-hand finger being pressed backwards as much as possible, one can often detect a resonant note from the presence of intestine beneath.

All these characters are wanting when enlargement of the abdomen is due to *cystic disease of the ovary*, or to *pregnancy*, or to *distension of the bladder*. Moreover, all of these rise from the pelvis into the front of the abdomen, pushing the intestines backwards. Consequently, when the patient lies upon the back, the front of the abdomen yields a dull note on percussion.

Thus, then, most cases of ascites present positive characters, the recognition of which renders it impossible for a mistake to be made.*

Another sign of some value is prominence of the umbilicus, or occasionally the presence of a protrusion there containing fluid. This is very different from the deep depression of the navel in cases of mere *obesity*. Moreover, in the latter case two transverse lines can generally be traced—above the pubes and above the umbilicus.

But it sometimes happens that enlargement of the abdomen is really due to the presence of fluid in the peritoneal cavity, and yet that the signs which are distinctive of ascites are wanting, the whole of the front of the abdomen being dull, in whatever position the patient may lie. This may arise in two ways. When the quantity of fluid is very large the intestines may stretch the mesentery to its full extent, and yet, perhaps, be unable to reach the anterior abdominal wall. But this very rarely occurs if the parts concerned are in a normal condition. In almost all cases, when the anterior part of the abdomen is dull in ascites, the reason is that the mesentery has been shortened by chronic inflammation, so that it tethers the bowels closely, and prevents their floating.

Under these circumstances the results of percussion may be said to be *negative* so far as concerns the diagnosis between ascites on the one hand, and ovarian disease, pregnancy, &c., on the other hand. We have then to consider what are the *positive* signs of these several conditions.

Now *pregnancy* is distinguished by many indications: the shape of the tumour, and its gradual increase from below, the state of the breasts, the absence of menstruation, and the condition of the cervix, independent of our being able to feel the movements of a living fœtus, or to hear the beatings of its heart, or the rush of blood in the placenta.

The positive signs of an *ovarian cyst* are likewise in many cases conclusive. The patient may be able to say that the swelling distinctly began on one side of the abdomen. Again, a careful examination of the swelling will often lead to the detection of a solid nodule, if it be due to an ovarian tumour; or the outline of the cyst may be felt at some part of its circumference; or, at least when the patient draws a deep breath, a transverse line (corresponding to the upper border of the tumour) may be seen to descend. On this last sign Sir Spencer Wells lays especial stress, and also on the facts that whereas in ascites the greatest circumference of the abdomen is at the level of the umbilicus, in ovarian disease it is often some inches below this; and again, that in ascites the umbilicus usually retains its natural position, being about one inch nearer

* I do not make an exception for the case of an ovarian cyst containing air as well as fluid, because I can hardly believe that the physical signs would then be really like those of ascites. Alteration in the position of the patient might be attended with changes in the percussion-note; but these would generally be limited to a part of the abdomen. In the instances of this kind that I have seen, a very marked splashing sound has been caused by manipulation of the abdomen; and the outline of the cyst has also been very evident.—C. H. F.

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to the pubes than to the ensiform cartilage, while in ovarian disease its distance from the pubes is increased.

Sometimes, however, all the positive signs of ovarian disease are wanting on the one hand, just as are those of ascites on the other. It may then be impossible to make a diagnosis. In such cases, when paracentesis is performed, the character of the fluid which is drawn off often clears up the doubt as to the nature of the disease. That which comes from the ovarian cyst is frequently viscid and of a dark greenish-brown colour, quite unlike the secretion of a serous membrane. Its viscidity is said to depend upon its containing a modification of albumen (paralbumin of Scherer) which does not coagulate when boiled with a small quantity of acetic acid. Paralbumin is said never to be present in ascitic fluid; and, on the other hand, the latter often deposits fibrin, which is absent from the contents of an ovarian tumour. Thus it is said that a liquid containing both paralbumin and fibrin must necessarily have been originally secreted by an ovarian cyst, which afterwards burst into the peritoneal cavity. Ovarian fluid, however, is not always viscid, nor of a dark colour; it may be pale yellow, and in fact undistinguishable in appearance from the fluid of ascites.

The fluid drawn from the single cysts which form in the *parovarium** from the remains of the Wolffian body is characteristic,—clear, transparent, and consisting of nothing but water and salts, unaltered by heat. The only liquid like it is the contents of a hydatid cyst, but that contains hooklets.

In cases of ascites we have next to determine whether it is *passive*, and due to portal obstruction or to general dropsy, or whether it is *active*, the result of inflammation of the peritoneum. It is often supposed that the various forms of chronic peritonitis with effusion are comparatively rare; but at Guy's Hospital they are very frequent, and include at least one third of all the cases of ascites which occur independently of heart disease or Bright's disease and unattended with jaundice.

So long as the quantity of fluid in the abdomen is not very large, one can generally without much difficulty distinguish ascites caused by obstruction of the portal veins from effusion due to chronic disease of the peritoneum. In cases which come under the first head, the area of dulness in the right hypochondrium is diminished, the intestines float freely towards the anterior wall of the abdomen, there is often a history of intemperance, with the chronic disorders of the digestive organs that result from it, the face is often blotchy, and the urine is high-coloured, depositing lithates stained with purpurine. In cases belonging to the second head, the front of the abdomen is very generally dull from retraction of the bowels, there may be no history of intemperance, the patient may have a clear complexion, and (in the case of malignant or strumous disease of the peritoneum) the omentum may be felt hardened and nodulated; or, again, there may be a cancerous tumour of the ovary, or evidence of cancer of the stomach, or a cancerous nodule at the umbilicus.

But whenever the abdomen is greatly distended with fluid—so that it is universally dull in front, and yet one cannot tell whether the mesentery tethering the intestines backwards is contracted or not—the cause of the effusion is beyond recognition by means of physical examination; and it is in these very cases, when they occur in females, that it is impossible to determine with certainty the absence of cystic disease of the ovary. We have then to judge from the general appearance of the patient, and

* Known as the organ of Rosenmüller, and answering to the epididymis in the male.

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a knowledge of her habits; but these afford very uncertain grounds for diagnosis.

When paracentesis has been performed, the nature of the fluid may throw some further light upon the question. *a.* The characters of the fluid contained in cystic tumours of the ovary have already been described. *b.* In cases of cancerous disease of the peritoneum, the fluid, although free from viscidity, is often of a brownish colour, or reddened, from the presence of blood. *c.* In cases of simple chronic peritonitis, and also in cases of ascites from disease of the liver, the fluid is generally straw-coloured. *d.* In very rare cases an opaque white fluid has been removed by tapping; its milky appearance is the result of admixture with chyle, some lacteal vessel having opened into the peritoneal cavity by ulceration.*

A thorough physical examination of the abdomen should always be made after paracentesis; this often clears up a doubtful case by leading to the discovery of a solid tumour, or of some disease of the liver or intestines, that could not previously be detected.

Origin.—First, ascites may be due to chronic inflammatory effusion as above described, whether simple, tubercular or cancerous; secondly, it may be part of general dropsy, usually cardiac or renal; thirdly and most frequently, it depends on portal obstruction. This obstruction may be the result of any of the following diseases; but to distinguish in practice between passive and chronic inflammatory effusion is often as difficult as in cases of hydrothorax, hydropericardium, hydrocephalus, and hydrocele.

1. *Cirrhosis of the liver* is the most common cause of passive ascites, or dropsy of the peritoneum. It will be fully described hereafter (p. 367).

2. *Chronic inflammation of the capsule of the liver*, or, as it is often termed, *perihepatitis*, is also a frequent cause of ascites, passive and inflammatory. At Guy's Hospital there is one fatal case of it for every five of dropsy from cirrhosis of the liver; and this proportion would be greatly increased if we were to take into account those cases in which thickening of the capsule of the liver is merely a part of a general chronic peritonitis. If in a case of ascites the urine be healthy, there is but little likelihood that the cause is inflammation of the capsule of the liver; but when in a case of renal dropsy the abdomen is filled with fluid to a disproportionate degree, this is probably due to perihepatitis rather than to cirrhosis.

3. *Simple chronic atrophy* is another affection of the liver which may very occasionally cause ascites. The 'Pathological Transactions' furnish two very striking cases of this kind, in which the abdomen contained a large quantity of fluid (cf. p. 375).

4. *Syphilitic affections of the liver* sometimes cause effusion into the peritoneal cavity. A striking case, which appears to have been of this kind, was recorded by Dr Grainger Stewart. A patient had ascites, for which she was tapped twenty-one times, the enormous quantity of 606 pints being removed in the course of these operations. At first the paracentesis had to be repeated every fortnight, but the intervals gradually became longer, until at length she regained tolerable health. In twenty years

* At the end of 1878 the present writer had a patient with this chylous ascites, a woman.

The fluid was much like milk in appearance, opaque, yellowish white, alkaline, with sp. gr. 1016. There was no subsidence after twenty-four hours, but scanty flakes of fibrin had separated, probably due to the accidental admixture of a trace of blood. There was no precipitate on heating after the addition of acetic acid, but it formed abundantly when ferrocyanide of potassium was added, and also with nitric acid.

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(1860–82) there occurred in Guy's Hospital about six cases of fatal ascites due to this kind of disease. In several of them the liver could be felt during life to be enlarged and adherent to the parietes, with an uneven and nodular surface, and these characters more than once enabled a correct diagnosis to be made.

5. *Carcinoma of the liver* is another disease that may give rise to effusion of fluid into the peritoneal cavity, but comparatively seldom in large quantity. The presence of malignant growths in the substance of the organ itself is to be distinguished from the cases in which cancer merely involves the different structures in the portal fissure. Both affections will be discussed afterwards (*v. infra*, p. 384, *et seq.*).

6. Occasionally ascites is found to depend on the trunk of the portal vein (not its branches within the liver) being obstructed by plastic inflammation—*pylephlebitis adhesiva.* This is a very rare condition, and is seldom seen except in association with advanced cirrhosis or capsulitis of the liver, by which the circulation through the vein had been evidently greatly obstructed before death. Frerichs states that thrombosis of the portal vein may be suspected when ascites develops itself very rapidly, particularly if the fluid should reaccumulate quickly after tapping. But the reports of cases given by this writer fail to establish his statement. Indeed, it appears that the flow of blood through the portal vein is often arrested by cirrhosis of the liver as completely as it could be by an actual obliteration of the vein; and it is certain that the fluid may collect again with remarkable rapidity after paracentesis when cirrhosis is the cause of ascites, without there being any further obstruction from thrombosis of the vessel.

A case of ascites from adhesive portal phlebitis occurred many years ago (1863) in a boy of ten at Guy's Hospital: no other lesion was found.

Although adhesive pylephlebitis is a very rare disease, thrombosis of the portal vein is not at all uncommon, and probably takes place towards the end of many cases of cirrhosis, perihepatitis, and other cases of hepatic obstruction leading to ascites.

Prognosis.—In almost all cases of ascites this is unfavourable. Some of the diseases that give rise to it are, from the first, malignant; and others do not cause effusion of fluid into the abdomen until they have reached an advanced stage. The first remark applies to cancer of the liver or peritoneum; the second to renal, cardiac, and hepatic dropsy. In cirrhosis of the liver, especially, death occurs in the majority of cases in six months after the detection of ascites. There are, indeed, exceptions to this rule. One patient who recovered from ascites and jaundice under medicinal treatment remained well for several months, after which the fluid reaccumulated, and he returned to the hospital to die. He had been supposed to have a syphilitic affection of the liver, but it turned out that the disease was cirrhosis. A patient lately in Philip Ward (1886) had been four years before in the hospital with hæmatemesis, ascites, and other signs of cirrhosis which had all disappeared.

When recovery takes place from ascites (whether after paracentesis or otherwise) the probability is that the effusion was the result of either chronic peritonitis or perihepatitis.

In children and young persons a form of ascites is not infrequent which is curable, even when it is the result of tuberculous peritonitis.

In some rare cases the umbilicus, having been first forced outwards, gives way, and allows the contents of the abdomen to escape. Sir William

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Jenner has recorded a case in which (no doubt from the presence of air as well as fluid) the rupture was attended with a report loud enough to be heard at a distance from the patient's bed. The fluid may continue draining away for a time, but this scarcely postpones the fatal issue.

Treatment.—Sometimes diuretics may be prescribed with advantage. Of these none appears to be more efficacious than copaiba; but it very often disturbs the stomach, so that the patient cannot continue to take it. The resin is far less likely to disagree, and is no less efficient than the oleo-resin. The acetate or the bitartrate of potass, the spirit of nitrous ether, the compound spirit of juniper, the decoction of broom-tops, the infusion of digitalis, are other remedies of approved value; and a favourite prescription at Guy's Hospital has always been a diuretic pill containing the grey oxide of mercury, powdered digitalis leaves, and powdered squill (of each a grain), which is given night and morning. A useful and pleasant remedy is "Imperial drink," an infusion of lemons with cream of tartar and sugar, of which the patient should drink as freely as possible.

Purgatives, also, are useful, especially those which cause watery discharges from the bowels, such as the compound jalap powder. Murchison recommends an electuary composed of this powder, mixed with confection of senna; and in giving the usual advice that aperients should be taken in the morning, he lays stress upon the reason for this, namely, that otherwise the food which has been recently taken may be swept away, and so the nutrition of the patient suffer. He also insists on the caution required in the administration of drastic purgatives, lest they should set up fatal enteritis. Our experience at Guy's Hospital fully confirms the importance of this warning.

Paracentesis.—In most cases, however, tapping for the withdrawal of the fluid is sooner or later necessary. It should be performed only when the distress caused by the distension of the abdomen becomes insupportable.

The best indication that it is really necessary is perhaps afforded by the state of the breathing, which becomes greatly hurried and very shallow, from the diaphragm being pressed upwards and the lower ribs stretched. The heart also is felt beating above the nipple, and not in the usual place; but it is to be noted that this is often observed before the ascites has reached a very advanced stage.

In performing paracentesis abdominis, the surgeon should use a trocar of moderate size. This is to be introduced in the median line below the umbilicus, it having first been ascertained that the spot selected yields a dull note on percussion, and consequently that the intestines are not in the way. The bladder should have been previously emptied. The trocar is then fitted with a long piece of elastic tubing by which the fluid can be carried into a pail placed below the patient's bed, and the entrance of air prevented.

The operation is by no means unattended with risk, immediate and prospective. The patient has sometimes fainted, and even died, while the fluid was escaping through the trocar. It has, however, long been recognised that the cause of such an accident is the sudden removal of pressure from the viscera, and the danger is obviated by having a jack-towel folded round the abdomen before the operation is commenced, which is held by assistants and tightened as the fluid escapes. When the fluid ceases to flow, the operator removes the cannula with one hand, while with the other he grasps the surrounding integument, so as to prevent the entrance of air into

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the abdominal cavity. A pad of lint is then placed over the wound, and upon this a few broad strips of plaster.

It occasionally happens that this fails to close the opening into the peritoneal cavity. The fluid then keeps oozing out, and saturates the patient's clothing. Such cases generally terminate fatally, but the leakage may be stopped by a suture.

In other instances tapping is quickly followed by peritonitis, which proves fatal in the course of two or three days. Cases of Bright's disease are particularly liable to such a result.

If this be escaped, the fluid almost always begins at once to reaccumulate, being, indeed, poured out much more quickly than before, in consequence of the absence of pressure upon the serous surface. The operation soon has to be repeated, and the patient is again exposed to the same risks as before, though less in degree, with an ever-increasing certainty that the relief will be but temporary and of short duration. Sooner or later he dies, exhausted by the drain of fluid, or by diarrhœa, or by hæmorrhage from the stomach or bowels.

But even though paracentesis abdominis may thus fail to prolong life, it is not therefore useless. It almost always affords great relief to the patient's sufferings, and it should never be delayed when the urgency of the symptoms demands its performance.

Moreover, in some rare cases this operation is as successful as one could possibly wish. There may be no return of the ascites at all; or, as is more often the case, the fluid may be very slow in reaccumulating. Moreover, paracentesis, by relieving the kidneys and veins of pressure, may do much to assist diuretics and increase the flow of urine.

The most favourable cases are those of ascites from primary peritoneal effusion described on p. 317. In one such case under the writer's care, the patient, a woman of about forty, recovered completely after a single tapping.

In cases of chronic peritonitis with thickening the number of times paracentesis can be borne is sometimes surprising. The boy whose case is mentioned at p. 318 was tapped nearly a dozen times. At the present time a patient (Mrs S.) is under the writer's observation, who first came to him in 1884 with ascites, due in all probability to rupture of a compound ovarian cyst, and she has now, at the end of the year 1890, been tapped more than 150 times.

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DISEASES OF THE LIVER

JAUNDICE AND GALL-STONES

"Lurida præterea fiunt quæcunque tuentur
Arquati: quia luroris de corpore eorum
Semina multa fluunt, simulacris obvia rerum;
Multaque sunt oculis in eorum denique mixta
Quæ contage sua palloribus omnia pingunt."
LUCRETIUS.

"Væ meum
Fervens difficili bile tumet jecur."
HORACE.

Bilious or hepatic dyspepsia—disturbance of secretion or elimination of bile—disturbance in nitrogenous metabolism—lithæmia—bilious symptoms—treatment by diet, drugs, and exercise.
Icterus—symptoms—tests for bile-pigment and for bile-acids in the urine.
Idiopathic jaundice: its course, pathology, and diagnosis.
Symptomatic jaundice—Febrile jaundice—from general pyæmia—from portal pyæmia, suppurating hydatid, and acute tubercle—Jaundice from cirrhosis—from simple and malignant obstruction of the ducts—from tumours, &c.
Effects of permanent jaundice on the liver and gall-bladder—effects on the bowels and the skin—Pathological theories of jaundice.
Treatment of jaundice—primary and symptomatic—of permanent jaundice.
Gall-stones—Their structure and varieties—Symptoms and events of biliary colic—treatment—Inflammation of the ducts and gall-bladder—Weil's disease.

THE disorders of the liver are more various than those of any other gland in the body. For beside secreting bile, it also controls the portal part of the systemic circulation; it is concerned in two important metabolic processes, the formation of glycogen and the formation of urea; it contains a large amount of cytogenic or lymphoid tissue, and has an important share in blood-making; it contains at different periods different amounts of fatty matters, which it almost certainly secretes; and lastly, beside its glandular, hydraulic, and metabolic functions, each of which is liable to derangement, the liver is at least as prone as other organs to inflammation, chronic and acute, to the varied kinds of degeneration and atrophy, to the invasion of new growths, the formation of calculi, and the immigration of parasites.

It is, therefore, very difficult to arrange the diseases of the liver satisfactorily.

We shall, as before, adopt a clinical rather than a pathological point of view, and in the present chapter will begin with the functional disorders of secretion, the graver obstructions which produce jaundice, and the concretions which mechanically interfere with the excretion of the bile, as well as the diseases of the excretory channels.

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The two other chapters, dealing with the structural diseases of the liver, will follow an anatomical arrangement, as being convenient and not more artificial than any other which is at present possible.

BILIOUS DYSPEPSIA.—We have already spoken of a kind of dyspepsia probably associated with chronic gastric catarrh, which is characterised by a furred tongue, sluggish bowels, and a sallow complexion (p. 164). Such cases have a certain relation to jaundice and hepatic derangement, which it is well to discuss in the present place.

Beside fulness and distension in the epigastrium after meals, some dyspeptic persons complain of a dull aching in the right hypochondrium, with a sense of weight there, which is often greatly increased by lying on the left side, and is also worse after meals. Another pain, of which such patients sometimes complain (particularly if they read medical works), is situated in the right shoulder. The conjunctivæ have a yellowish tint. The bowels are confined and the motions pale, while the urine is high-coloured, and as it cools deposits lithates of a bright pink colour.

These disorders are most common in the middle period of life, in those who take little exercise, or who eat or drink to excess. They are often at once removed by a few days' shooting or hunting, or by any other active exercise which is sufficiently attractive to induce men to give up sedentary habits.

Probably most people of the richer classes take more food and drink than they require, and many of the poorer do the same. Men get *into* certain ways as regards diet when they are young, or when they are leading active lives; but later on, when less vigorous, and able to take less exercise, they are apt to forget that their habits ought to be altered likewise. Some kinds of food are much more likely than others to produce the symptoms above described. The most injurious are believed to be fatty and saccharine matters, malt liquors, particularly porter and the stronger kinds of ale, port, madeira, champagne, and brown sherry. The evil effects are most marked in hot climates, and in the warmer seasons of the year.

Theory of this condition.—Patients are wont to speak of the symptoms under consideration as indicative of a "torpid state" of the liver, and to think that they are caused by a deficiency in the amount of bile secreted by that organ. The correctness of this opinion was formerly supposed to be established by the fact that the complaint is often easily removed, at least for a time, by certain medicines (particularly mercurials), which also bring away from the bowels a considerable quantity of semi-fluid or fluid fæces, apparently loaded with bile. But there are great difficulties in the way of this theory. A large number of experiments have been made to determine whether mercury and the other drugs above alluded to possess the power of increasing the amount of bile secreted; and no such power has been discovered.

Another way of explaining the complaint is to attribute it to congestion of the portal system, and the remedies for it are those supposed to act by emptying the overloaded blood-vessels. It is well known that digestion is always accompanied by an augmented flow of blood through the liver. Hence it is not a far-fetched hypothesis to suppose that the liver becomes permanently congested in people who eat and drink too often, too much, or too richly. Moreover we know that excess in drink is a cause of a characteristic form of hepatitis.

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There are, however, certain facts of the case which this theory leaves unexplained, particularly the presence of excessive quantities of lithates (urates) in the urine of these patients, and the superiority of mercurials over other purgative drugs which ought to be no less efficacious if the indication for treatment were to relieve congestion.

The late Dr Murchison, in his Croonian Lectures (1874), supplemented the ancient theories upon this subject by bringing in certain facts which appear to show that the healthy liver plays an important and perhaps the principal part in carrying on those chemical changes by which albuminous substances are disintegrated in the body, and which normally result in the production of urea. Some of these facts have been acquired by physiological experiments upon animals, but others are pathological, particularly the fact that urea is absent from the urine in yellow atrophy of the liver when the hepatic cells are destroyed.

He argued that in bilious dyspepsia there is not only defective secretion of bile, but also interference with the normal processes by which albumen is disintegrated in the liver, with the result that instead of urea (CH_4N_2O), lithic or uric acid ($C_5H_4N_4O_3$), a less oxidised body, is formed. He supposes that mercury has a double action. Whether or not it increases the amount of bile secreted by the human liver, it certainly increases the quantity which is passed from the bowels, so that less of the biliary ingredients is reabsorbed, and the blood is freed from their presence in excess. Moreover, he thought it likely that mercury may have a special power of promoting or in some way influencing the disintegration of albumen.

The significance of these views is by no means limited to the class of cases which we are now considering; for the substitution of lithic acid for urea as the final product of disintegration of albuminous substances within the body has further consequences. The comparative insolubility of lithate of soda prevents its being readily excreted by the kidneys like urea. Hence it accumulates in the blood, and a condition arises which Murchison proposed to term *lithæmia*. The urate of soda is very apt to crystallise out in the cartilages of the joints and elsewhere; and this is *gout*. Again, even when the lithates have been separated from the blood by the kidney, the acid is often deposited from the urine either in the renal pelvis or in the bladder. We have then the common form of *gravel*, and those important varieties of *calculus* of which lithic acid and lithates are the main ingredients.

Symptoms of lithæmia.—There is often a feeling of oppression and heaviness, with lassitude or irresistible drowsiness coming on about an hour after a meal. Muscular "rheumatism," lumbago, sciatica, and severe cramps in the legs and other parts of the body may be another indication of lithæmia.*

Another frequent symptom of hepatic disorder is *headache*. The difficulty is to be sure whether we are right in ascribing this to the supposed

* Murchison quotes from Bence Jones these two remarkable instances. The first occurred in a gentleman aged forty, who for years had constantly had deposits of lithic acid and lithates in the urine. He then became subject to attacks of violent pain in the stomach, coming on an hour or more after a late dinner. The pain was intermittently spasmodic; its greatest intensity was reached in half a minute, it then relaxed, to return as badly as before in two minutes. When about an hour had passed the suffering gradually subsided, leaving tenderness on pressure and irritability after food for two or three days. After the attack the urine always deposited crystals of uric acid. The complaint had lasted several months, but under careful dieting and the use of alkalies it entirely ceased.

The second case is that of a patient, also the subject of lithæmia, who was seized with violent cramps in the rectum, coming on six or eight hours after food, and lasting from half an hour to an hour. The same treatment was completely successful in this case.

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excess of lithates in the blood (suppressed gout or lithæmia), or to the reabsorption of bile, or to constipation. Whatever its exact pathology, it is a dull, heavy pain, seated in the forehead, or more rarely in the occiput. It generally comes on when the patient first wakes in the morning, and it may either quickly pass off or last the greater part of the day. This kind of headache usually follows some indiscretion in diet, or is preceded by constipation. It is quite distinct from what are commonly called "bilious" or "sick headaches," which were described among the diseases of the nervous system as migraine (vol. i, p. 775). Still, one cannot deny that typical migraine is also sometimes more or less due to lithæmia: Dr Liveing mentions a family in which the father had gout, his son migraine, and his grandson again gout; and Trousseau speaks of having seen migraine and gout alternate in the same patient, an attack of gouty inflammation in certain joints being followed by the cessation of sick headaches which had previously been of frequent occurrence.

Another effect of lithæmia is *giddiness*. Dr Wilks says that, if due to digestive disorder, swimming in the head is especially apt to come on when the patient stoops or lays his head upon the pillow, and that it often passes off when he assumes the erect posture.

Another symptom, first described by Graves, which appears to be caused by lithæmia, is *grinding the teeth*. He relates four remarkable cases of this kind. The affection is described as depending upon a disagreeable uneasy sensation referred to the teeth, and relieved for the moment by grinding them together. When the habit has become confirmed the teeth have worn down to their sockets. During sleep the grinding entirely ceases, so that the affection is altogether different from that which is so common a symptom of irritation of the brain, and especially of tubercular meningitis. Graves states that all the cases he had seen were in patients of confirmed gouty habit.* He was never able to discover any means of alleviating this troublesome complaint.

Possibly *convulsive attacks* may sometimes be due to the same cause. Murchison relates the case of a gentleman who had long suffered from hepatic derangement, and who became subject to severe spasmodic twitchings in his legs. These were followed on three occasions by epileptiform seizures. A little later he had a first attack of gout, and afterwards he suffered frequently from that disease, but he had no return of the convulsions or muscular twitchings.

Noises in the ears are a more frequent effect of lithæmia. One patient, says Murchison, has the feeling of a strong wind blowing into his ear; another compares the noise to that of flowing water, or describes it as a singing or buzzing; while in yet another the sound pulsates with the beats of the heart.

Then, again, there may be *sleeplessness*. Lithæmic patients are often heavy and drowsy after a full meal and fall asleep at once when they go to bed, but after a few hours they rouse, and they may then lie awake for

* Some observers have supposed that the mere fact of the teeth being ground down, so as to show the dentine in section, is to be regarded as evidence of a "gouty diathesis" or of lithæmia. According to Mr Moon, however, there are several causes which may lead to this condition. One of these is that formation of the jaws which gives what is termed an edge-bite; another is the absence of enamel at the summits of the teeth. Early wearing down of the teeth often depends not on their being too soft or on a gouty diathesis, but on the food being too hard. In races who live on coarsely prepared flour and hard vegetable food the teeth are ground down to the gums, as in old horses.

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hours, or keep dozing off and waking again after unquiet dreams. Such patients never sleep so well as after a dose of calomel or blue pill. Depression of spirits and irritability of temper are well known to be frequent effects of the same cause.

Hepatic and particularly flatulent dyspepsia causes *palpitation* and fluttering of the heart, with intermission or, more rarely, irregularity of the pulse.

Lastly, Murchison enumerates chronic catarrh of the fauces, "gouty" bronchitis, and spasmodic asthma as occasional results of lithæmia.

Treatment.—Formerly many cases of headache, giddiness, and dimness of sight, which are now easily cured by quinine and similar remedies, were submitted to a mercurial course with injurious results; but of late years this kind of practice has fallen into disuse. The younger school of physicians, unable to prove that mercury is capable of increasing the amount of bile secreted by the liver, forgot that this after all is not the whole question. Hence they gave up the use of remedies by which the class of cases just described are for the most part readily and safely relieved. For it is unquestionable that three or four grains of blue pill, with as much of the compound colocynth pill, followed by the traditional haustus sennæ or some less nauseous aperient, do great good in cases of "torpid liver." Podophyllin has been very much recommended for cases of this kind, but the slowness and uncertainty of its action, and the disagreeable griping it causes in some persons, are great objections to its use. A much better substitute for blue pill is euonymin, in doses of two or three grains.

The popular "cure" for a torpid liver, suppressed gout, biliousness, or lithæmia, is a visit to certain German watering-places, particularly Carlsbad. All these waters contain sulphate of soda and chloride of sodium, and the two former sulphate of magnesia also. The proper dose is about five ounces of Püllna water, seven ounces of Friedrichshall water, eight or ten ounces of Carlsbad water. The necessary quantity should be mixed with a little hot water, and taken the first thing in the morning, or about an hour before breakfast. It secures a free action of the bowels, and with this advantage over vegetable purgatives, that there is less constipation afterwards, nor does the dose always require to be increased. On the contrary, the quantity taken may sometimes be gradually reduced without any diminution of its efficacy. After a course of about six or eight weeks the remedy should, however, be omitted, at least for a time; though patients sometimes continue to take it regularly for four or five years.

An instructive lecture on this subject, by Sir Henry Thompson, appeared in the 'Lancet' for January 13th, 1872. He recommends Friedrichshall water in persons of "a uric acid diathesis." Many patients, however, perhaps chiefly women, find they do better with Hungarian bitter water, Hunyadi Janos; and many others have no reason to forsake the cheaper saline laxatives of Epsom, Cheltenham, Seidlitz, or Rochelle.* Whichever is selected, it should be taken before breakfast in not less than a tumbler of warm water.

* Most laxatives are better in combination. This is probably one advantage of natural waters over the sulphate of magnesia or of soda, or the tartrate of potash and soda, alone. But it is easy to imitate the waters of spas by adding a little carbonate of soda and common salt to the combined sulphates. What is sold as citrate of magnesia often consists of bicarbonate of soda and tartaric acid. The combination known as "Lamplough's Pyretic [or Antipyretic?] Saline" contains the same ingredients with about two per cent. of chlorate of potash; "Eno's [so-called] Fruit Salt" adds to these sulphate of magnesia and sugar. These saline aperients are probably less injurious than patent purgative pills and lozenges and "tamar," which often contain aloes, jalapin, or gamboge.

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Regulation of the diet is of the utmost importance, particularly as regards alcoholic liquids. The stronger wines and malt liquors should be prohibited; and the patient should be limited to a very moderate allowance of light but sound Bordeaux or Rhine wine with his principal meals, or of brandy or whisky largely diluted with water.

In most cases of this kind the mineral acids (particularly the nitro-hydrochloric), with gentian and taraxacum, appear to be very serviceable.*

The patient should also be made to take exercise. Of all kinds horse-exercise is the best. Rowing is also excellent, and any exertion which produces deep breathing and free sweating. Walking, however good in other ways, is perhaps least useful for this hepatic form of dyspepsia. A quarter of an hour's game at rackets, for instance, is far more beneficial to most persons than an hour's walk. This advice applies particularly to persons much engaged in business which keeps them on their legs all day. A long walk only makes them more tired, and unable to digest the heavy meal which their sense of exhaustion prompts them to eat; whereas a much shorter time, spent in riding or rowing, exercises the whole body, and after a short rest, or, if needful, a quarter of an hour's sleep, they can enjoy dinner with a zest "that after no repenting draws."

ICTERUS.†—When the colouring matter of the bile fails to escape by the natural passage it is carried into the general circulation by the hepatic veins, and is deposited in the skin and other parts, so as to give the patient a yellow colour. This constitutes *jaundice* or *icterus*.

A large number of the diseases of the liver are attended with jaundice, but by no means all. Moreover, it is present when there is little other reason to suppose the liver to be affected; not only in yellow fever and certain remittents, but also occasionally in typhus, in relapsing fever, and in pneumonia.

Jaundice is itself no *disease*, due to a single cause, and capable of being treated with drugs without further investigation of origin. On the contrary, it is one of the best examples of a morbid condition, which appears an entity to the laity, as it once appeared to physicians, but which we now regard as only a *symptom*, to be traced to its cause whenever we can do so.

General symptoms.—The parts that most obviously display the yellow colour are those which are naturally pale. The redder parts of the skin are far less decidedly altered in appearance than those which are less florid, and in the superficial mucous membranes a similar difference is still more striking. For whereas even in extreme degrees of jaundice the lips and lining of the cheeks show comparatively little change, the yellow hue is ex-

* A nitro-hydrochloric acid bath was advocated by Sir Ranald Martin for the "liver cases" of India. It is made by mixing two ounces of strong hydrochloric acid and one ounce of strong nitric acid with two gallons of water in a glazed earthen or wooden vessel, at a temperature of 96° to 98°. The feet only are placed in the bath, while the inner side of the thighs, the right hypochondrium, and inner side of the arms are sponged with the liquid. The process is repeated each night and morning for half an hour at a time.

† Ἴκτερος, *galbula*, the golden oriole, which, by the doctrine of similars, was believed to cure a jaundiced person who caught sight of the bird. *Icterus* was applied both to the disease and the patient.—The Latin term for a person affected with jaundice was *arquatus*, and for the disorder *morbus arquatus*, i. e. *arcuatus*, from *arcus*, the rainbow. It is so used by Celsus and by Lucretius in the passage from his fourth book which stands at the head of this chapter. Other synonyms were *aurugo* (*i.e.* the golden disease) and *morbus regius* (Cels. iii, 24).—*Fr.* Ictère, Jaunisse.—*Germ.* Gelbsucht. In English the disease is popularly, by a natural tautology, called the yellow jaundice.

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exceedingly well marked in the conjunctivæ, through which in health is seen the pearly-white sclerotic beneath. Just as we place a sheet of white paper beneath any transparent substance, the colour of which we desire to scrutinise, so we always look to the conjunctivæ for the first signs of icterus, or for the faint indication of retention of bile-pigment too slight to produce general jaundice, but indicative of the bilious dyspepsia or lithæmia above described.

When jaundice is present, the internal parts are affected as well as the surface of the body. All the paler *mucous membranes* have a marked yellow colour. The same is true of the connective tissue generally and of the serous membranes; and also of any serous effusions.

Of the *viscera*, the lungs and the kidneys, and almost all other organs which are not so red as to conceal their yellow tint, are evidently jaundiced. The liver displays the same colour, often in an extreme degree. It has been said that the brain participates in this change, which its natural whiteness would of course make very evident. But later observations in the deadhouse at Guy's Hospital confirm those of Dr Moxon, who stated that the brain shows no abnormal colour even in advanced jaundice.

The transparent humours of the *eye* are sometimes yellow, and sometimes not. It is generally supposed that (as Pope, following Lucretius, said) "all looks yellow to a jaundiced eye;" and by actual trial this may be demonstrated in many patients. But sometimes they are able to discriminate yellow or buff objects from white without difficulty.

The colour of the *skin* in jaundice varies according to its intensity and its duration. If the natural escape of the bile be suddenly and completely arrested, the body may quickly assume a deep orange hue. But in many cases this is more gradually developed, the tint being at first a pale sulphur yellow. The whole cutaneous surface is not equally discoloured; the jaundice is generally more marked in the face, arms, and abdomen than in the legs.

When jaundice has existed for a considerable time the colour frequently undergoes a change; it is no longer yellow, but becomes greenish, and after a time passes into a dark olive colour. This change doubtless corresponds with the alteration which bile undergoes when exposed to the air by the conversion of bilirubin (its principal colouring matter) into biliverdin, which again after a time turns brown, passing into what is called choletelin. Persons in whom the skin assumes the dark green colour above referred to were formerly said to have "black jaundice,"* and it was supposed to be an indication that the hepatic disease was of a malignant nature. We now know that it means only that the jaundice has lasted for a long time, but it is true that such cases are generally cancerous. Perhaps for the production of a green or "black" tint in jaundice it is necessary that the flow of bile through the ducts should be *completely* arrested.

There is seldom much difficulty in discovering whether a patient is jaundiced or not. Only a very careless or inexperienced physician could

* In the pathology of Galen, while the yellow bile was secreted by the gall-bladder, the black bile was formed by the spleen. Hence the *dyscrasia* or ill temperament due to excess of yellow bile was called choleric or bilious; that due to excess of black bile, melancholic or atrabilious.

"Quatuor humores in humano corpore constant:
Sanguis cum cholerâ, phlegma, melancholia.
Terra, melan: aqua, phleg: et aer, sanguis: coler, ignis."
Regimen Sanitatis Salernitanum, v. 257.

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mistake for it the greenish-yellow hue of chlorosis, or the yellowish waxen tint often seen in cancerous disease of the abdomen, or the dusky sallow look of malaria. Nor ought anyone to confound with the olive-green tint of black jaundice the brown or bronze colour associated with disease of the adrenals, although it must be remembered that before Addison's discovery these patients were supposed to be jaundiced. In all the conditions just mentioned, but particularly in the last, the conjunctivæ retain their natural pearly-white appearance.

Cases are, however, sometimes met with in which it is difficult to be sure whether a slight degree of jaundice is or is not present. The doubt then generally lies between icterus and idiopathic (so-called pernicious) anæmia. In that disease the conjunctivæ often look yellowish, but this, as in other cases, particularly in old persons, is really caused by the presence of a little fat in the submucous tissue. The yellow colour is partial, instead of being uniformly distributed over the whole surface of the conjunctivæ, the shape of the lobules can be seen, and the vessels which supply them.

One must bear in mind that the yellow tint of jaundice is invisible by gas- or candle-light.

Several of the *secretions* of the body contain biliary colouring matter in jaundice. The sweat is yellow, so that the patient's linen is often much stained under the armpits; and the lachrymal secretion is discoloured. The milk may be bile-stained, as observed by Bright;* but the saliva is colourless, and the secretions of the muciparous glands remain unaffected. Dr Fenwick has pointed out that in cases of jaundice the sulphocyanide of potassium, normally present in the saliva, is not to be found, and the writer has confirmed this observation. The gall-bladder and the ducts of the liver itself secrete a colourless mucus. That the intestinal mucus and succus entericus contain no bile-pigment is evident from the fact that the fæces are of a greyish-white colour, or (to use the common expression) "clay-coloured."

Dropsical serum is deeply stained with bilirubin in jaundiced patients.

The colour of the *urine* in jaundice may vary from a yellow, scarcely deeper than natural, to a dark brown, a greenish brown, or a black so intense that one can recognise its colour only by looking at the margin of the fluid, or pouring some of it out in a thin layer, or making it froth. The presence of bile-pigment in the urine is a necessary part of jaundice, as we should expect from the diffusibility of bilirubin. There is only one condition in which the urine may for a short time have its natural appearance, although the patient's skin is still yellow. This is when the cause of the jaundice has been suddenly removed, particularly if it has lasted for a long time. The bile-pigment then ceases to circulate in the blood, and the kidneys no longer excrete it; but the skin does not at once give up all the colouring matter that had been deposited in its tissues, and it remains for a few days yellow. With this single exception the urine contains bilirubin when there is jaundice. Indeed, we sometimes obtain more delicate indications of slight or early icterus from the urine than from the conjunctiva.

Test for bilirubin.—The presence of bile-pigment in the urine is not to be assumed from the colour alone. We have a chemical test which is capable of detecting it in the most minute proportion. This is known as Gmelin's test. It consists in the addition of fuming nitric acid (containing nitrous

* Heberden affirms the contrary, and an error of observation is incredible in either case. The writer has never seen jaundice during lactation, and has inquired in vain of physicians more likely to observe the coincidence.

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acid) to a small quantity of the urine. This causes a beautiful play of colour if bile-pigment be present. A good way of employing the reagent is to pour a drop or two of the urine on the flat surface of a white plate, and then carefully to add to it a single drop of the nitric acid. Around the drop a series of colours is developed, rapidly passing through the shades of green, blue, and violet, into red, and finally becoming a dirty yellow. Neubauer and Vogel recommended, for the detection of small quantities of bile-pigment in urine, that the nitric acid (which must not contain too much nitrous acid) should be poured about an inch high into a conical glass, and that a little of the urine should then be carefully spread over its surface by means of a pipette. The play of colour begins where the fluids come into contact with a beautiful green ring, which gradually extends upwards, and at its under surface exhibits a blue, violet, red, and lastly a yellow ring. Mere darkening of the urine by oxidation of the natural urobilin of urine into a reddish-brown colour is a familiar effect of nitric acid quite distinct from that just described.

When nitric acid is added to urine in the way described above, red and violet rings may be produced by another substance, of which a small quantity is present in healthy urine, and which is increased under various pathological conditions. This was once called uroxanthin, but a better name is *indican*, for it is the same principle which, when obtained from the indigo plant, has long been known as the mother-substance of the indigo pigments.

The most delicate of all methods of applying Gmelin's test for bile-pigment is to shake large quantities of the urine successively with chloroform. This extracts any bilirubin that may be present, and when nitric acid is afterwards added the reaction is apparent.

Tests for bile-acids.—There has been considerable difference of opinion as to whether the glyco- and tauro-cholic acids of the bile are excreted by the kidneys in jaundice, and whether this is the case in some forms of jaundice and not in others.

The principal chemical test for the biliary acids, or rather for the cholic acid which they both contain, is known as Pettenkofer's. It consists in the admixture of a few drops of syrup or a few grains of sugar (either sucrose or glycose) with the liquid suspected to contain biliary acids, and the subsequent addition of strong sulphuric acid, precautions being taken to prevent the development of too great heat. A beautiful violet colour appears if the acids of the bile are present. But Pettenkofer's test cannot always be satisfactorily applied to urine unless the urinary pigments are first separated, for if this be not done the sulphuric acid may blacken the liquid, so that no violet colour can be seen. Still there is no great difficulty in applying it to demonstrate the presence of cholic acid when bile has been purposely added to urine. It is very doubtful if the presence of the bile-acids has ever been observed in urine without their having been so added.

Moreover, it is improbable that a large quantity of the biliary acids should be found in the urine in any case of jaundice. Only small proportions of those acids are discharged from the body in health. According to Bischoff not more than a quarter of the amount of biliary acids poured into the intestine by the liver passes away in the fæces, and even this has undergone important chemical changes. It is probable that the rest is reabsorbed into the blood, and is there further transformed, possibly, as Frerichs supposed, into bilirubin.

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IDIOPATHIC JAUNDICE.*—In a large proportion of cases of jaundice we can ascertain scarcely anything, whether by examining the patient or by asking him questions, beyond the fact that his skin and conjunctivæ are of a deep yellow colour, that the urine contains much bile-pigment, and that the stools are clay-coloured. There is not, nor has there been, any pain or uneasiness in the region of the liver. Very often the patient says that he feels perfectly well, and would not know that anything was wrong with him but for seeing his yellow face in the glass.

After a variable period—sometimes as much as six weeks—the jaundice subsides. The first sign of improvement is generally that the motions regain their natural colour; the urine soon ceases to contain bile-pigment, and a few days later the skin and conjunctivæ regain a healthy appearance. This favourable change often takes place about the twenty-first day; but in some cases it occurs earlier than this, and in other cases very much later. At no period of the complaint can one generally make out that the liver is enlarged, whether by palpation or by percussion; from beginning to end there is nothing to throw light on its cause. For jaundice of this kind, "simple," "primary," "idiopathic," and "benignant" seems to be suitable adjectives.

Pruritus.—Jaundice is sometimes attended with itching of the skin. In certain persons papules develop themselves whenever the skin is scratched; hence when they are jaundiced they often present an eruption of pimples, the summits of which become quickly destroyed by the finger-nails. This rash has been mistaken for scabies. Graves observed urticaria develop itself under the same circumstances; and he also noticed that itching of the skin sometimes precedes jaundice by a considerable interval; in one of his cases this was a period of ten days, in another of two months. The late Dr Addison used to teach the same fact, and said that he had once suggested beforehand the possibility that an attack of jaundice might be impending when a patient complained of itching, for which no explanation could be found, and that his prediction had been justified by the result.

Slow pulse.—Another symptom sometimes observed in simple jaundice is extreme infrequency of the beats of the heart. The pulse occasionally falls to 50, 40, or even 20 in the minute. It has been found in experiments upon animals that the pulsations of the heart are much reduced in frequency by the injection of the salts of the biliary acids into the circulation.† It was, therefore, supposed that in such cases of jaundice the blood contains these acids. But chemists have hitherto failed to discover them, except in traces, in the urine, whether in this or in other forms of jaundice; and, since they are readily diffusible, one cannot suppose that it is possible for them to accumulate in the blood in sufficient quantity to affect the heart without being freely excreted by the kidneys. An important question is whether a slow pulse in jaundice makes the prognosis unfavourable. There appears to be no ground to suppose that it does. Like xanthopsia and pruritus, this symptom is more often seen in idiopathic than in secondary jaundice, and though frequent is by no means constant.

Ætiology.—The complete absence of bile from the fæces in this form of jaundice affords a presumption that there is some obstacle to its flow; and

* Simple jaundice—Catarrhal jaundice.

† Röhrig, 'Ueber d. Einfluss d. Galle auf d. Herzthätigkeit,' 1863. See also a paper by Dr Legg ('Proc. Royal Soc.,' 1876) with references to Traube's observations, and a fuller account with tracings in his work on 'The Bile, Jaundice, and Bilious Diseases,' p. 204.

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the common theory is that simple jaundice depends upon *catarrh* of the larger bile-ducts. It is believed that their lining membrane is swollen, and that mucus is secreted, which obstructs the channel. One difficulty is that patients never die from, and rarely during, this idiopathic and benignant form of jaundice, so that we have little or no knowledge of its morbid anatomy. Another is that we have no corresponding instances of spontaneous catarrh of a duct, with obstruction and reabsorption of the secretion, in the case of the ureter, or other ducts of glands.

A more probable suggestion is that catarrh of the duodenum obstructs the oblique and narrow passage of the duct through the walls of the gut; but here the difficulty is that the jaundice does not more constantly follow what is probably a frequent disorder. Moreover, we should have expected that chronic catarrh would have produced permanent jaundice, as chronic obstruction in the nasal duct produces permanent epiphora.

In acute yellow atrophy of the liver the ducts are always found after death to be pervious; and it is difficult to suppose that when a case of this kind begins as one of simple jaundice it is at first due to a catarrhal inflammation of the ducts, and that this afterwards subsides without leaving a trace. Again, there is no evidence or likelihood of catarrh of the gall-ducts when icterus accompanies pneumonia.

Jaundice sometimes follows directly upon the shock of some mental *emotion*. Sir Thomas Watson mentions the case of a young medical friend of his who became jaundiced from anxiety before an examination at the College of Physicians; and he refers to another case in which an unmarried woman, on its being accidentally disclosed that she had borne children, became in a very short time yellow. Similar instances are not rare. Murchison places cases of this kind in an entirely different category from those which he attributes to catarrh of the larger bile-ducts; but beyond the fact that the former are caused by mental emotion, while the latter are not, no differences can be found between them, either in their symptoms or their course.

An affection having the characters of simple jaundice has occasionally prevailed *epidemically*. Several instances of this were collected by Frerichs. Murchison mentions a remarkable outbreak of the same kind which occurred at Rotherham. In 1862 this town was visited by enteric fever, which proved very fatal. Early in the following year jaundice became epidemic. It is said that in February no fewer than one hundred and fifty persons were suffering from it; and there was this curious circumstance, that none of those who were attacked had passed through the fever. Other examples are recorded among our troops in India by Morehead and Goodeve.

Diagnosis.—The characters which distinguish idiopathic jaundice from that which is a symptom of another malady are chiefly negative; and one might expect that the diagnosis would be somewhat uncertain, since in some cases of cirrhosis, of cancer of the biliary passages, and even of gall-stones, jaundice may be the first symptom. But whereas these diseases seldom occur in young subjects and in those who have hitherto enjoyed good health, "simple" jaundice seems particularly apt to attack the young and robust. Hence it does not often happen that a mistake is made in supposing jaundice to be idiopathic and benignant, when it is really due to organic disease. Practically the only serious difficulty to be considered is the possible, though happily very rare, supervention of acute atrophy of the liver.

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SYMPTOMATIC JAUNDICE.—Jaundice is not infrequently seen in cases of mitral disease with an enlarged and congested liver. The naturally low pressure in the portal vein appears to be overbalanced by the increased pressure in the right auricle and hepatic veins, and the stasis thus produced prevents secretion of bile.

As a complication in some of the *specific fevers* jaundice is not infrequent. This is always the case in "yellow fever." In "relapsing fever" jaundice is a frequent and not necessarily an unfavourable symptom. It sometimes, though very seldom, occurs in typhus, and in this disease almost every patient who becomes jaundiced dies. In enteric fever and scarlatina jaundice is extremely rare.

The icterus which frequently follows the bites of venomous *snakes* may be mentioned in this connection.

Another disease, attended with fever, in which jaundice may occur as a complication is *acute basal pneumonia.* This has been supposed to be due to an extension of inflammation through the diaphragm to the upper surface of the liver—an almost absurd notion, for there is no sign of inflammation of the diaphragm, pleura, or peritoneum between liver and lung; pneumonia cannot "extend" to anything but pulmonary tissue; and even if inflammation does attack the liver, it does not in itself cause jaundice. At least one case of this kind has been observed at Guy's Hospital in which jaundice occurred as a complication of pneumonia of the *left* side.

General pyæmia is often accompanied with jaundice; indeed, a slight yellowness of the skin is one of its most frequent symptoms. Some years ago Dr Wilks investigated the question whether those cases of pyæmia in which abscesses occur in the liver are or are not particularly liable to be accompanied by jaundice, and he came to the conclusion that the local disease had nothing to do with the production of this symptom. Indeed, in most of the febrile diseases of which jaundice may be a symptom it is probably dependent upon changes in the blood or elsewhere, and not upon any morbid state of the liver; to use terms at one time much used, it is *hæmatogenous*, not *hepatogenous.*

Portal pyæmia.—Besides these general maladies, there are certain diseases of the liver itself which are attended with fever, often with rigors, and local pain with tenderness, and which may also give rise to jaundice. Icterus is a rare and accidental symptom in cases of *tropical hepatic abscess.* But it often accompanies a special form of suppuration in the liver, which has been termed purulent *pylephlebitis*, or inflammation of the branches of the portal vein and of the connective tissue in which they are embedded.

The following is a striking case of this kind. A man aged thirty-seven was admitted into Guy's Hospital under the care of the late Dr Barlow, exceedingly ill with jaundice, fever, and delirium; and he died in two days. He had been quite well a week before, except that he suffered from stricture of the rectum. The liver was very large, and its tissue was suppurating throughout. The branches of the portal vein were all distended with soft thrombi of a brownish colour. The main trunk of the vein contained a dirty-looking fluid. The inflammation of the liver was evidently due to the absorption of some unhealthy material by the veins ramifying in the coats of the rectum, which was extensively ulcerated.*

* Other causes of suppurative pylephlebitis are—ulcerative affections of the stomach or bowels, suppuration of the spleen, suppuration of the mesenteric glands, and the penetration of one of the veins which go to form the portal trunk by foreign bodies; in a case quoted by Frerichs, a fish-bone had entered the inferior mesenteric vein.

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Another cause of jaundice with pyrexia is suppuration of the portal canals throughout the liver, excited by the presence of gall-stones in the ducts. The possibility of this occurrence must never be forgotten in any case of biliary colic in which febrile symptoms show themselves.

An inflamed hydatid cyst in the liver may set up suppuration along the portal canals, affecting the branches of the bile-ducts rather than those of the portal vein. The explanation of this lies in the fact that a suppurating hydatid often communicates directly with a branch of the bile-duct of considerable size. Hence membranous portions of the hydatid, or of its capsule (detached by sloughing), may enter the bile-duct and obstruct it. An interesting case of this kind occurred in Guy's Hospital a few days later than the case of pylephlebitis above mentioned. The liver was found after death to contain a suppurating hydatid cavity which held three pints of fluid. The bile-ducts throughout the organ were suppurating, and the main canal was obstructed by a large piece of detached membrane rolled up into a cylinder.

A real but a very rare cause of febrile jaundice is *acute tuberculosis* of the liver. An instance of this occurred to Murchison in a woman forty years of age. Another case was observed some years ago at Guy's Hospital. A man, aged thirty-seven, died with febrile symptoms of typhoid character. The history was imperfect, but he was stated to have had jaundice only five days. After death the liver was found to be full of tubercles, there being as many as fifty to the square inch. There was also pneumonia of the left lung.

Jaundice from cirrhosis of the liver.—Writers generally state that jaundice is seldom caused by cirrhosis, and it is true that in the majority of cases this affection runs its whole course without marked icterus. But there is, nevertheless, a considerable minority in which jaundice is present, and not rarely it constitutes the chief symptom of the disease. Among one hundred and thirty cases occurring in the *post-mortem* room of Guy's Hospital, in which the liver was found after death to be cirrhosed, jaundice existed in thirty-four, and in ten it was deep or intense. During this period, however, there were examined in the same place only some sixty other cases in which jaundice was a principal symptom. Thus, among the causes of jaundice, cirrhosis of the liver is far from taking an insignificant place.

The fact is that the frequency with which cirrhosis of the liver occurs is far in excess of that of the other organic diseases that give rise to jaundice; and consequently, although jaundice is not a very common result of cirrhosis, cirrhosis is by no means an uncommon cause of jaundice.

Jaundice due to this disease has some peculiar characters. It is frequently gradual in its onset. It is often slight in degree. There is seldom, perhaps never, complete absence of bile from the fæces. It is unattended with pain, and so differs from jaundice due to gall-stones or cancer. On the other hand, it is very often associated with the other symptoms of cirrhosis which will hereafter be described, and particularly with ascites. In fact, the concurrence of jaundice with ascites is met with in scarcely any disease excepting cirrhosis and cancer.

Icterus gravis.—Jaundice is an early and constant symptom of the remarkable disease known as *acute yellow atrophy*. This will be fully treated in the next chapter.

Malignant jaundice.—Icterus is often due to *cancer*, either of the liver itself or of the biliary passages, and will come under notice as a symptom of malignant growths in and about the liver.

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Icterus neonatorum.—Deep and permanent jaundice may be caused in infants by a congenital *obliteration of the common bile-duct*, apparently resulting from intra-uterine perihepatitis; several instances of this have been recorded. The jaundice has appeared a few days after birth; it has been attended with hæmorrhage from the bowels and skin, and especially from the umbilicus. This last is sometimes the cause of death, which in other cases is due to progressive atrophy, attended with vomiting and diarrhœa; in two instances the child lived as long as six months.

Jaundice in infants is also said to arise from plugging of the common duct by inspissated bile, as in a case quoted by Murchison. But in many cases of supposed jaundice in new-born children the yellow tint is the result of changes of the blood in the over-congested skin, "the vivid redness of the new-born baby" (to use Murchison's expression) "fading, as bruises fade, through shades of yellow into the genuine flesh-colour."

Stricture.—Another very rare cause of permanent jaundice is *stricture* of the common duct, resembling an ordinary stricture of the urethra. This condition is generally caused by cicatrisation of an ulcer, itself probably set up by a gall-stone; or the submucous tissue may be converted into an indurated mass of new growth.

External pressure.—Permanent jaundice may also be caused by an *external tumour*, of any kind, pressing upon the hepatic or common duct. Tuberculous glands in the portal fissure have occasionally obstructed the flow of bile; and an aneurysm of the hepatic artery has sometimes been the cause of jaundice. Such cases have generally been attended with severe paroxysmal pain, like that produced by gall-stones.

Gall-stones.—Jaundice is a characteristic symptom of an impacted calculus, and also of the singular cases described by Weil (p. 356).

Anatomical effects of persistent jaundice.—Whatever its cause, jaundice with obstruction of the ducts leads to a definite series of further changes. The gall-bladder and all the biliary passages become greatly dilated and distended, at first with bile, afterwards with mucus of a greenish tinge, or perfectly colourless. The gall-bladder may thus come to contain many ounces, and may be felt as a rounded or pear-shaped mass below the liver.

Very often there are gall-stones in it as well as mucus, and their presence has been sometimes detected by palpation. They yield a peculiar crackling sensation, which has been compared to that produced by grasping a bag of hazel-nuts or rolling pebbles about in the mouth.

Sometimes the gall-bladder suppurates and points externally. The result of this is the production of a fistulous opening, through which after a time green bile is discharged in quantities of from eight ounces to two pints daily. The jaundice may then subside; but this change is of no benefit to the patient, who rapidly loses flesh and strength, and before long dies exhausted.

The hepatic ducts become dilated in cases of permanent jaundice; they may even become larger than the branches of the portal vein with which they run, and may be visible on the surface of the liver as cylindrical tubes or sacculated pouches. The enlargement of the ducts appears to be the cause of the fact that the liver as a whole is larger than natural in the early stages of this form of jaundice. But after a time the organ begins to shrink, and at length it becomes considerably smaller than in health.

Another change in the liver consists in its assuming a dark olive-green colour, which all writers describe as darker than that of other parts of the

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body. Doubtless this is due to the oxidation of the bile-pigment contained in the hepatic cells, converting it into biliverdin, exactly as in the skin. The connective tissue in the portal canals becomes thickened when the common bile-duct is obstructed. This has lately been dwelt upon by Dr Wickham Legg, who has shown that in the lower animals the operation of ligaturing the bile-duct is quickly followed by an overgrowth of connective tissue as great as in intense cirrhosis. The author's observations led him to believe that a similar change occurs very frequently in cases of obstructive jaundice in man, although not to the same degree.

The liver-cells appear not to undergo any change beyond being somewhat reduced in size. In 1843 Dr Thomas Williams related a case in which jaundice was caused by malignant disease of the head of the pancreas, and in which almost all the cells of the liver were found to be broken down, fatty globules and granular matter being present in their place ('Guy's Hosp. Reports,' 2nd ser., vol. i, p. 444). But no one has since recorded a similar observation, except when cerebral symptoms had been present, such as to justify the opinion that the disease which caused the jaundice was complicated with acute yellow atrophy. This occurred in a case which Murchison has placed on record in the 'Pathological Transactions' (vol. xxii, p. 159).

Fatty stools.—Certain minor results, which sometimes follow jaundice, demand a brief notice. In 1832 Bright recorded some cases of jaundice in which the alvine evacuation contained a substance like fat, which either passed separately from the bowels, or soon divided itself from the general mass, and lay upon the surface; "sometimes forming a thick crust, particularly about the edges of the vessel, if the fæces were of a semi-fluid consistence; sometimes floating like globules of tallow which had been melted and become cold; and sometimes assuming the form of a thin, fatty pellicle over the whole, or over the fluid parts in which the more solid fæces were deposited." This state of the fæces was sometimes so marked as to have been noticed by the patient before Bright saw the case. The oily matter had generally a slight yellow tinge, and a most disgustingly fœtid odour. Bright was himself disposed to regard this peculiar condition of the evacuations as due to disease of the head of the pancreas and of the duodenum, and he seems to have thought that the presence of jaundice in his cases was accidental. Later writers also have generally attributed the symptom under consideration to obstruction of the pancreatic secretion, supposing that it prevented the fatty matters taken in the food from being digested and absorbed.*

Dr Walker, of Peterborough, brought before the Royal Medical and Chirurgical Society, in 1889, two cases in which the fæces were colourless though the patient was not jaundiced, and after death the pancreatic duct was found occluded, but not the bile-duct.

Emaciation.—Dogs in which a biliary fistula has been formed require a larger quantity of food to maintain their nutrition than before. This corresponds well with the circumstance that in all protracted cases of jaundice the patient becomes exceedingly thin and emaciated; although he may be entirely free from other conditions which might account for the wasting.

* Bidder and Schmidt found that in dogs, in which the bile-duct was ligatured, the amount of fat that could be absorbed from the intestines was reduced to less than one half, and sometimes even to as little as one fifth or one seventh of the quantity that the animal could digest before. These experiments suggest a doubt whether the presence of fat in the fæces in Bright's cases was not caused by the obstruction of the common bile-duct rather than of the duct of the pancreas.

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Flatulence, &c.—Another result of the prolonged absence of bile from the bowels is that their contents undergo putrefactive changes, the occurrence of which is prevented, under normal conditions, by the powerful antiseptic properties of bile. Hence the evacuations often have a very fœtid odour, and gases are generated which cause tympanitic distension of the abdomen. The contents of the intestines may probably in this way acquire irritant properties, and so set up diarrhœa. More often obstinate constipation is present in jaundice, and this is accounted for by the hypothesis that the bile is the "natural purgative" or stimulant to the peristaltic action of the bowels.

Xanthelasma.—Persistent jaundice, from whatever cause, is apt to lead to a remarkable affection of the eyelids and other regions called originally "vitiligoidea" by Addison and Gull ('Guy's Hospital Reports,' 1851 and 1887), but now known as xanthoma or xanthelasma. It will be described in a future chapter among diseases of the skin.

Theory of jaundice.—It has been supposed that, pathologically, there are two distinct forms of jaundice, in one of which the bile-pigment is secreted by the liver as usual, but, owing to *obstruction*, is afterwards reabsorbed into the blood; while in the other the secreting action of the liver is *suppressed*, so that the bile-pigment in the skin and urine must have been formed by some other organ or in the blood itself. Undoubtedly in certain cases of jaundice the ducts are mechanically obstructed, while in other cases they are patent. The question is whether the jaundice is essentially different in its origin in these two classes of cases. Must we admit there are two kinds of icterus—the one hepatogenous, the other hæmatogenous?

One distinction between these two forms of jaundice has been supposed to be that bile-acids are present in the urine in cases of obstructive jaundice, and are absent when the bile-ducts are free; but we have seen this distinction to be untenable (p. 339). Nor does the state of the fæces afford such a criterion. In acute yellow atrophy, in which the ducts are unobstructed, the motions are sometimes, if not always, free from bile; and, on the other hand, we had in Guy's Hospital a case in which jaundice was due to obstruction of the common bile-duct by a cancerous growth, which, however, only partially occluded its channel, so that the fæces remained of their natural colour.

The theory of a hæmatogenous form of jaundice is inconsistent with the physiological doctrine that the bile-pigment is in health formed by the liver, and does not pre-exist in the blood. But the evidence on which this doctrine is founded is perhaps not conclusive. The direct removal of the liver in frogs and in geese does not lead to jaundice, but how far these experimental results apply to man admits of question. Again, no bile-pigment can be detected in the blood in health; but it is quite conceivable that, though the bile-pigment is formed in the blood, it may never accumulate in sufficient quantity to be detected by chemical tests, if we suppose that the liver, with its large and active circulation, is engaged in removing it as fast as it is produced.

The pathological evidence on this question is liable to be misunderstood. Thus Murchison lays stress on the fact that the gall-bladder and bile-ducts are sometimes found after death to contain only a grey mucus, although during life there had been no jaundice. This would be a striking argument if the liver were always diseased in such cases. But Dr Moxon found that, of four instances of this kind, one only was a case of fatty liver accompanying phthisis, the others being cases of pyæmia or pneumonia. It is evident

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that these cases merely show that under certain circumstances the formation of bile-pigment may be arrested. They have no bearing on the question whether the bile-pigment is formed by the liver or elsewhere.

Murchison did not believe that the presence or absence of obstruction of the ducts makes any important difference in the way in which jaundice is produced. He points out that the osmotic currents between the blood and the contents of the biliary passages and intestines are remarkably active, and that even in health much of the bile which is secreted by the liver is probably reabsorbed into the blood. He argues that we have only to suppose that this reabsorption is excessive, or that the reabsorbed pigment fails to be properly got rid of, and we have at once an explanation of the occurrence of jaundice with patency of the ducts. But, as Dr Moxon has pointed out, this theory, that jaundice is in all cases due to reabsorption, is entirely inconsistent with the fact that in jaundice the biliary passages are almost always found to contain, not bile, but an almost colourless mucus. This is the case not only in acute yellow atrophy of the liver, but also when the ducts are permanently obstructed by stricture or gall-stones. As Dr Moxon remarks, the contrast is at first sight very astonishing between the deep yellow fluid found in distant serous cavities in such cases and the clear liquid which is present in the ducts of the liver itself. The absence of bile from the biliary passages is, however, easily explained. The secretion of bile takes place under very low blood-pressure. Hence, when the common duct is obstructed, the entrance of bile into the biliary passages is at once arrested. But the mucus secreted by the walls of these passages and by the gall-bladder can undoubtedly continue to be formed under a much higher resistance. Consequently it soon displaces the last trace of bile; and like all mucous fluids, it is itself unstained by bile-pigment, even when jaundice is present.

This explanation evidently assumes that a colourless mucus will be found in the bile-ducts in cases of obstructive jaundice only when the obstruction is complete. And a search through the pathological records of Guy's Hospital for the last twenty years fails to discover any exception either to this rule or to that from which it is derived: on the one hand, there is no case of long-continued complete obstruction in which the ducts contained a liquid highly charged with bile-pigment; on the other hand, there is no instance of partial obstruction in which they contained a colourless mucus.*

In protracted jaundice the liver-cells contain bile-pigment which has stagnated there until it has become green by oxidation; and they are deeply stained in acute atrophy of the liver: facts which favour the hæmatogenous origin of jaundice without obstruction. It is more easy to believe that in acute yellow atrophy the function of the liver is completely arrested than that the liver-cells pour back the bile-pigment into the blood as soon as they have formed it, the ducts being perfectly patent.

We must admit, on the one hand, that the theory of hæmatogenous jaundice is not in accordance with experimental results; and, on the other,

* Certain writers have endeavoured to account for the presence of a fluid unstained with bile in the biliary passages in cases of jaundice by supposing that the smaller ducts are plugged with inspissated bile, which thus cuts them off from the canals into which they should open. But it is surely inconceivable that all the smaller ducts throughout the organ should at the same time be obstructed in this way. For there would be no alternative but to suppose that in all cases of complete obstructive jaundice the bile-ducts up to the very point where their radicles meet the walls of the hepatic cells are plugged with a non-diffusible mucous fluid.

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that the large group of cases of jaundice without obstruction cannot at present be explained without it.

Icterus by obstruction and reabsorption of the secreted bile, from the bile-ducts into the lymph-passages of the liver and so into the general circulation, is a well-ascertained process. Slight degrees of obstruction may probably produce jaundice, as in cases of nutmeg liver and possibly of catarrh of the duodenum. But unless we admit that the hæmoglobin of the blood may, under morbid conditions, be decomposed into hæmatoidin (or bilirubin) in the spleen or elsewhere, and failing of excretion by the liver continue to circulate and stain the tissues and the urine, we have no satisfactory means of accounting for the icterus of acute yellow atrophy, of fevers of nervous origin, or even for the so-called "simple" jaundice of healthy adolescents.

Frerichs's supposition, adopted by Murchison, of excessive secretion of bile and of reabsorption of the excess from the intestine, is unsupported by evidence and contrary to analogy.

Treatment of jaundice.—The *idiopathic* form is so uncertain in its course that it might well appear a hopeless task to determine whether remedies are capable of abridging its duration. On the Continent the most efficacious remedy is believed to be the administration of certain mineral waters—those of Vichy, Ems, Kissingen, Marienbad, and Carlsbad. Those of Vichy are most strongly recommended by French physicians, while German writers speak most highly of Carlsbad. Since all these springs contain a considerable quantity of the salts of soda (especially the sulphate and carbonate) it is interesting to find that the same salts are believed in this country to be useful in the treatment of jaundice.*

Carbonate of soda with or without taraxacum, and saline laxatives, to which many physicians add an occasional blue pill, is the traditional treatment for "simple" jaundice; but our ignorance of its pathology and the uncertainty of its duration make it difficult to measure the efficacy of these drugs. Dilute nitro-hydrochloric acid has a scarcely less established reputa-

* It is true that we seldom give the salts of soda alone, but combine them with remedies such as taraxacum and rhubarb, which are either thought to exert a specific action on the liver, or regarded as useful by regulating the bowels. That in this way it is possible to bring an attack of jaundice to an end I feel confident, and I think that the following two cases go far to prove it. (1) A man aged fifty-nine came to me on January 9th, 1874, suffering from jaundice, which was not very deep, but had already been of two months' duration. There was some tenderness and fulness over the liver. I ordered him to take half a drachm of spiritus ammoniæ aromaticus in a mixture of rhubarb, soda, and calumba. He came again on the 16th, and said that for five days his motions had continued to be clay-coloured, but that on the 14th they began to contain bile, and that they were now quite dark coloured. On testing his urine I found that it contained very much less bile-pigment than before. His jaundice was much diminished, and in the course of another week it entirely disappeared. (2) A bargeman came to me on February 23rd, 1872. Ten months before he had become suddenly jaundiced without pain. A month afterwards he had been seized with excruciating pain over the liver, lasting some hours, and he had since had three similar attacks. He had been under treatment both in Guy's Hospital and at King's College, but without result. He remained jaundiced the whole time. Having ascertained the treatment that had been previously employed in Guy's, I ordered him to take ten grains of carbonate of soda, with a scruple of extract of taraxacum, and (as he had some dyspeptic symptoms) half a drachm of tincture of hop three times a day, and a grain of opium at night. On March 1st he came to me again, and assured me that he was very much better, having lost the pain and sickness. On March 4th he noticed that his motions resumed their natural appearance, and before long he was well. I do not suppose that all cases of simple jaundice will subside equally rapidly under such treatment, but I think it certainly deserves a fair trial.—C. H. F.

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tion in cases of jaundice, and certainly patients may quickly lose their yellow colour while taking this medicine.

With regard to the general management of the disease, the patient should not be kept in bed, nor even within doors. He should be allowed to take moderate exercise, and to have his usual diet, from which, however, fat, pastry, and malt liquors should be excluded.

When the cause of the jaundice has been removed, the fading of the yellow colour of the skin may, according to Murchison, be hastened by warm baths, by giving the patient diuretics and diaphoretics, and by the administration of benzoic acid in four-grain doses three times a day.

With regard to the treatment of the various diseases which may give rise to *jaundice with pyrexia*, there is but little to be said; they are almost more invariably fatal than acute atrophy itself. The administration of quinine may somewhat lower the pyrexia, and if the issue should be at all doubtful, may perhaps incline the balance in the patient's favour; while stimulants seem indicated, as in other cases of infective fever.

When *permanent* jaundice has once declared itself, and the obstruction of the common duct is complete and irremediable, it is no longer advisable to prescribe carbonate of soda, or dilute nitro-hydrochloric acid, or taraxacum. The more faith we have in the efficacy of those remedies in simple jaundice, the more we shall fear that they may now do harm. Regulation of the diet is perhaps the most important part of the treatment. It has been shown experimentally by Bidder and Schmidt in Germany, and by Bennett and Rutherford in this country, that dogs with artificial biliary fistula may live for years provided they are supplied with and will take a sufficiently large quantity of food. It is true that in those animals the escape of bile through the fistula causes a drain which is wanting in jaundice in the human subject, but the experiments at least suggest the conclusion that a large supply of nutriment should if possible be maintained. At the same time its quality should be carefully attended to. In dogs whose common duct has been tied the daily quantity of fat that can he absorbed from the food is greatly diminished. Evidently, therefore, oleaginous and fatty articles of diet should be taken very sparingly, if at all, by persons with permanent jaundice.

Something may be done to counteract the absence of bile in the intestines by the administration of the purified bile of the ox or pig. Murchison recommends that this should be given in doses of from three to six grains, about two hours after meals, in capsules or pills coated with a solution of tolu in ether, so that they may pass through the stomach unaltered. A glycerin extract of the pancreas may possibly do good in such cases.

Ox-gall has the further advantage of taking up the antiseptic function of the natural bile. With this object, as well as that of relieving the flatulence, creosote, thymol, turpentine, vegetable charcoal, &c., may likewise be prescribed with benefit. Occasional laxatives are generally required, and the milder ones should be preferred.

The *itching* caused by jaundice is sometimes so troublesome as to require special treatment. Warm alkaline baths have sometimes proved serviceable. The use of the flesh-brush is recommended by Murchison, and the internal administration of the bicarbonate of potass. Bicarbonate and bromide of sodium, in full doses, are sometimes effectual. Very often, however, this symptom is one which baffles all efforts to relieve it. Belladonna internally and a hydrocyanic acid lotion externally are probably the best means of

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relief. Opiates must sometimes be resorted to, but not infrequently they aggravate the irritation. Dr Goodhart has found the injection of pilocarpine useful.

GALL-STONES.—One of the most frequent and important causes of jaundice is obstruction of the common bile-duct by calculi; the impediment to the flow of bile into the duodenum is then obvious and demonstrable. They are probably formed in the gall-bladder, not in the duct.

Gall-stones are of two kinds. Some consist almost entirely of bile-pigment; others are made up mainly of cholesterine.

The former are small, dark reddish, olive-brown, or almost black in colour, irregular in outline, and so soft that on pressure they break down into a coarse gritty powder. They are small and multiple, often counted by scores or hundreds. Baillie records more than 1000 taken from a single gall-bladder, and preserved in Dr William Hunter's museum.

The latter kind are hard and smooth on the surface; they split with a semi-crystalline fracture, displaying lines radiating from their centre, with the glistening aspect of cholesterine. In size and colour they are very variable. Some are three and a half to four inches in circumference; a gall-stone of this size is generally single, and fills the whole gall-bladder, so that it has one rounded end answering to the fundus, and another tapering, which corresponds with the cystic duct. Others are small, of the size of marbles, peas, or scarcely larger than pins' heads. Several are often found in the same gall-bladder, and sometimes an enormous number; but this is not so frequent as with pigment-calculi. Their surface is usually white or stone-coloured, and the superficial layers are also pale—often separated by a well-marked dark shell (seen as a band in section) from the deeper layers, which have generally a more or less deep yellow or brown hue from bile-pigment.

Cholesterine calculi are seen on section to be made up of concentric layers, which show radiating glistening lines in each successive layer, and break with a crystalline fracture. The origin is probably a little inspissated mucus impregnated with lime-salts. To this bilirubin is attracted, and so a nucleus is formed, upon which cholesterine is slowly deposited.

Age and sex.—The liability to the formation of gall-stones increases as persons advance in years. They scarcely ever occur in children, and are rare under fifty years in men and under forty in women. Sometimes, however, gall-stones are found in those who are only twenty-five or thirty.

Women are much more liable to gall-stones than men. Perhaps this is due to the fact that they are more apt to lead sedentary lives and to become stout.

Symptoms.—Calculi are frequently latent. They are often discovered in the dead bodies of those who during life had not complained of any symptoms. In these cases, however, they are not found in the bile-ducts, but in the gall-bladder, which is sometimes closely contracted over them. In such cases they have no clinical importance. When the cystic duct is blocked by a calculus, the walls of the gall-bladder go on pouring out mucus. It may then become much enlarged and form a tumour in the abdomen below the liver, which may be mistaken for a pendulous hydatid cyst.

When the calculus is impacted in the common bile-duct severe symptoms follow, and constitute what is termed a fit of the gall-stone, or biliary colic. First comes sudden and agonising *pain*, sometimes so excruciating that the

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patient is bent double and rolls upon the floor, and cannot restrain cries of pain. Epileptiform convulsions are said to have been observed. After a while the intensity of suffering abates, and it is replaced by a constant dull aching, which continues until the more acute pain returns. The seat to which these agonising sensations are principally referred is the right hypochondrium, but generally they also shoot into the right scapular region and back, and downwards to the navel, or they spread over a large part of the abdomen.

Another marked symptom is *shivering*. The face is pale, the skin cool, and the whole body often covered with a cold sweat. The *temperature* is, as a rule, normal. Sir Dyce Duckworth has, however, seen several cases of biliary colic in which there has been pyrexia, in one of which the temperature rose nearly to 104°, and remained high for several days. In these cases there is probably suppuration, or at least acute inflammation of the gall-bladder or duct. Murchison also mentions that pyrexia is not uncommon.

The *pulse* is much reduced in force and volume; sometimes it is slow, sometimes quickened, but more generally it is of normal frequency. There is great exhaustion, the patient may swoon away, and it is said that fatal collapse has been known to set in.

Vomiting is a very frequent and characteristic symptom, and hiccough is not uncommon.

Jaundice is not one of the earliest symptoms of an "attack of gall-stones." It is evident that until the calculus has passed from the cystic into the common duct no jaundice will arise. Generally speaking, however, after a few hours, or at the longest a couple of days, the patient's urine contains bile-pigment, and a little later the conjunctivæ and the skin become yellow.

It is often supposed that an attack of biliary colic is invariably attended with pain. But a few years ago a case occurred at Guy's Hospital in which a man died in a surgical ward of hernia who had previously had jaundice, which (it is expressly said) was unattended with pain. The gall-bladder contained numerous gall-stones, and the common duct was dilated so as to admit the finger.

An attack of gall-stones may terminate in several different ways. Most commonly the jaundice sooner or later subsides; sometimes it passes off in three or four days, sometimes it lasts several months. Indeed, even the shorter period exceeds the limit within which an attack of gall-stones may occasionally run its course, for it may end in twenty-four or thirty-six hours; but in that case it is unattended with jaundice, which, as we have seen, seldom appears until the pain has lasted for some time. A good example of protracted jaundice from gall-stone is given by Murchison, that of a man who was jaundiced continuously for more than six months. Even in his case, however, the pain was not constant, but repeatedly went away for a week at a time. At last the jaundice disappeared, and the man returned to work.

When an attack of jaundice from gall-stones subsides it is usually because the calculus has passed into the duodenum. The next thing is for it to be voided in the fæces, in which it may often be found without much difficulty. Formerly writers said that if water is added to a stool containing a gall-stone, the latter will rise to the surface, from being lighter than the liquid; but it is now known that this is a mistake. When first voided gall-stones have really a higher specific gravity than water; it is only when they have

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been dried that they float. The best way to detect a gall-stone in the fæces is that recommended by Murchison, namely, to mix them with water and pass the whole of it through a sieve. In some cases, however, after the subsidence of an attack, no gall-stone can be found, in spite of careful search. Possibly it is retained for a time in the bowels, or it may have undergone disintegration, particularly if it was one of those friable calculi which consist almost entirely of bile-pigment; or, again, it may never have escaped into the duodenum, but have slipped back into the gall-bladder. The last alternative is the one that comes most naturally into one's mind when a patient has numerous and transitory attacks of biliary colic in quick succession, and when yet no calculus can be found in the evacuations. One is then apt to suppose that all the attacks are caused by a single gall-stone slipping to and fro in the duct. But it must not be forgotten that a very large number of calculi are sometimes present in the same gall-bladder, and that a great many have been found in succession in the fæces. Sir Thomas Watson relates the case of a patient who collected fifty-five calculi from his stools within a space of five weeks. The discovery of the concretion after an attack of jaundice is not only important as verifying the diagnosis, but it may also help to answer the question whether the complaint is likely to recur. If the gall-stone was alone in the gall-bladder its form is rounded; but if it was one of several it is very likely to show flat surfaces or facets where it touched the stones in contact with it.

Results.—As above stated, attacks of gall-stones have been reported so severe as to prove directly fatal. But it is doubtful whether, in any well-authenticated case, an autopsy has shown that death was really due to this cause, and not to concomitant inflammation of the ducts. Sometimes, although happily rarely, a gall-stone sets up ulceration, which reaches the peritoneal surface of the gall-bladder, allows bile to escape into the serous cavity, and fatal peritonitis ensues.

Some years ago a woman in Guy's Hospital, who for some few years had repeated attacks of jaundice, for four or five weeks suffered continuously from this symptom and from pain in the abdomen, which became more severe until she died. At the autopsy acute peritonitis was discovered, which had been caused by the escape of bile through an ulcerated opening in the hepatic duct. The common bile-duct was obstructed by a gall-stone. Murchison relates the case of a lady who died in about a week from a second attack of jaundice, and in whom the fatal result was due to peritonitis set up by perforating ulcers in the fundus of the gall-bladder, themselves caused by gall-stones.

In other cases the ulceration of the gall-bladder or biliary passages caused by gall-stones has set up a local pyæmia, attended with the formation of abscesses in the liver, and leading within two or three weeks to a fatal result.

Biliary colic frequently occurs over and over again in the same individual, and a patient who has had this complaint once should therefore always be warned that he is likely to suffer from it again. Sometimes, but not always, the first attack is the most severe. When a concretion of some size has once passed through the common duct into the duodenum, it is easier for another calculus of the same size to perform the same journey. The cases in which biliary colic terminates rapidly within a few hours, and even without causing jaundice, are chiefly those in which several previous attacks of the same complaint have occurred. These instances of repeatedly

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recurring biliary colic are often very trying both to the patient and to his medical attendant; but in the majority of cases the attacks sooner or later cease to return. At any rate, in several instances of this kind the patients, even when advanced in years, have afterwards enjoyed excellent health, and have ultimately died of some other disease.

Sometimes, however, the jaundice persists until death. The termination of such a case may be due to some complication. In one instance it resulted from erysipelas of the face; in another from the supervention, at the same time, of acute endocarditis and acute meningitis. In another case, again, the patient fell into a comatose state, and died in a fortnight after the commencement of his last attack of jaundice; and a fourth case was probably similar, of which no history is preserved beyond the facts that it proved fatal a few days after admission to hospital, and that the body was well nourished, as in death from some acute disease.

During the twenty-one years from 1854 to 1874 inclusive no case is recorded in our pathological reports at Guy's Hospital in which death occurred with chronic exhaustion and wasting as the simple result of the jaundice due to impaction of a gall-stone in the common bile-duct. The twenty-five volumes of the 'Pathological Transactions,' again, contain only two such cases; and even these may be said to have proved fatal by complications. One is recorded by Murchison: the patient, who had for many years been subject to attacks of gall-stones, died after six months of jaundice, having suffered during the last three weeks from greatly increased pain and vomiting, with hæmorrhage from mucous membranes; the common bile-duct was obstructed by a large cylindrical gall-stone, which was ulcerating into the bowel by the side of the orifice of the duct. The other case came under the observation of Dr Wale Hicks; the patient died seven months after the attack commenced, but the jaundice, instead of being persistent, gradually faded, and at last entirely disappeared, and the hepatic tissue was found to have broken down into granular matter and oil; moreover, the obstruction in this case was not complete.

One remarkable result of the presence of gall-stones is the development of cancer of the gall-bladder or the bile-ducts. Within the period of twenty-one years already referred to there were at least twelve cases in which, gall-stones being present, there has been likewise malignant disease of these structures. In some instances the clinical history has pointed distinctly to the view that the jaundice was originally due to an ordinary attack of biliary colic, and that the development of the cancer was secondary; indeed, one case seems to admit of no other interpretation. A man aged forty-five died of jaundice which had lasted four months. Dr Moxon found that the gall-bladder was very large, containing hundreds of gall-stones; the common bile-duct at its commencement was greatly narrowed, and its walls were thickened by a cancerous growth; *below* the narrowed spot it contained three or four facetted gall-stones, just like those in the gall-bladder. This part of the duct was also dilated, and had evidently been accustomed to the passage of gall-stones before the cancer had begun to form. But in the great majority of the cases in question no gall-stone has been impacted in the duct at the seat of the cancer; the concretions have been found in the gall-bladder itself, which has often been contracted round them, and empty, or containing only a little purulent mucus. Thus it appears probable that if the malignant growth had not developed itself, all the symptoms would have subsided, and the health of the patient would have been restored.

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Probably cancer of the bile-ducts in association with gall-stones arises as the result of their irritation; whereas, when gall-stones are discovered in the bodies of those who have died from cancer of the breast or of other organs, this is probably due to the fact that both cancer and gall-stones are apt to occur in elderly persons. Whatever the explanation is, the fact remains true that when a patient who has had attacks of biliary colic dies of protracted jaundice the ducts are found as a rule to be affected with cancer.

Treatment of gall-stones.—In an attack of "biliary colic" the patient should first be placed in a hot bath; and afterwards fomentations or poultices should be applied to the abdomen. If there be much tenderness on pressure in the right hypochondrium, a few leeches often give great relief, according to the testimony of many writers. But these measures will not suffice for the relief of the agonising pain without the administration of opium or morphia, in full doses and frequently repeated. In a patient previously in good health two grains of opium are not too much to begin with, followed by a grain every two or three hours until ease or sleep is obtained, it being of course understood that the effects are carefully watched, and that he has no albuminuria. Very often the stomach is too irritable to retain the anodyne, and then the subcutaneous injection of a quarter of a grain of morphia may be resorted to with signal advantage. Another antispasmodic, of which Murchison speaks highly, is the extract of belladonna, which he prescribed for this purpose in half-grain doses. The inhalation of chloroform has sometimes proved very effectual, and is certainly the speediest and most effectual way of relieving the pain when it is at its worst.

Dr Prout recommended the administration of large draughts of hot water, containing one or two drachms of the bicarbonate of soda to the pint; and even when the stomach rejected the first portion of the fluid, he used to persevere, believing that it diminished the severity of the retching. If, however, the vomiting be very violent, it should be checked by effervescing draughts, dilute hydrocyanic acid, and the like. Many of the older physicians, and even Bright, prescribed antimony in the treatment of biliary colic, with the hope of relaxing spasm, and so facilitating the expulsion of the calculus; but this remedy is now justly discarded on account of its tendency to aggravate the vomiting. A mixture of turpentine and ether is a once famous remedy for biliary colic, but it is usually rejected by the stomach of modern patients.

There are no means at present known of *preventing* the formation of gall-stones, or dissolving them before they become impacted. Active exercise, spare diet, and occasional cholagogues are believed to be of some service, and the sulphate and phosphate of soda are often prescribed. Chloroform is a chemical solvent of cholesterine and of bilirubin; hence it has been given when gall-stones were suspected; five or ten up to thirty drops may be taken in spirits and water ('Brit. Med. Journ.,' 1890, i, p. 50). Perhaps a better plan is to give chloral hydrate in doses of ten or fifteen grains. The succinate of the peroxide of iron has been recommended as a solvent, but without even theoretical probability of its being useful.*

* It is a question whether the treatment of the effects of biliary colic by mercurials is wisely abandoned in the present day. Sir Walter Scott had a most severe attack of this kind, as the pain, vomiting, and jaundice prove, though it was treated as "cramp of the stomach," and writes, July, 1819: "No less a deity descended to my aid than the potent Mercury himself, in the shape of calomel, which I have been obliged to take daily, though in small quantities, for these two months past. Notwithstanding the inconveniences of this

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Operative treatment (cholelithotomy) has in recent years been sometimes followed by brilliant success.

Inflammation of gall-bladder and biliary passages.—The catarrhal inflammation of the common bile-duct which has been supposed to be the cause of "simple" jaundice, is, as we have seen (p. 340), a purely hypothetical affection. But we occasionally find the ducts in a state of suppuration. This is usually the result of the irritation of a gall-stone, which may lead to ulceration of the mucous membrane, and that again to a cicatricial stricture.

Still more frequently the *gall-bladder* is inflammed by the presence of a calculus, and suppurating may burst into the peritoneum or open externally or into the colon. In a case recently in the writer's knowledge, an elderly gentleman died from what could only be described as idiopathic suppuration of the gall-bladder with consequent portal pyæmia. There had been no history of jaundice or biliary colic, but if such cases of ulceration and suppuration are not due to calculi, their pathology is quite unknown.

When the gall-bladder is distended by an impacted calculus, it occasionally becomes adherent to the abdominal parietes; an abscess may then be developed which points externally, and when it breaks or is opened by a surgeon, gall-stones are discharged with the pus. The site of the external opening is by no means always directly over the gall-bladder; it may be at the umbilicus, or even in the left side of the abdomen; nay, a case has been recorded in which two biliary calculi made their way into the connective tissue of the vagina. It is important to note that in cases of this kind there are (or may be) no symptoms of the presence of gall-stones until they are found in the discharge. The common bile-duct is often quite free, while the cystic duct is completely closed; and thus neither is there any jaundice, nor does any bile enter the gall-bladder and mix with the pus. Hence the abscess is often supposed to be seated in the abdominal walls, or in the substance of the liver, and months or even years may pass before the real nature of the case declares itself. In the meantime the patient has a fistulous opening in the side, which, however, need not prevent the enjoyment of good health; and when all the gall-stones have come away it may at length heal up.

The *treatment* of gall-stones and of a suppurating gall-bladder by operation, has been followed in recent times by some remarkable successes (see Dr Marion Sims' case, 'Brit. Med. Journ.,' June 8th, 1878, p. 811; and Mr Lawson Tait's, 'Med.-Chir. Trans.,' 1880, and subsequently; also Mr. Mayo Robson's, *ibid.*, 1890, and 'Clin. Trans.,' Oct. 25th, 1889). Reference to earlier proposals of the operation, and isolated cases of its execution, will be found in the 'London Med. Recorder,' April 15th, 1881, p. 153. During the last few years cholecystotomy has become a recognised operation, and though it must always be dangerous, in the hands of skilful surgeons it has undoubtedly saved many lives (see Sir Spencer Wells' remarks in his "Bradshaw Lecture," 'Lancet,' Dec., 1890).

Gall-stones in the intestines.—When a gall-stone escapes by ulceration into some part of the intestine it may be voided *per rectum;* and since a concretion which takes this course is often very large, its passage through

remedy, I thrive on it most marvellously, having recovered both sleep and appetite." ('Lockhart's Life,' vol. vi, p. 113.)

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the anal orifice may be attended with severe pain and violent straining, the cause of which cannot be explained until the gall-stone is discovered. Probably when a large stone thus makes its way out of the body it has passed from the gall-bladder directly into the hepatic flexure of the colon; but more often, when the stone is of moderate size, it is with the duodenum that an ulcerated gall-bladder communicates.

As mentioned in a previous chapter (p. 253), the gall-stone may then become impacted in the jejunum or the ileum, so as to produce obstruction of the bowel.

The recognition of this condition is, as we saw, difficult, and rests usually on probability derived from the age and sex of the patient and previous accounts of jaundice and pain.

The treatment is by opium, so as to relax the grip of the bowel on the gall-stone. When, after abdominal section for obstruction, the cause has been found to be an impacted calculus, it has sometimes been possible to manipulate it through the ileo-colic valve, and safely leave it there. In other cases the gut has been opened and the stones extracted with complete success. Crushing the calculus by means of padded forceps without wounding the intestine, and breaking it up by means of a needle, are methods which have been suggested, but not as yet carried out.

Weil's disease.—Cases of jaundice with enlarged and tender liver have been described as occurring epidemically in certain towns of Germany, and also in Servia and in Egypt. They have been grouped together and named after the physician who recorded some cases in 1886. He found it occur among male adults, and usually among butchers, but this was probably an accident. It runs an acute febrile course for eight or ten days, is not contagious, is apt to relapse, and has a favourable issue. Beside the enlarged and tender liver, the jaundice and pyrexia, there is a slow pulse and occasionally albuminuria. The pathology is unknown, but probably many cases are icterus from impacted gall-stones with inflammation of the bile-ducts, and this would agree with the fact that sometimes the result is permanent jaundice.

Other cases of epidemic jaundice referred to the same category appear rather to belong to relapsing or remittent fever, and some which ended fatally with hæmorrhage in various organs are more like acute yellow atrophy. (See 'Wiener med. Wochenschrift,' No. 26, 1890.)

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INFLAMMATORY DISEASES OF THE LIVER

"And let my liver rather heat with wine
Than my heart cool with mortifying groans."
Merchant of Venice.

ACUTE SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER—*Geographical distribution—Ætiology—Relation to dysenteric ulceration—Statistics—Morbid anatomy—Characters of the pus—Direction of rupture—Symptoms—Physical signs—Diagnosis from acute perihepatitis—from abscess between the liver and diaphragm—from suppurating hydatid or gall-bladder—Prognosis—Treatment of acute hepatitis—Paracentesis of hepatic abscess.*

CHRONIC INTERSTITIAL HEPATITIS—CIRRHOSIS OF THE LIVER—*History—Anatomy—Ætiology—Effects upon the portal circulation—Early symptoms—Hæmorrhage—Jaundice—Ascites—Cerebral symptoms—Hypertrophic cirrhosis—Prognosis and treatment.*

Perihepatitis—Hypertrophy—Simple chronic atrophy—Syphilitic hepatitis and gummata of the liver.

ACUTE ATROPHIC HEPATITIS—ACUTE YELLOW ATROPHY OF THE LIVER—*History—Symptoms—jaundice—percussion—the urine—cerebral symptoms—hæmorrhage—Course and event—Anatomy—Ætiology and pathology—Diagnosis—Prognosis—Statistics—Treatment.*

PASSING from disorders of the secretory functions of the liver to its structural diseases, we will take first hepatic inflammation.

The liver is not liable to ordinary simple, primary, or idiopathic inflammation as the result of cold, like bronchitis and pleurisy, nor to acute inflammation secondary to a definite preceding cause, as endocarditis to rheumatism. The substance of the liver, consisting of epithelial cells and connective tissue with blood-vessels, is subject to the following three forms of hepatitis, different in their pathology, and no less different in their clinical features:

1. Acute suppurative inflammation, producing either a single abscess or multiple pyæmic abscesses like those of septic lobular pneumonia. This we may compare with abscess of the brain.

2. Chronic interstitial inflammation with subsequent fibrous change, contraction, and hardening, leading to atrophy of the glandular parenchyma—"cirrhosis," a disease closely analogous in its pathology to sclerosis of the spinal cord, to chronic interstitial pneumonia, and to the most chronic form of Bright's disease. It is from this analogy that we commonly speak of cirrhosis of the lung and cirrhosis of the kidneys, while some pathologists name the corresponding hepatic disease, not cirrhosis, but sclerosis of the liver.

3. There remains the most rare, most obscure, and most remarkable disease of all that affect this organ—an acute affection which leads to rapid atrophy, and is wanting in many of the characteristics of inflammation. It is open to question whether it should be regarded as hepatitis at

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all. But it appears to bear some analogy to acute pneumonia on the one hand and to acute Bright's disease on the other; and unless we admit it to be a parenchymatous inflammation, as peculiar to the liver as pneumonia is to the lung, it is altogether unique in pathology no less than in its clinical aspect, and we must either treat of it apart, or name it by one of its most striking features, Icterus gravis, and group it (as was done in the first edition of this work) with jaundice from obstruction. On the whole it appears best to place it near hepatic abscess and cirrhosis; but the arrangement is mainly one of convenience, and each of the three sections of the present chapter will be treated independently.

ACUTE SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER.—In the liver, as in the other solid viscera (kidneys, spleen, lungs, testes, brain), ordinary inflammation does not end in suppuration. With the important but still doubtful exception of tropical abscess, hepatic suppuration only occurs as the result of septic, bacterial, infective inflammation. Abscesses of the liver are frequent in cases of pyæmia, general or portal, and in the latter affection pus may be found in the portal canals throughout the organ.

Tubercular and typhoid ulceration do not produce multiple pyæmic abscesses, while dysentery does.

Primary hepatic abscess in India, China, and other hot climates is very common, and indeed takes an important place in the European death-rate. It is therefore not without reason called tropical abscess of the liver. In the West Indies it is said to be comparatively rare.

In England, apart from pyæmia, abscess of the liver, and particularly a large single abscess, is very rare. Indeed, some writers have stated that it is never seen except in those who have lived in a hot climate. This statement, however, is not accurate. Dr Fagge collected in Guy's Hospital fifteen cases in which death was caused by the formation of a single large abscess (or in one instance two large abscesses) in the liver. Only five of these cases occurred in persons who had come from China, or India, or the West Coast of Africa; but in ten there was no such history, and most of the patients it was positively stated had never been out of England.

Dr H. J. Campbell has extracted for this third edition sixty-nine cases of hepatic suppuration from the *post-mortem* records of Guy's Hospital during the last twenty years, 1870—1889. Forty-three of the patients were men and twenty-six women; the ages varied from childhood to old age. In forty-six the abscesses were multiple and in twenty-three single.

The causes were very similar in both cases; there was no instance of an idiopathic tropical abscess. General, surgical or internal, pyæmia accounted for fourteen multiple, and three single, abscesses. Portal pyæmia from ulceration, in several cases dysenteric, and in some of these tropical, accounted for eleven cases of multiple and eleven of single abscess. Pyæmia from the female pelvic organs produced four cases of multiple and one of single abscess, and prostatic pyæmia one case of single abscess. A suppurating hydatid cyst furnished seven cases; injury to the ribs one. Extension of inflammation from a perforating ulcer of the stomach or duodenum, or from suppuration around a cancerous growth, led to seven cases. Suppuration of the bile-ducts, usually from an impacted calculus, is a frequent cause of multiple hepatic abscesses, and led to six cases of this series. Lastly, one case was due to actinomycosis.

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The following is the complete tabular statement:

	Single.	Multiple.	Total.
Injury to ribs	1	0	1
Extension from a neighbouring organ	2	5	7
Suppurating hydatid	4	3	7
Suppuration of bile-ducts	0	6	6
General pyæmia	3	16	19
Intestinal pyæmia	11	11	22
Pelvic pyæmia	2	4	6
Actinomycosis	0	1	1
	23	46	69

All the above sixty-nine cases were fatal, but several have occurred in which the abscess has been opened with a good result.

The following cases, which have been under the writer's care, are not included in the above list:

Three were tropical; one traumatic; several from general or portal pyæmia (mostly dysenteric or pelvic in origin); one from perforation of a gastric ulcer; one, beside a second among the sixty-nine, was due to inflammation of the appendix; and one to a basal vomica in the lung perforating the diaphragm. Two others turned out to be suppurating hydatid cysts, and one to be due to actinomycosis.

It has long been known that tropical abscess in the liver and dysentery are often associated, and various opinions have been held as to the connection between these two diseases. Annesley supposed that sometimes, as the result of an hepatic abscess, the bile acquires peculiarly irritating properties, and thus sets up inflammation and ulceration of the intestine. Dr George Budd in 1842 first taught that dysentery is the earlier of the two diseases, and that abscess in the liver is the result of absorption from one of the intestinal ulcers; in other words, that the pathology of the so-called tropical or single abscess of the liver is essentially the same as that of the multiple abscesses of portal pyæmia. Dr Budd's view for some time received general acceptance, but it is rejected by almost all recent writers of experience in tropical diseases.

It is argued that if dysenteric ulcers in the colon were the cause of abscess of the liver, the same result ought to follow other forms of intestinal ulceration, such as occur in phthisis or in enteric fever.

Another argument is that many cases of hepatic abscess, in which recovery takes place, run their whole course without any symptoms of dysentery. It is, however, possible for dysentery to begin insidiously, or to remain altogether latent (cf. p. 234). In England this is often the case, and it probably is so in India likewise.

Again, it is said that abscess of the liver does not occur in some epidemics of dysentery, and is comparatively uncommon in certain countries where dysentery prevails—in China, for instance, as compared with India. That Dr Baly did not meet with it in the epidemic of dysentery at Millbank may perhaps be explained by a certain length of time being required for the development of suppuration in the liver, which thus may be wanting in rapidly fatal epidemics.

But the strongest argument that abscess of the liver is not secondary to dysentery, is the fact that many cases have been recorded in which, after death from tropical abscess, the intestines have been found to present no sign of past or present inflammation. Murchison met with a case of this kind in a European soldier in Burmah. The man had never had dysentery,

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although while under observation he was suffering from persistent diarrhœa. He died, and an enormous abscess, holding four quarts of pus, was found in the liver, but neither the small nor the large intestines nor the stomach presented any cicatrices or trace of recent ulceration. Again, Mr Waring collected 204 cases of abscess of the liver, in exactly one fourth of which the intestine is said to have been perfectly healthy, and Dr Morehead mentions that he has notes of twenty-one similar cases.

Among the fifteen fatal cases of large abscess of the liver at Guy's Hospital there were three in which no sign of ulceration was found in the intestines; one of these was a tropical case. In eleven others it is expressly stated that the bowels were or had been diseased. In one case which came under Dr Moxon's observation there was only a minute cicatrix in the gut, so small that it might easily have been overlooked ('Path. Trans.,' vol. xxiv). Among the sixty-nine cases, there was disease of the bowels in twenty-two.

It seems premature to give up the theory that abscess of the liver is secondary to dysentery until fresh facts shall have proved that this theory is untenable. The positive observations which support it are very strong; in this country dysentery and hepatic abscess are each so rare that their frequent association would be a most extraordinary circumstance unless they are more than casually connected; and it is the rule that the ætiology of a disease can best be studied, and its origin best unravelled, in the countries where it is not too common.

Even in Dr Murchison's cases dysentery was present in three fourths.

In India the current opinion at present would appear to be that dysentery and abscess of the liver are common results of the same causes; or it is supposed that inflammation extends from the mucous membrane of the intestine to the largest gland which opens into it, just as it does from the urethra to the testes in cases of gonorrhœa. The latter analogy would account for the occurrence of hepatitis, but not for the formation of a circumscribed abscess; and we might still ask, why not also abscess of the pancreas? Moreover, the duodenum is seldom, if ever, the seat of dysenteric ulceration.

The connection between dysentery and hepatic abscess is far more probably by the veins of the portal system.

If previous dysenteric ulceration be rejected as a cause of hepatic abscess, no other can be assigned. Dr Morehead thinks that elevation of the temperature may sometimes explain the occurrence of hepatitis during the hot months of the year in plethoric persons newly arrived in India. But it appears from the statistics of the European General Hospital in Bombay that the admissions of patients with hepatitis are relatively more numerous during the months which follow the cold season, and during the cold season itself, than during the hot months. Hence if heat be concerned in production of abscess of the liver, it can be only as a predisposing cause; while exposure to cold may possibly act as an exciting cause. That neither one nor the other is sufficient of itself, nor yet the two together, is obvious.

There is no reason to believe that intemperance has anything to do with the disease.

Local injury—such as a blow in the right hypochondrium—has sometimes been supposed to set up suppuration in the liver. A case in point occurred at Guy's Hospital in 1876. A drunken woman was run over, but

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the ribs were not fractured. She died with symptoms of pneumonia of the right base, and after death there was found in addition a large abscess in the right lobe of the liver, which had not perforated the diaphragm. A similar case under the writer's care was that of a little boy who in like manner had his ribs injured but not broken.

In a remarkable case, which happened in 1881, an abscess in the liver was caused by the perforation of a large phthisical vomica in the base of the right lung, which opened into the liver.

In a case, which occurred under the writer's care in the same year, two acute abscesses of the liver in a boy of ten were found after death to have been caused by a pin lodged in the appendix cæci; a similar example was recorded by Dr Payne in the 'Pathological Transactions' for 1870 (vol. xxi, p. 232).

Excluding cases of general pyæmia, suppurating hydatid cysts, and tropical cases in soldiers and sailors from abroad, hepatic abscesses in Guy's Hospital have been, with few exceptions, due to portal pyæmia; from dysentery, ulcerative colitis, fistula *in ano*, gastric ulcer, or typhlitis.

Anatomy.—The pathological processes which are concerned in the production of abscesses of the liver have been minutely studied by German observers, and are fully described by Rindfleisch. He distinguishes a "thrombotic" from an "embolic" variety. In the former the inflammation occupies the walls of the interlobular branches of the portal vein. These, and the sheath of connective tissue round them, are swollen by an infiltration of leucocytes until the columns of hepatic cells become compressed and perish. The adjacent masses of infiltrated connective tissue then come into contact, and they form small white nodules, which are very similar in size to lobules. These "pseudo-lobules" in their turn melt away, and form an abscess cavity. But fresh suppurative foci are constantly making their appearance at its periphery, and give a ragged character to its inner surface. In the "embolic" variety the portion of hepatic tissue which corresponds to the distribution of the plugged vessel becomes intensely congested, the circulation in it is entirely arrested, and it sloughs *en masse*. The lobules round it undergo reactive inflammation; they become enlarged, and those nearest the sloughing part are permeated by numerous pus-corpuscles, which lie outside the secreting cells, between them and the capillaries.

The hepatic cells appear to take no active share in the formation of pus, which begins in the connective tissue.

It should be added that the observations on which these statements rest appear to have been made in cases of the small multiple abscesses of pyæmia, which are not now under consideration; but it is probable that they may be applied generally.

The characters of abscesses of the liver vary greatly in different cases. When of recent formation and rapid growth they may possess no limiting membrane whatever, the pus lying in an irregular cavity formed by reddened and softened hepatic tissue. If they have been of somewhat longer standing they are lined with a layer of opaque yellowish material, the formation of which has been described above. Very old abscesses have a dense fibrous wall, which may be three or four lines in thickness and so hard as to feel like cartilage, or it may even be partly calcified. Sometimes, in making an autopsy, one finds such an encysted and indurated abscess more or less detached from the surrounding tissues and bathed in pus, so

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as to be limited only by the substance of the liver. In such cases no doubt the abscess has been of long standing, but has undergone rapid extension shortly before death.

The pus of an hepatic abscess is, according to Dr Budd, yellowish and free from odour. But the records of autopsies at Guy's Hospital state that in three cases it was greenish and either mucoid or curdy, and in two others reddish or reddish brown in colour. This is of some interest, because Budd asserted that the pus of an abscess in the liver is never red so long as it is confined *in situ*, but becomes so when it is expectorated through the lung. He regarded this kind of expectoration as affording conclusive evidence of the existence of an hepatic abscess. But Dr Morehead, who at one time held the same view, says that in more than one case of pneumonia he has observed sputa having precisely the same characters. In one case of hepatic abscess that came under the author's care in Guy's Hospital some years ago, the fluid removed by the trocar was of a brickdust colour, looking not unlike anchovy sauce; and it had a most peculiar and nauseous odour.

An abscess of the liver may sometimes attain an extraordinary size. We once found one to hold six pints; but Maclean mentions one which contained altogether nineteen.

When the pus has reached the surface of the liver, it may point externally in the right hypochondriac or epigastric regions; or it may rupture into the peritoneum; or it may discharge itself into the stomach, duodenum, or colon. In this last event the patient may vomit a considerable quantity of matter, or pass it *per rectum*, but in many cases the pus cannot be traced. Sometimes, again, it burrows towards the lumbar region, or it may perforate the diaphragm into the pericardium or the pleura, or if adhesions have previously formed, into the lung, and thus be evacuated through the trachea.*

Symptoms.—In many cases abscess of the liver remains entirely *latent*, and is found after death in the bodies of those who have never been known to suffer from any symptoms of the disease. Cases of this kind are mentioned by all writers on tropical diseases, for it is chiefly in persons who have been in hot countries that latent abscess of the liver has been met with. The author once examined the body of a gentleman who died of protracted diarrhœa a few years after his return from China. There was an abscess the size of a walnut in the back of the liver, although careful inquiry during the patient's life had failed to elicit any evidence that the liver was otherwise than perfectly healthy.

Again, an abscess of the liver, which up to a certain point has gone on increasing in size without affecting the patient's health, may suddenly give rise to the most serious symptoms. Maclean mentions the case (occurring in the Mauritius) of a man, apparently in good health, who had walked seven or eight miles in search of employment, when he complained of pain at the pit of the stomach, and in a few hours died; an abscess of the liver, lined with a dense fibrous membrane, had burst into the pericardium. Another case is that of a man who had been invalided from India on account of "chronic hepatitis," but who when he arrived at Fort Pitt had apparently recovered so completely that he was sent to the depôt for duty. Some weeks afterwards, while he was straining at stool, "something gave

* Dr Morehead says he has seen three cases in which a patient suffering from hepatic abscess expectorated pus, but no perforation could be discovered after death.

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way;" and this proved to be an hepatic abscess, which, as in the last case, had ruptured into the pericardium. Maclean supposes that the thick capsule which most of these latent abscesses possess prevents their causing constitutional irritation, by forming a barrier, so to speak, against the disturbing influence of the pus. But the growth of a dense capsule is of course the work of time; and the abscess in these cases is latent from the very first, before any capsule exists.

In most cases, however, abscess of the liver gives rise to symptoms which are strongly indicative of its presence; and the results of physical examination of the abdomen are often conclusive. The symptoms are a sense of fulness and weight, or even pain, in the right hypochondrium, pain in the right shoulder, inability to lie on the right side, fever, rigors, and cough.

The *pain* which attends abscess of the liver is exceedingly variable in degree. It is often much more intense in the right shoulder than in the hypochondriac regions. Budd mentions a case in which the pain in the shoulder was for a long time most severe; and when the abscess was opened the pain was relieved. The pain over the liver itself is often increased by pressure, and also by the patient drawing a deep breath, or turning over on to the right side.

In cases of hepatic abscess *cough* is often present. It is generally short and dry, and is no doubt due to reflex irritation.

Pyrexia is often a marked symptom. Maclean states that in every case of suppuration of the liver that had recently been under his observation at Netley the thermometer showed a rise of from one to three degrees. But these were, no doubt, cases in which the existence of abscess was suspected on other grounds; and we cannot be sure that fever would always reveal its presence if it were undiscoverable by other means. In the case above mentioned, of hepatic abscess from a pin in the appendix cæci, the temperature rose to 106°; and this, with the great increase of hepatic dulness, local tenderness, and absence of other causes of fever, led to the diagnosis of suppuration of the liver.

Dr Morehead believes that *rigors* are not of much value as indicative of suppuration in the liver; they may be present when there is no abscess; and, on the other hand, they are often absent when an abscess is forming.

Jaundice appears not to be a symptom of single tropical abscess, unless by a rare accident, from pressure upon one of the main ducts. With multiple pyæmic abscesses it is frequent (cf. p. 342).*

The *physical signs* of hepatic abscess depend mainly upon its position and its size. Dr Twining, of Calcutta, believed that even a deep-seated abscess often indicates its presence by producing a peculiar rigidity of the upper part of the rectus abdominis muscle on the right side; and there is no doubt that this condition of the muscle is an important sign of disease in some one of the viscera immediately subjacent; but it by no means points especially to the hepatic abscess. Enlargement of the liver is of much more diagnostic value, particularly if the outlines of the organ be altered, if there be bulging or tumefaction in one particular direction, and if any spot can be detected which is soft and fluctuating. Where enlargement of the liver can be clearly

* See on this and other symptoms of multiple hepatic abscesses the late Dr Carrington's account of thirteen cases of multiple small abscesses of the liver ('Guy's Hosp. Rep.,' 1883).

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made out, there is generally much tenderness on pressure in the hepatic region; and round any fluctuating point there is almost always inflammatory induration of the abdominal parietes.

These last decided indications of hepatic abscess are of course absent when the back part of the liver is the seat of the disease. Even then, however, there may be an increase in the area of hepatic dulness, which, if well-marked symptoms be present, may make the diagnosis sufficiently clear.

Diagnosis.—That the detection of hepatic abscess is often very difficult may be inferred from the statements already made.

According to Maclean, there is another affection of the liver which is common in India, and apt to be mistaken for suppurative hepatitis, viz. acute inflammation of the capsule of the liver, or *acute perihepatitis*. The symptoms of this disease, however, are said to resemble those of pleurisy rather than of abscess of the liver. The pain is sharper than in the last-named disease, and it is more decidedly aggravated by pressure or by a full inspiration, or by movement of the patient's body. Acute perihepatitis is probably not very uncommon in this country, for the liver is often found after death to be fixed to the diaphragm by adhesions, which appear to correspond with those which would be left by an acute rather than by a chronic inflammation; but it is doubtful whether this affection is at present capable of recognition during life. Sometimes, however, a peritoneal *rub* may be heard over the surface of an enlarged liver.

Even where there are distinct signs of the existence of an abscess in the right hypochondrium, one must not take for granted that this is seated within the liver. It may be embedded in the *abdominal walls*. Again, in the 'Guy's Hospital Reports' for 1874, a series of cases, six in number, was published by the author, in which a collection of pus existed in the *right hypochondrium* between the upper surface of the liver and the diaphragm. This affection is most frequently caused by direct injury to the part, or by extension of inflammation from disease of one of the other abdominal viscera. In these respects it differs from abscess of the liver itself, but clinically it is scarcely capable of being distinguished by physical examination. A correct diagnosis between these two diseases is not a matter of merely scientific interest; for a subdiaphragmatic abscess can probably be more readily cured by evacuation of its contents than one embedded in the liver, since its walls can more easily come into apposition.

Again, a *suppurating hydatid* of the liver may easily be mistaken for simple abscess if the case be first seen when inflammation has already been set up within the capsule of the cyst, and if the existence of a painless tumour previously should have escaped the observation of the patient. Indeed, in such cases it is really impossible to determine the nature of the disease until the contents of the abscess are discharged, and submitted to microscopical examination. The treatment is the same as for a single large hepatic abscess, and the result is equally successful, so that the question of diagnosis is not of great importance.

Lastly, it is said that a *suppurating gall-bladder* might be mistaken for an abscess situated in the right lobe of the liver (cf. p. 355).

Prognosis and treatment.—The treatment of a case of acute hepatitis, in which suppuration is feared, must vary according as there is or is not reason to believe that pus has already been formed.

Writers of large experience in India concur in stating that in the early

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stage of the disease ipecacuanha is a potent and valuable remedy. Maclean says that this drug is nearly as efficacious in suppuration of the liver as in tropical dysentery, and that it should be given in the same manner, viz. in doses of from fifteen to twenty grains repeated at intervals of five, six, or eight hours. Antimony is also used by some surgeons in India, either in addition to the ipecacuanha or separately. It is important to relieve pain and give sleep by the hypodermic injection of morphia. Fomentations or poultices are kept applied to the hepatic region.

It appears to be established that under such treatment patients often quickly recover after having had symptoms exactly like those which are recognised as the early symptoms of hepatic abscess; but can one be certain that suppuration would have occurred in these cases if left to themselves?

When there are grounds for believing that an abscess has actually been formed in the liver, the only question is when and how the pus should be evacuated by the surgeon. No doubt hepatic abscesses sometimes subside spontaneously, for in making *post-mortem* examinations dried-up abscesses have sometimes been found with cheesy contents. This, however, occurs more rarely than was formerly supposed; for not only have dead hydatids probably often been mistaken for cured abscesses, but it is certain that the same view was generally taken of syphilitic growths in the liver before their real nature was understood; so that the spontaneous subsidence of an hepatic abscess without discharge of its contents cannot be anticipated when well-marked symptoms of suppuration have once shown themselves, and particularly if its site is indicated by definite physical signs.

Cases in which an hepatic abscess has discharged its contents spontaneously not infrequently end favourably, the cavity after a time closing and pus ceasing to be formed. It would appear that the prospect of such a termination has hitherto been greatest in those cases in which the abscess has made its way through the lung. Dr Stovell is said to have reported nine recoveries out of sixteen cases of this kind; nor is recovery infrequent when an abscess discharges into the stomach or intestine. Morehead had five cases which took this course, and three of them ended in recovery. Rupture into the peritoneum or pericardium is quickly fatal; but fortunately abscesses of the liver seldom take either of these directions.

When an abscess points towards the exterior of the body, and presently breaks and discharges its contents through the skin, the patient sometimes recovers. Maclean confirms Morehead in the statement that this is much more likely to occur when the point at which the abscess reaches the surface is near the ensiform cartilage than when it is in the right hypochondrium or in an intercostal space. This distinction apparently depends upon the collection of pus being in the former case seated in the left lobe of the liver, and therefore smaller.

Some authorities, including Budd, once held that even when an abscess is actually pointing it should be allowed to break of its own accord.

Within the last twenty years, however, the whole aspect of this question has been changed by the introduction of the aspirator of Dieulafoy, and of the various appliances of the antiseptic method.

No better illustration of the advantage of treating cases of hepatic abscess with the aspirator could be given than the following case, which is reported in the 'Medical Times and Gazette' for April, 1874, from the Madras Hospital.

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An Englishman was admitted with a tender swelling extending from the hepatic region downwards to the level of the umbilicus. He had daily accessions of fever, and other symptoms indicative of deep-seated suppuration. As there was fluctuation in the centre of the swelling, this was tapped with the aspirator, and four ounces of pus were withdrawn. The relief, however, was but partial; and when the operation was twice repeated, only a small quantity of matter came away each time. The patient's condition kept getting worse. The existence of a second abscess was therefore suspected; and as there seemed to be a slight bulging of the right lower ribs, it was determined to make an exploratory puncture in this position. The needle of the aspirator was introduced, and the syringe was soon seen to fill with pus. Forty ounces were withdrawn, and in a few weeks the patient left the hospital well.

Such a case as this contrasts strikingly with those which are given by the older writers on the diseases of India as showing the danger of opening hepatic abscesses. In that country the practice of thrusting trocars into the liver is of ancient origin, and even when no pus is thus obtained it appears seldom to be dangerous and often beneficial—possibly by relieving congestion and preventing the formation of an abscess. With antiseptic precautions and better means of diagnosis, surgeons in India and China now aspirate hepatic abscesses with boldness and success. Usually, after once tapping, an incision is made and a drainage-tube inserted. Some surgeons provide for safe adhesions being formed by previously stitching the liver to the abdominal walls. If the aspirator should not be at hand an ordinary trocar might probably be used quite as safely, provided that all the details of the antiseptic method were attended to. It is now fully established that the introduction of an instrument of this kind into the substance of the liver is not of itself attended with any risk. When an opportunity arises of examining the parts soon after the performance of such an operation the track of the trocar can scarcely ever be discovered.

But a strong objection to puncturing an abscess without an aspirator is that the pus is very likely to refuse to flow through the cannula. This occurred in a case which was under the author's care in 1875; and it was not until suction was made that it began to escape. Eleven ounces of viscid reddish pus were then drawn off; the man had not the slightest elevation of temperature afterwards, and was kept in bed only as a matter of precaution. The tumour altogether disappeared. He had been in foreign service as a mariner off the Indian and West African coasts; but he had bought his discharge four years before he came into the hospital, and since then had been working as a carman. It appeared that he had suffered severely from diarrhœa, but had not had dysentery.

Even when the trocar does not reach any pus, marked relief may be afforded by its introduction into an inflamed liver. In a case of Dr Ralfe's at the Dreadnought Hospital ('Lancet,' ii, 1876), in which hepatitis came on in the course of an attack of dysentery, only a small quantity of blood flowed into the aspirator syringe, but the patient declared that he felt instantly relieved, and did in fact experience no more pain in the right hypochondrium, where it was before severe; and his temperature, which had ranged from 99·4° to 102·2°, fell in two days to normal.*

* See the papers by Dr Harley and Messrs Willett, Marsh, Thornton, and Lawson Tait in the 'Brit. Med. Journ.' for Nov. 13th, 1886.

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CHRONIC INTERSTITIAL HEPATITIS, OR CIRRHOSIS OF THE LIVER.—In strong contrast to the acute suppuration of the liver just described, there is an insidious and very chronic process of hepatic inflammation, which is unattended by fever, and never leads to suppuration, but to the production of fibrous tissue, which shrinks and thus strangles the secreting parenchyma. The condition in question is that known as *cirrhosis*, or granular liver; or more familarly as "gin-drinker's" or "hob-nailed" liver.

The earliest representation of this disease was discovered by Dr Payne in the 'Philosophical Transactions' for 1685, where the hob-nail character is quite obvious. He also quotes an excellent clinical account of the case of a drunken German nobleman who died of ascites and jaundice, with a hard liver, epistaxis, and thrombosis of the portal vein, admirably recorded in 'Bonet's Sepulcretum,' 1679. (See Dr Payne's interesting paper in the 'Pathological Transactions' for 1889).

Anatomy.—A liver affected with cirrhosis presents very remarkable characters. Instead of being red, it is pale, and mottled with grey and yellow tints. It is exceedingly tough and hard, so that one may be unable to crush it by the pressure of the finger; its tissue may even creak when cut with a knife. Its external surface is not smooth and even, as in health, but presents numberless round elevations of all sizes, from small granules up to the size of peas, or even larger. On section similar islets are found closely packed throughout its substance. They are often of a bright yellow colour, and on this account Laennec invented the name of *cirrhosis* for the disease (κιῤῥός, yellow). At this time they were regarded as morbid products, and were called "tubercles" by Baillie.

The microscope, however, shows that they consist of liver substance which is scarcely distinguishable from that of the healthy organ. The material which is really morbid is that which lies around and between the yellow bodies. This is a greyish, somewhat translucent substance, which under the microscope is found to consist of leucocytes, nuclei, and fibrous tissue, in different proportions in different cases. Sometimes, but very seldom, it is made up entirely of cells, constituting what is termed embryonic tissue. This is in the earliest stage of the disease, when death rarely occurs. In the great majority of instances it consists of well-developed fibrous tissue, which may contain small aggregations of corpuscles, or be separated by them from the healthy liver substance. The presence of these exudation-cells is a proof that the disease was still advancing at the time of death.

It is the fibrous material which gives the cirrhotic liver its peculiar characters. The "embryonic" or granulation tissue lies between the hepatic lobules, around the terminal branches of the portal vein. As it undergoes development its separate portions coalesce. Thus they gradually form fibrous rings, surrounding the hepatic lobules or groups of lobules. But (like all young connective tissue) the new material has a strong tendency to contract. It therefore compresses the secreting cells contained in the lobules. Some of these undergo absorption, allowing the adjacent fibrous rings to coalesce. They may thus form extensive tracts of a whitish-grey colour, containing only here and there a few isolated groups of hepatic cells. Other lobules, again, become squeezed up into the rounded yellow granules or nodules above described. The reality of the compression is evident from the fact that, when a section is made through a cirrhosed liver, the yellow masses at once rise and project above the cut surface. The secreting cells

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in the yellow masses do not lie in definite columns, but are disarranged, so that it is usually impossible to determine how many original lobules each mass contains.

The distribution of the blood-vessels in a cirrhosed liver is very remarkable. If the hepatic artery be injected, one finds that the new fibroid tissue, which looks quite bloodless, is abundantly supplied with vessels from this source. But the branches of the portal vein often appear to be almost completely obliterated. Rindfleisch says that in one case he found it impossible to force injection beyond the three or four main divisions of its trunk. He therefore supposes that in this disease the bile is mainly elaborated from arterial blood.

As a rule, the hepatic ducts are but little obstructed in cirrhosis. But isolated nodules are often found to be of a dark yellow or green colour, which is evidently due to local jaundice, caused by obstruction of the corresponding ducts.

Size of the cirrhotic liver.—In advanced cases the liver is generally smaller than natural, and sometimes it is very greatly reduced in weight. The *post-mortem* records of Guy's Hospital contain several instances in which the organ has weighed as little as thirty-two or thirty-four ounces, and cases have been recorded in which it has been still smaller. But not uncommonly a cirrhosed liver is found after death to be of the natural size, or even above it. When considerable enlargement is present, the organ is almost always loaded with fat. We have had one remarkable instance in which a liver, which was of gristly hardness, weighed nine pounds; and it contained so much fat that it would actually float in water.

Dr Price, now of Reading, collected the cases of cirrhosis which occurred in Guy's Hospital from 1875 to 1883 inclusive ('Guy's Hosp. Rep.,' xlii, p. 295). They were 142 in number—108 men to 34 women. There was admitted intemperance in half the cases. In only nine was there evidence of syphilis, and in six of these there was also evidence of drink.

The weight of the liver was less than fifty ounces in 29 cases only, and in two of these the patients were below adult age (16 and 14). Excluding accidental causes of enlargement, there were 63 cases above sixty ounces. In 33 cases the weight varied between fifty and sixty ounces. Ascites was present in 58 out of 72 cases, and more often when the liver was below than when it was above the normal weight. Jaundice was present in 30 out of the 72, and more often when the liver was above than when it was below the normal weight. When death was preceded by coma, jaundice was almost invariably present. Granular kidneys were more often found in association with hypertrophic than with atrophic cirrhosis.*

It is a separate question whether a minor degree of enlargement of the liver does not constantly occur at an earlier stage of cirrhosis. That this should be the case is quite consistent with the pathology of the disease; for it begins as a subacute hepatitis with exudation of leucocytes, and atrophy of the lobules comes on only later. Bright long ago stated that he had noticed the enlargement of the liver when cirrhosis was beginning, and had traced its gradual diminution in the later stages. But the physician has rarely an opportunity of observing this; and it has not yet been proved that an appreciable enlargement of the liver is constantly

* Dr Pitt's subsequent observations continue and confirm those of Dr Price ('Path. Trans.,' 1889, p. 348).

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present at first, while it is certain that death often takes place when the organ is far larger than natural. Hence, a patient with a large cirrhotic liver has not therefore better prospects than if the liver were small.

Hypertrophic cirrhosis as a supposed separate disease from that now being discussed will be mentioned below (p. 373).

Ætiology.—The main *cause* of cirrhosis of the liver is the abuse of alcoholic liquors. The terms "hobnailed liver" and "gin-drinker's liver" have long been accepted in this country as synonymous. Dr Dickinson found that cirrhosis was present in 22 out of 149 persons whose trade it had been to make or sell or carry stimulants, and who died in St George's Hospital; while it occurred in only 8 out of 149 other persons who had been unconnected with the liquor traffic, and who therefore may be presumed to have included a smaller number of the victims of intemperance. It has been objected that the disease is sometimes seen in children; but many observations appear to show that this may really be an argument in favour, rather than against, its alcoholic origin. Dr Wilks some time since had a little girl, eight years old, under his care at Guy's Hospital suffering from what proved to be a very small hobnailed liver; she had been fed on gin, and had taken as much as half a pint daily. Niemeyer quotes from Wunderlich two very similar cases which occurred in sisters, aged eleven and twelve years respectively, who had each drunk spirits to excess. Dr Taylor's case in a boy who died at eight, was alcoholic in origin ('Path. Trans.,' 1880, p. 119). The disease is very seldom met with among those who are known to have been temperate in their habits. Gin and whisky are believed to be far more efficient in producing cirrhosis than wine or beer.

It has been supposed that other ingesta besides alcohol may set up this form of chronic inflammation in the liver; and the excessive use of spices and other condiments has been charged with causing it in India.

Cirrhosis has been attributed to the prolonged presence of gall-stones in the hepatic ducts; and persistent jaundice from obstruction of the biliary passages appears more or less constantly to lead to an overgrowth of the connective tissue in the portal canals—in fact, to a slight degree of cirrhosis. H. Mayer, Wickham Legg, Charcot, and Simmonds have produced this *biliary* cirrhosis in rabbits and cats by ligature of the common bile-duct.

Tubercular cirrhosis has also been described, but, as in other cases, the name has been perhaps too easily given to a mere increase of interstitial fibrous tissue, without the characteristic anatomical and clinical features of cirrhosis (cf. 'Path. Trans.,' xxxiii, p. 172).

Chronic heart disease, again, is believed by some pathologists to be a cause of cirrhosis; but it is exceedingly doubtful whether this ever by itself gives rise to the affection, although, by retarding the circulation through the liver, it may perhaps enable comparatively moderate quantities of alcohol to produce it.

Repeated intermittent fever is another supposed cause of cirrhosis of the liver, but probably it does not operate alone.

Syphilis produces effects on the liver which resemble cirrhosis, but are really quite different (cf. p. 374).

In concluding that excessive indulgence in alcoholic liquors is the principal cause of cirrhosis, one cannot, however, deny that well-marked cases are occasionally met with in children and others who were certainly not intemperate.*

* Such cases have been ascribed to scarlet fever (see Barlow, 'Path. Trans.,' 1877,

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The late Dr Anstie, in investigating certain other effects of chronic alcoholism, found that in the great majority of his cases there was no marked symptom of disease of the liver; nor did such symptoms show themselves, although he watched the patients for a considerable time. He therefore inferred that the abuse of stimulants could not be the cause of cirrhosis. But in drawing this conclusion Anstie cannot have allowed sufficient weight to the fact, of which he was doubtless aware, that this disease often reaches an advanced stage without manifesting itself by definite symptoms. At Guy's Hospital, the liver is accidentally found cirrhosed (in persons who have died of injury or of some other disease) once for every two cases in which cirrhosis has been the cause of death. In many of these cases the organ is indurated in an extreme degree, yet the patient certainly suffered from none of the more marked symptoms of such an affection; so that there can be little doubt that in many of Anstie's cases the liver was really more or less cirrhosed.

The frequency with which cirrhosis of the liver is thus latent is one of the most remarkable features of the disease; but there has been hardly a single instance in which the organ has been found, under such circumstances, to weigh less than fifty ounces. This might be taken as showing that the affection was not really so advanced as it appeared to be, and that if life had been prolonged the characteristic symptoms would have developed themselves; but the cases appear to be too numerous to admit of such an explanation. Moreover, on casting up the ages of persons in whose bodies cirrhosis of the liver was discovered without there having been marked symptoms during life, it results that the average age was higher by about five years than that of those persons who died of the effects of the disease. If this fact should be confirmed by a wider experience, it would prove that cirrhosis is not always (as we have been inclined to suppose) a progressive disease, but rather that after having reached a certain degree of development it may become stationary, and remain so for the rest of the patient's life.

As regards the *age* of patients who die from the effects of cirrhosis of the liver, it appears from the records at Guy's Hospital that between forty and fifty years of age there is a larger number than in any other decennial period. The proportion of males to females was 102 to 26.*

Effects.—The early symptoms of cirrhosis of the liver are exceedingly indefinite. They are chiefly those which have already been described as indicative of "bilious dyspepsia" (p. 332), together with other symptoms of chronic alcoholism, restlessness at night, tremor of the tongue and hands, irritability of the bowels, and nausea.

The digestive disorders which thus usher in the more serious effects of cirrhosis may be in part due to the impairment of the functions of the liver. But another and perhaps a more important cause of these symptoms is the

vol. xxviii, p. 355) or to rickets. One of the most conclusive cases of typical cirrhosis which was not alcoholic in origin was lately recorded by Mr D'Arcy Power, in an elderly clergyman who had suffered for many years from biliary colic and had passed gall-stones ('Path. Trans.,' 1890, p. 152). Another case in a girl of twelve is reported by Dr S. Mackenzie (*ibid.*, 1889, p. 339).

* "This disease is hardly ever met with in very young persons, but frequently takes place in persons of middle or advanced age; it is likewise more common in men than women. It is more apt to occur in those who have been accustomed to drink spirituous liquors."—Baillie ('Morbid Anatomy,' p. 228). So Heberden: "Men are more commonly affected with scirrhous (*i. e.* cirrhotic) lesions than women, because they are more given to intemperate drinking" ('Commentaries,' p. 253).

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portal *congestion* which results from the presence of the new fibrous tissue which is developed in the substance of the organ.

Pathologists have long found a difficulty in explaining how the blood returns from the chylopoietic viscera when the liver is affected with any considerable degree of cirrhosis. Some of it no doubt escapes through the anastomoses which exist at the upper and lower limits of the distribution of the rootlets of the portal vein. Thus the œsophagus is often found to be surrounded by a plexus of dilated vessels, which had carried upwards a part of the blood from the stomach; and hæmorrhoids are very frequently present, which may be taken as an indication that some of the blood from the rectum had passed away into adjacent branches of the iliac veins. But these communications seem quite insufficient to make up for the great obstruction that must exist in many cases of cirrhosis. Rindfleisch states that in one extreme instance, which he investigated, the portal blood passed directly into the inferior cava through a number of dilated anastomoses between the mesenteric and spermatic veins. Frerichs lays stress upon the existence of vessels in the newly formed adhesions between the liver and the diaphragm and abdominal wall. He also adopts the statements of Sappey with reference to certain accessory branches of the portal vein, the chief of which run along the round ligament of the liver to reach the under surface of the diaphragm. In two cases of cirrhosis Sappey found this vessel distended to the size of the little finger. Some years ago the author observed a large vein in this position when attempting to inject the portal vein in the body of a patient whose liver was hobnailed.

Whatever may be the precise course taken by the portal blood, there seems to be no doubt that much of it gets into the veins which ramify over the abdominal walls and pass upwards into the internal mammary veins. For the superficial vessels of the abdomen become greatly over-distended in cases of cirrhosis, and this can be explained in no other way, unless it could be shown that the trunk of the inferior cava itself were greatly compressed, and of this there appears to be no evidence whatever.

The congestion of the portal system of vessels which thus results directly from cirrhosis of the liver accounts for the fact that after death from this disease the *stomach* is generally found to be reddened and lined with thick mucus, and that the *spleen* is often enlarged. There has, indeed, been some difference of opinion about the state of the spleen. According to some writers it is almost invariably increased in size; whereas others have said that this is seldom the case. Frerichs found that the spleen was enlarged in half his cases; and this statement has been adopted by most subsequent writers.

It must be added that congestion and dilatation of blood-vessels is not limited in this disease to the radicles of the portal vein, but occurs likewise in distant parts of the body, where it is very much less easy of explanation. Thus a very frequent, and valuable symptom of cirrhosis of the liver is the presence on the patient's cheeks of a number of minute red lines and points, consisting of minute cutaneous vessels that have become varicose—not any form of the affection known as acne rosacea, although this too is often the result of intemperance, but what are termed "*stigmata.*" Beside the face they may often be found on the chest and abdomen.

Hæmorrhage from the other mucous membranes than the stomach and bowels and purpuric spots upon the skin are also frequent effects of cirrhosis of the liver.

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Dr Carrington observed pyrexia in eighteen out of forty-four cases of uncomplicated cirrhosis ('Guy's Hosp. Rep.,' 1883).

The most constant effects of cirrhosis are four: hæmatemesis, ascites, jaundice, and cerebral symptoms.

Hæmatemesis, of which the characters have already been described (p. 174), is one of the early symptoms. It is often very profuse, the blood is dark, sometimes clotted, and it is not accompanied by the pain of gastric ulcer or carcinoma. Melæna is a usual result: if unaltered blood is passed from the bowels it means hæmorrhage from the intestinal veins.

Ascites.—This condition, which has been fully described already, is the most constant effect of cirrhosis of the liver. It is usually abundant, and gives all the physical signs described in the preceding chapter (p. 323). It is probably a purely passive effusion.

Jaundice, as produced by cirrhosis of the liver, has already been mentioned at p. 343, where it was stated that among 130 cases in which the liver was found after death to be hobnailed, there were thirty-four in which more or less jaundice existed, and nineteen in which it was intense. This statement, however, hardly does justice to the frequency of icterus, in comparison with the other clinical symptoms of cirrhosis, for in more than forty of the 130 cases the cirrhosed state of the liver was accidentally discovered in the *post-mortem* room. This would leave less than ninety cases in which the cirrhosis produced marked effects during life; and among these the proportion of cases in which some jaundice was present would be more than one in every three. The liver is generally enlarged in these cases. In nearly half the instances which occurred in Guy's Hospital with intense jaundice the organ weighed more than seventy ounces, once as much as 131 ounces. It almost always contained much fat.

Cerebral symptoms, especially drowsiness and coma, frequently usher in the fatal termination in cases of cirrhosis which produce jaundice; but they are also common in cases which give rise, not to jaundice, but to ascites. Moreover, when, by means of diuretics and purgatives, one is able to clear the peritoneal cavity of its fluid, the successful action of such remedies seems often to avail the patient very little, for he presently becomes stupid and unconscious and dies, although his abdomen may be perfectly flat and empty. It is a question whether the removal of the fluid by tapping does not sometimes hasten the fatal issue.

In addition to the drowsiness and coma which are the chief symptoms observed in cases of this kind, Frerichs mentions noisy delirium and (in one instance) spasmodic contractions of the left side of the face. We had one patient who, although he could be partially roused, seemed to be quite unaware of being in bed and in the hospital, and, when asked where he was, always named some street in the city where he had previously lived. This man lay for two or three weeks in a semi-comatose condition.

The cause of these cerebral symptoms is obscure. It has been supposed by some writers that they result from a disintegration of the secreting cells of the liver, like that which occurs in acute yellow atrophy. But after several times carefully examining the tissue of the organ in such cases, the author always found numerous liver-cells in an apparently unaltered state. The microscopical characters were, in fact, undistinguishable from those of any other cases of cirrhosis. Frerichs, however, states that in his cases a large quantity of leucin separated from the organ, and that the bile-ducts con-

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quantity of pale bile. The condition in question has " or "cholæmia.*

sis.—Charcot believes that when cerebral symptoms aundice and without ascites, the liver will be always found nat the cirrhotic change has then begun, not around the portal canals, but within the lobules. He also believes that ular or "monolobular" hypertrophic cirrhosis is not, like the nobnailed liver, due to drink.

s no doubt a true clinical observation that cases of cirrhosis with ed jaundice run a more rapid course as a rule, although one such atient in Guy's Hospital had persistent jaundice for seven years, and died at last of hæmatemesis, and not of cholæmia. It is also true that early and extreme ascites accompanies the more atrophied and contracted condition of the cirrhotic liver. But certainly we meet with livers which are above normal weight in persons who have been intemperate, and who suffer from ascites with little or no jaundice.

Charcot described the formation of new biliary ducts within the lobules in cases of hypertrophic or "biliary" cirrhosis. It is difficult to prove that these are not the remains of hepatic tissue which have survived the disease, but other good observers confirm Charcot's view. Dr Saundby has, however, shown that these new-formed ducts may be present when there is no jaundice, and absent when jaundice is well marked.

The *prognosis* of cirrhosis is always very grave, though we have some evidence that it may be latent, or possibly may be arrested (*supra*, p. 371). But when it has caused ascites the fatal result is well-nigh inevitable. Death may occur from failure of the heart, from cholæmia, or from pulmonary disease.

Its *treatment* is first that of hepatic dyspepsia (p. 335), and afterwards that of ascites (p. 329). The patient must, at any stage except perhaps the latest, be induced if possible to give up all intoxicating liquors.

There are some other diseases of the liver, pathologically very different from true cirrhosis, but closely resembling it in their clinical aspect, and they will, therefore, be most conveniently noticed here.

Chronic inflammation of the capsule of the liver, or, as it is often termed, *perihepatitis*. In this affection the organ is remarkably deformed; it no longer has a sharp edge, but is converted into a rounded mass. Its capsule is opaque, and often forms a separable layer, which, when stripped off, leaves a smooth surface. The alteration in the form of the liver is in part caused by the contraction of this thickened capsule; but very commonly its anterior edge is also folded over on to the dorsum in a way that is difficult of explanation. In a case of this kind the margin of the liver touched a part of the convex surface that should have been four and a half inches distant in a direction from before backwards, and when the capsule was removed the organ returned to its natural shape.

The weight of a liver affected with perihepatitis is generally about the

* Another theory, which was propounded by Dr Austin Flint, jun. (of New York), is that the cerebral symptoms in these cases, as well as in those of acute yellow atrophy, are due to the accumulation of cholesterine in the blood. He supposed that in health one of the functions of the liver is to eliminate from the blood cholesterine; and in a case of cirrhosis which terminated by coma he found that there was a large increase in the amount of cholesterine contained in the blood. He, therefore, invented the name "*cholesteræmia*" for the state in which such symptoms are developed.

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same as that of the healthy organ. Its tissue is commonly soft, and is very often loaded with fat. It is seldom cirrhotic, but there is sometimes an excess of fibrous tissue in the course of the larger portal vessels.

Perihepatitis is a not infrequent cause of ascites. It does not cause jaundice nor cholæmic symptoms. Unlike cirrhosis, perihepatitis seems to be very rarely found in the bodies of persons who die of other diseases or are killed by accident; whence it appears that it never remains latent, but always advances until it causes ascites. Again, in cirrhosis the kidneys are generally healthy, but in the majority of cases of perihepatitis they are diseased.

With regard to other than renal causes of perihepatitis very little is known. It is often a locally exaggerated form of general chronic peritonitis.

Simple hypertrophy of the liver, distinct from the vascular turgescence caused by heart disease, is not very uncommon. It is found in beer-drinkers, and sometimes in cases of diabetes. Wilks and Moxon record a liver of the former kind which weighed 80 oz., and was healthy in texture. They also describe compensatory hypertrophy when part of the organ has become atrophied from pressure or from syphilitic growths, and note a case in which the whole right lobe was atrophied, and the left weighed 56 oz., thus making up the loss. Local circumscribed outgrowths of hepatic tissue are pathological curiosities only, and are probably referable to a form of innocent glandular tumour, adenoma.

Simple chronic atrophy is a rare affection of the liver. In a case recorded by Dr Cayley ('Path. Trans.,' 1868) the liver weighed only twenty-two ounces; the left lobe had almost disappeared, being only an inch wide. In another case, one of Murchison's (*ibid.*, 1867), the organ weighed twenty-five ounces; its margin was thin and flat, forming a kind of rim, which consisted only of connective tissue and vessels enclosed between the two layers of the capsule. This rim measured in one place an inch across. In both cases the substance of the liver was of a dark colour, and quite free from induration.

Minor degrees of atrophy of the liver are by no means uncommon, particularly in old people and in those who die of wasting diseases, such as cancer of the stomach or œsophagus. It does not, however, accompany phthisis, for when the rest of the body is emaciated to the utmost from this cause the liver is usually enlarged by deposition of fat. Atrophy of the liver is often found as the result of chronic perihepatitis.

This affection seldom gives rise to ascites or any other symptoms.

Syphilitic affections of the liver.—These present very different characters in different cases. Sometimes gummata are scattered through the hepatic tissue, which is in other respects healthy. The condition is generally unattended with any symptoms; but it may happen that one of the gummata is so placed as to obstruct the circulation through the organ, and thus cause ascites. A case of this kind occurred at Guy's Hospital, in which one of the hepatic veins, close to the inferior vena cava, was so narrowed that it would only just admit a probe. More often the gummata, instead of being embedded in the hepatic tissue, lie in the midst of broad fibrous bands, which traverse the liver from one surface to the other, forming deep notches or depressions, or cutting off large masses from their continuity with the rest.

The deformity thus produced is very great. Even when the syphilitic

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interstitial hepatitis has been more uniform in distribution, the fibrous bands are coarser and the islets of healthy tissue larger and less regular than those of true cirrhosis. The process is one of cicatrisation and puckering, and firm or caseous gummata are found as the foci of the star-like depressions. Perihepatitis often accompanies the interstitial fibrosis.

A very different effect of the syphilitic poison on the liver has been found in the case of infants. It is a diffuse interstitial hepatitis leading to a uniform pale and very firm texture. It was first described by Gubler in France in 1852 (cf. 'Path. Trans.,' xvii, p. 167).

Again, beside containing gummata and fibrous bands, the syphilitic liver is often lardaceous, and then it may reach a very great size, weighing from six to seven pounds, while the capsule is generally thickened and adherent to adjacent parts. In such cases ascites is especially apt to occur. Dr Grainger Stewart recorded the following case, which appears to have been of this kind: a patient had ascites, for which she was tapped twenty-one times, the enormous quantity of 606 pints being removed in the course of these operations; at first the paracentesis had to be repeated every fortnight, but the intervals gradually became longer, until at length she regained tolerable health. In twenty years (1862–82) there occurred in Guy's Hospital about six cases of fatal ascites from gummatous and lardaceous disease. In several of them the liver could be felt during life to be enlarged and adherent to the parietes, with an uneven and nodular surface, and these characters more than once enabled a correct diagnosis to be made.

ACUTE YELLOW ATROPHY OF THE LIVER.*—This is the name given to one of the most remarkable diseases known, rare in its occurrence, obscure in its pathology, fatal in its results, and unique in its anatomy.

History.—For many years it had been known that cases of apparently idiopathic jaundice occasionally do not run a favourable course, but become complicated by hæmorrhage and cerebral symptoms, and end in death by coma; they were called *icterus gravis*, or malignant jaundice. But the first clear and complete cases were published by Bright in the first volume of the 'Guy's Hospital Reports' (1836), under the title "Intense Jaundice without Mechanical Obstruction, apparently depending upon Inflammatory Action in the Liver." The following is his account of these two typical cases of acute yellow atrophy (Nos. 5 and 6, pp. 624—630).

(1) A woman aged twenty-eight, of dissolute habits, while taking mercury, was attacked by abdominal pain, and jaundice quickly followed. Dr Bright saw her on the third day; the urine was then bile-stained and the fæces clay-coloured; on the twenty-first there was drowsiness, followed by delirium. She died comatose on the twenty-third day of the jaundice.

At the autopsy there was intense staining of every tissue with bile; the organs of the head, chest, and abdomen were healthy except the liver, which weighed only thirty-seven ounces, and was "soft or flaccid to the touch," with no trace of peritoneal inflammation.

(2) A German girl, aged eighteen, was admitted into Miriam Ward January 11th, 1832, labouring under icterus. "The skin was of a brilliant yellow, and the cheeks, which were flushed, were the colour of a very ripe apricot." She had probably been ill for nearly four weeks, and the jaundice

* *Synonyms.*—Icterus gravis—Icterus typhoides (Lebert)—Irish yellow fever—Ictère malin—Ictère hémorrhagique essentiel—Die acute Leberschmelzung (Rokitansky)—Atrophia hepatis flava sive acuta (Frerichs)—Hepatitis parenchymatosa diffusa.

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had gradually deepened to its present tint. She had lately sat by the fire in a kind of doze. That evening she vomited, and "lay in a perfectly torpid state the whole night, apparently suffering no pain; but towards the morning became delirious, so that it was with difficulty she could be restrained in her bed." Dr Bright ordered "two grains of calomel every two hours, and the ammonia julep (Mist. Ammoniæ Co. of the present Guy's Pharmacopœia) every four hours, besides wine if she became more depressed." Purging enemata and a blister over the liver were also ordered; the head was shaved and mustard was applied to the feet. She continued, however, completely comatose all the following night, and died at ten in the evening of January 13th.

At the autopsy, excepting a universal and deeply jaundiced tint, the brain, heart and lungs, stomach and intestines, pancreas, and kidneys were found normal. The spleen was soft. "The liver was unusually small, and for the most part of a brightish yellow colour, with portions marked with purple or deep brown."

Dr Bright remarks, "In this case, as in the last, no obstruction could be discovered in the ducts which could have prevented the flow of bile from the liver." "The immediate cause of death in this case, as in the last, was the poisoning influence of the bile on the system." "The bile must have been rapidly absorbed into the system almost at the moment of its formation, and its profuse mixture with the blood seems to have acted as a poison, aud hence the immediate cause of death. I am inclined to consider this as the result of a decidedly inflammatory state of the organ." Again, he remarks that, in these severe cases of jaundice with nervous symptoms, "the tendency to hæmorrhage comes on very early and is excessive."

A few years later, in 1843, Graves, of Dublin, published in his famous 'Clinical Lectures' (2nd ed., 1848, vol. ii, p. 255) three cases of jaundice in sisters. One, a girl of seventeen, was attacked in July, 1840, with vomiting and icterus, followed by hæmatemesis, violent delirium with convulsions, coma, and death on the seventh day. No autopsy was permitted. The second, aged eleven, died in March, 1841, with similar symptoms on the fifth day. After death the liver was of "natural size," dull yellow with dark spots, and bile in the gall-bladder; the brain (examined first) was much more vascular than usual; the thorax was not examined. The third sister, aged eight, was taken ill with jaundice and vomiting the following June. She was actively treated by bleeding, leeches, and calomel, with James's (the compound antimonial) powder; the alarming symptoms disappeared after three days, and she recovered from her jaundice in about three weeks more.

Graves's cases were quoted as examples of acute atrophy by Budd and by Trousseau, and also by Frerichs in his well-known treatise. But there is no evidence that the last was anything more than ordinary *icterus*; the first is incomplete, and in the second there was no atrophy of the liver discovered.*

Rarity.—Acute yellow atrophy of the liver is not a common disease. Murchison says that although delirium and a brown tongue constituted a certain passport into the London Fever Hospital, only one case occurred

* Isolated cases of rapid and fatal jaundice, which may with more or less probability be referred to yellow atrophy, have been quoted by Frerichs and Trousseau from Rubeus (1660), Boerhaave, and Morgagni.

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among 3000 patients admitted in a period of six years. In Guy's Hospital there are notes of the inspection of only eleven cases in twenty-seven years, 1864–90.

Symptoms: Icterus.—The jaundice of acute yellow atrophy is very deep but not otherwise remarkable. The conjunctiva and urine are bile-stained, and at least in most cases the stools are clay-coloured. Icterus may precede the graver and characteristic symptoms not only by a day or two, but by weeks.

Nervous system.—In addition to the jaundice, there are marked cerebral symptoms. Headache and intolerance of light are often first complained of. Before long the consciousness is more or less impaired. The patient now becomes very restless, screaming and tossing about, and sometimes fiercely maniacal. Violent convulsions sometimes follow. Ultimately a state of complete coma is developed; the pupils become widely dilated and insensible to light; the urine and fæces are passed involuntarily; the breathing is stertorous; and the scene ends in the death of the patient, almost always within five days from the commencement of the characteristic symptoms. It is, indeed, said that acute yellow atrophy has sometimes destroyed life in less than twenty-four hours. According to Niemeyer, the majority of cases end fatally on the second day, but this is certainly not the case.

Physical signs.—It is possible by means of *percussion* to trace from day to day the gradual diminution of the liver. From a normal measurement of four inches vertically in the right mammary line—reaching from the fifth intercostal space downwards to the costal margin—the hepatic dulness may be watched as it undergoes reduction to three inches, two inches, and one inch, until at last it disappears entirely. One must not, however, suppose that the diminution of dulness over the liver is necessarily due solely to the wasting of the organ. As we have seen, the liver in acute yellow atrophy is remarkably flaccid and falls backwards away from the ribs, so that the intestines, if distended, ride over it. Indeed, this source of fallacy is not confined to cases in which the liver is really affected with acute atrophy. In all forms of jaundice the bowels are apt to become inflated with gas, and the consequent enlargement of the abdomen may cause the right hypochondrium to become gradually more and more tympanitic, and the area of hepatic dulness to diminish from day to day. This has led to the error of supposing the liver to be in a state of acute yellow atrophy when such was not the case.

State of the urine.—This does not generally contain a large quantity of bile-pigment; hence it does not look black when in bulk, nor in a thin layer has it so intense a saffron-yellow colour as is sometimes seen. It is said that Gmelin's test may give an imperfect reaction.

Besides the icteric discoloration, another and characteristic change in the urine is constant. The urea and lithic acid, and also the chlorides, sulphates, and earthy phosphates are greatly diminished in quantity or nearly absent; and in their place are found two new substances—*leucine* (amido-caproic acid) and *tyrosine* (amido-sulpho-peruvic acid), both products of albuminous decomposition. There is generally no difficulty in detecting them. They sometimes form a distinct deposit when the urine is left to stand for a time, or, if this is not the case, they may be made evident by evaporating a few drops with a drop of acetic acid on a glass slide. Tyrosine is easily recognised by its taking the greenish-yellow colour of the

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urine. It occurs in bundles or globular masses of needle-shaped crystals. Leucine presents the appearance of rounded, flat discs, generally marked with concentric rings.

The urine is, as a rule, albuminous; but it is uncertain whether this should be regarded as a symptom of a constantly concomitant nephritis (as Dr Grainger Stewart believes) or as "febrile," or as the result of the presence of leucine and tyrosine.

Hæmorrhage.—In some other forms of jaundice there is a tendency for the blood to escape from the vessels, but this is especially marked in acute yellow atrophy. Very frequently the patient vomits a dark fluid resembling coffee grounds, or otherwise altered blood. Petechiæ are often developed in the skin, and almost invariably, towards the end of the case, the evacuations have a dark brown or a tarry black colour, which is the result of hæmorrhage.

The stools.—With regard to the colour of the fæces in acute yellow atrophy different writers have made different statements. Murchison says "the jaundice appears to be due to a poisoned condition of the blood, and consequently bile is still found in the stools." The remark has often been made that it is a good sign for the motions to be clay-coloured in jaundice, there being then less danger of the supervention of cerebral symptoms. But it is certain that towards the end of a case of acute atrophy no bile enters the intestines, for after death the ducts and gall-bladder are found to contain an almost colourless mucus. Moreover, Frerichs says that in this form of jaundice the stools are dry and clay-coloured; and more than one case is recorded at Guy's Hospital in which such was the case. The question is not so easy of determination as might at first sight appear, on account of the great frequency of intestinal hæmorrhage in this disease. When the stools have been supposed to contain bile in acute atrophy of the liver, their dark appearance has generally been due to altered blood. We must, however, remember that the disease does not affect the whole substance of the organ uniformly, but attacks some parts earlier than others. Thus at its commencement bile very possibly continues to enter the intestines from those portions of the liver which have not yet become diseased.

Later symptoms.—The patient soon falls into a "typhoid state." His tongue is almost always dry and brown, and his lips and teeth are encrusted with sordes. According to Niemeyer and other writers, the *temperature* of the body is raised considerably above the normal. But this is only an occasional event; for in several of the cases that have occurred at Guy's Hospital the temperature was normal. In one instance it was below the average four days before death, but it began to rise two days later, and while the patient was dying it was found to be 101·6°. Exceptionally, higher temperatures have been recorded. Sir Dyce Duckworth noted the absence of pyrexia in three cases that were observed in St Bartholomew's Hospital. Frerichs says that in his cases the skin was usually cool, dry, and inactive, and he quotes Bright and Addison as having made particular mention of the same circumstance. The pulse is almost always accelerated, but in one of Duckworth's cases it was on two days about 50. Towards the end it becomes very small and intermittent.

When acute yellow atrophy occurs in a pregnant woman *abortion* or miscarriage almost always precedes the patient's death.

The majority of cases prove fatal in from one to five weeks, sometimes within two or three days. Their duration is often apparently lengthened

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by ordinary icterus preceding the special symptoms of the disease. It is possible that more chronic cases are examples of "red atrophy" (see the cases recorded by Dr Moxon, 'Path. Trans.,' 1872).

The *event* of acute yellow atrophy is not absolutely constant. A few instances of recovery have been recorded. Not long ago Dr Wilks had at Guy's Hospital a fatal case, in which there was a distinct history of a previous attack that had been recovered from. The patient had become delirious, and had such severe hæmatemesis that it was thought he would die in a few hours. However, he rallied and lived two months longer, at the end of which he again became delirious. Leucine and tyrosine were found in the urine, and he died a fortnight later. The liver was found by Dr Moxon to weigh forty-seven ounces. The left lobe and the adjacent part of the right lobe were small and dark looking. The lobules in them were distinct, but scarcely any hepatic cells were to be seen. The rest of the right lobe formed a soft, yellow, rounded, projecting mass. The marked contrast appeared to justify the supposition that the left lobe had become atrophied at the time when the cerebral symptoms first arose. Another case of recovery, followed by a second fatal attack, was recorded by the late Dr Frank Smith, of Sheffield ('Path. Trans.,' 1877, p. 236).*

Anatomy.—The first thing that strikes one, in making an inspection in a case of this kind, is the diminution in the size and weight of the liver. The organ forms a thin flaccid mass, which lies at the back of the abdomen, hidden by the ribs and by the distended intestines. Instead of weighing from fifty to sixty ounces—the usual weight of an adult liver—it weighs perhaps thirty-two ounces, thirty ounces, or even as little as twenty-three ounces.

When cut into, the tissue looks as though it were softened; but the finger is found not to penetrate it more readily than the healthy organ, for although it is so flabby, it is not friable. Its colour is greatly altered. Most of it has a bright orange-yellow tint; but some parts are dark red or purple. Sometimes roundish masses having this red or purple hue are scattered through the substance of the organ; sometimes one part, generally the left lobe, is almost entirely red, while the rest of the liver is mainly of a gamboge-yellow hue. To the naked eye it appears as though the red parts were less altered than the yellow, but the microscope shows that this is not the case. In both parts the hepatic cells have undergone destruction, and are replaced by a mass of granules and oil-globules; but in the red parts the destruction is complete ("red atrophy"), whereas in the yellow parts some of the secreting cells still remain visible, and towards the centres of the lobules may even retain their columnar arrangement ("yellow atrophy"). Among the remnants of the hepatic tissue are often to be seen crystals of leucine and tyrosine.

In some cases it has been thought that a pellucid nucleated material could be detected, supporting the detritus of the cells, and this was believed to be the case in two cases examined by Dr Fagge.

Waldeyer and Klebs have described, in the reddened parts, cells resembling those of the epithelial linings of the biliary ducts, arranged in regularly branching lines and tubes which seemed to have cæcal terminations (cf. p. 373).

* See a list of no less than twenty-eight cases of supposed recovery from acute yellow atrophy in Dr Wickham Legg's treatise 'On the Bile and Jaundice' (p. 676).

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The bile-ducts are found empty; their mucous membrane is unstained by bile-pigment. The gall-bladder is either empty or contains a few drachms of grey mucus or of a pale yellow or greenish fluid.

The kidneys can very generally be shown by the microscope to have undergone morbid changes. The epithelium of the tubules is granular and may be very fatty.

Ætiology.—The origin of the disease is exceedingly obscure. Among its exciting causes, mental emotions seem to take a foremost place. It has already been stated that anxiety or grief is a frequent cause of simple jaundice (p. 341); and cases arising in this way may prove fatal by the supervention of cerebral symptoms. Again, more than one instance has been recorded in which acute atrophy has followed directly upon a drunken debauch; and in several cases it has set in during the secondary stage of constitutional syphilis. It might be argued that one cannot in either of these conditions exclude the possibility that the jaundice was really due to depressing emotion or remorse; persons affected with syphilis often undergo great mental torture, which they carefully conceal. Pregnancy is another predisposing cause of the disease; out of 22 female patients referred to by Frerichs one half were pregnant, and of 88 collected by Thierfelder (quoted by Eichhorst) 30 were pregnant. Here, again, mental influences may possibly come into operation.

Sex and age.—Apart from pregnancy, acute yellow atrophy of the liver is far more common in women than in men, 88 out of Thierfelder's 143 collected cases.

It occurs principally at an early period of life. Five times out of six the patient is under thirty years of age. It is, however, rare in childhood, although Dr Goodhart had once under his care a typical case in a boy only two and a half years old ('Path. Trans.,' 1882).

Dr Tuckwell reported in 1874 two cases in boys, one seven years old, the other between four and five; and a third case in a girl, aged from four and a half, a patient of Dr West's in 1859 ('St Barth. Hosp. Rep.,' vol. x).

Pathology.—Some of the earlier writers on acute yellow atrophy of the liver, having found after death that the larger bile-ducts were free from obstruction, conceived the idea that the minute channels which issue from the secreting lobules of the organ might have undergone compression in consequence of swelling of the cells forming the periphery of the lobules themselves. And they supposed that the jaundice was really due to reabsorption of bile secreted by the cells forming the centres of the lobules. Rokitansky even imagined that the breaking down of the hepatic cells was due to a solvent action excited by the retained bile. These views, however, can be controverted by evidence of great weight, and at the present day it seems probable that acute atrophy of the liver is a *parenchymatous inflammation*, although we must admit that no precisely analogous disease can be found among those to which other organs are liable. This was the view originally taken by Bright.

Some uncertainty still prevails with regard to the origin of the leucine and tyrosine, which, as we have seen, are excreted in the urine in this disease. Most authorities suppose that in acute atrophy of the liver the chemical changes which should be undergone by albuminous substances in the blood are incomplete, so that, instead of urea and uric acid, the new bodies in question are formed; and this view accords well with the fact that urea and uric acid are more or less completely wanting. But other writers,

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basing their opinion on the fact that the normal liver during decomposition contains leucine and tyrosine, think that these substances are the direct products of the disintegration of the hepatic tissue.

Again, there is a doubt whether the granular and fatty changes in the epithelium of the renal tubules are the result of the disease of the liver, or whether both these conditions do not rather depend upon some common cause. The former view appears to the author to be the correct one, for the morbid changes in the kidneys are often comparatively slight.

What is the cause of the cerebral symptoms which form so striking a feature in acute atrophy of the liver? Frerichs thought that they depended upon the presence of leucine and tyrosine in the blood, but experimenters have hitherto failed to verify this supposition. Rokitansky started the theory that these symptoms were really uræmic and dependent on the renal changes, and Virchow has given it his support. But the character of the cerebral symptoms in acute atrophy of the liver is not the same as in uræmia, nor is nephritis constant.

It is possible that the disease may prove to be "specific;" Waldeyer and other pathologists have found microphytes in the liver, but they appear not to be constant. They were absent in Dr Cavafy's case ('Path. Trans.,' 1883) and in those examined by Klebs and by Senator.

Diagnosis.—This is not difficult in most cases, if attention be paid to the characteristic symptoms, particularly the rapidly diminishing liver-dulness, the hæmorrhages, the delirium, the stupor, and the presence of albumen, leucine, and tyrosine, as well as of bilirubin in the urine.

In a patient whom the writer saw with Mr Toulmin in 1889, the early part of the case was like enteric fever, with diarrhœa and very slight jaundice, and it was only after three weeks that the appearance of leucine and tyrosine with decided icterus cleared up the diagnosis; the liver-dulness then rapidly diminished, cerebral symptoms supervened and ended in death by coma, after five weeks' illness. Unfortunately no autopsy could be obtained.

The affection which most closely resembles acute yellow atrophy is one produced by *poisoning with phosphorus.* Within the last few years it has been shown that the toxic effects of this substance are by no means limited to the vomiting and purging which immediately follow its ingestion. In a few hours these generally pass off, and often the patient appears to be perfectly well for three or four days; but at the end of this time jaundice sets in, followed by delirium and coma, and these symptoms ere long prove fatal. According to some observers the liver is then found to be altered exactly in the same way as in cases of acute yellow atrophy; and they therefore speak of phosphorus-poisoning as one of the causes of this disease. But probably the appearances are always distinguishable from those seen in acute yellow atrophy. In some cases of poisoning by phosphorus, at any rate, it is certain that the liver presents characters which are very different. It is larger than natural, of normal shape, and of a pale buff colour, very fatty, but mottled with numerous ecchymotic spots. Under the microscope the cells appear to have undergone destruction by fatty degeneration; but there is often a similar difficulty in detecting the hepatic cells in other cases of fatty liver —and even in the physiological steatosis of stall-fed cattle and Strassburg geese—when there is every reason to believe that they are intact, although their outlines and nuclei are obscured by the oil-globules.

Clinically, poisoning by phosphorus differs from acute yellow atrophy

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particularly in the absence of leucine and tyrosine from the urine. The liability to hæmorrhage, however, forms a common and prominent feature of both diseases. After poisoning by phosphorus the uriniferous tubules are loaded with highly refracting granules, like those in the hepatic cells, and the fibres, both of the voluntary muscles and of the heart, are found to have undergone a granular fatty degeneration. Thus the morbid state produced by phosphorus appears to be an acute steatosis of the liver, kidneys, and muscles. In England such cases are rarely seen, but in Germany they are far from uncommon. Persons who wish to commit suicide there seem to use the heads of a bundle of lucifer-matches, just as among us they employ white arsenic for the same purpose.

In a case of poisoning which occurred at Guy's Hospital the temperature of the body was very low, at least for some hours before death, the thermometer standing in the axilla at 96·8°, and afterwards at 91·5°.

In another case, under the writer's care, a woman destroyed herself and her child, five years old, with phosphorus. The latter perished quickly as if by exhaustion, with little vomiting and no pain; but the mother lived for several days, and at first appeared to be recovering. There was no marked jaundice, no hæmorrhage, and no delirium, but she sank rapidly into coma. After death the liver was found in a state of fatty degeneration, and the same process had affected the kidneys and the heart, but there were no appearances like those of acute yellow atrophy. In the child the liver was very fatty, but the heart and kidneys were unaffected.

Statistics.—The results of Thierfelder's collection of 143 cases of this rare disease have been mentioned above (p. 380). The following is a summary of all the cases which have occurred at Guy's Hospital during the twenty-five years of the present writer's connection with it. They extend from 1866 to 1890 inclusive. Some cases of hepatic atrophy were doubtful, *e. g.* a liver weighing 31 ounces but without icterus; and others were referable to "simple atrophy," diffuse forms of cirrhosis, or atrophy secondary to the congestion of chronic cardiac disease.

Excluding these, there were during the twenty-five years, eleven cases, five in men and six in women. The ages were as follows: one boy only two and a half years old (Dr Goodhart's patient referred to above), a youth of eighteen, six patients between twenty-one and twenty-three, one of thirty-four, and one woman of forty-four, who appeared however to be much older.

The duration of the cases was from ten days to five weeks, but in three cases there had been a period of several weeks in which jaundice was present without other symptoms. There were present (beside jaundice) hæmorrhage, usually hæmatemesis, and delirium ending in coma. Albuminuria was seldom absent. The temperature was sometimes raised, sometimes subnormal, and in one case it rose to 104° F.

The weight of the liver after death varied in most cases (excluding the child's) between 26 and 33 ounces, but in three it was 40, 47, and 46 ounces, the last case being that of a woman. In these three cases the atrophy was partial but characteristic. In most cases the kidneys were in a state of tubular nephritis: in one only were they described as perfectly healthy.

Treatment.—No remedies for icterus gravis are known. Our recent cases of this most singular disease have not been actively treated, for it was taken for granted that they must terminate fatally. However, there are, as we have seen, a few exceptions to this rule; and since the disease

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seems not to attack the liver as a whole, but generally to spread through the organ from the left lobe, it is possible that its course may be influenced by treatment. Dr Budd recommended a mixture containing a drachm of the sulphate and fifteen grains of the carbonate of magnesia with half a drachm of the spiritus ammoniæ aromaticus three times daily. This advice seems to have been founded upon the brilliant results which certain Irish physicians formerly obtained from purging in similar cases of icterus gravis. The most striking examples were the cases recorded in the year 1834 by Dr Griffin, of Limerick. Four children of the same parents were attacked within a few weeks by jaundice, with cerebral symptoms. Two of them died, but two recovered after having been in a state of almost complete coma. The treatment—bleeding, blistering, and active purging—was the same which failed in the hands of Dr Bright.

Large doses of quinine, sulphocarbolate of soda, and perchloride of mercury have proved as useless as the older treatment. In our present ignorance of the origin and nature of acute atrophy of the liver, we cannot expect to do good, and may easily do harm by interference.

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NEW GROWTHS, DEGENERATIONS, AND PARASITES

ATTENDED WITH ENLARGEMENT OF THE LIVER

Væ meum
Fervens difficili bile tumet jecur :
Tum nec mens mihi nec color
Certa sede manet, humor et in genas
Furtim labitur, arguens
Quam lentis penitus macerer ignibus.—HORACE.

Malignant disease—Carcinoma hepatis—its anatomy and histology—its rarity as a primary disease and its most frequent antecedents—its symptoms, diagnosis and course—Cancer of the gall-bladder and bile-ducts.
Hypertrophy of the liver—Lymphatic overgrowth : leuchæmia hepatica—The fatty liver—general obesity and its treatment—Malarial enlargement—Lardaceous disease—Hepatic tubercle.
Hydatids of the liver—and of other organs—Other parasites of the liver.

THE remaining diseases of the liver are somewhat heterogeneous, and most of them have greater pathological than therapeutical interest. They agree anatomically in all causing enlargement of the liver, and clinically may thus be separated from atrophic cirrhosis and acute yellow atrophy. With the exception of the first, malignant disease, they are all free from jaundice and from ascites. Indeed, it is surprising how little extensive structural changes in this organ affect its functions, secretory or metabolic.

CARCINOMA.—Malignant disease of the liver has long been known to pathologists, and is of frequent occurrence. But it is rarely primary, and in most cases follows either cancer of the stomach, rectum, breast, or uterus, or else cancer of the gall-bladder or ducts.

Anatomy.—The usual form of cancer of the liver is that of numerous nodules scattered irregularly through its substance ; but some are almost sure to reach the surface. They may now and then be seen as minute white or yellow points not bigger than hepatic tubercles, but some are almost certain to be larger than this, and they grow from the size of peas to masses as large as a fœtal head.

These lumps are usually soft (*encephaloid* or *medullary*), and yield an abundant white juice on scraping. They may undergo caseous degeneration in the centre, and are often so vascular that hæmorrhage takes place into their substance, so as to justify the term formerly applied to them, as to the vascular excrescences of mammary cancer—*fungus hæmatodes*,—and occasionally they become almost cavernous in structure. As the result of central softening, the great tubera which are seen on the surface of the liver are marked by a depression in the centre which gives them a charac-

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teristic umbilical aspect, like the leaves of navelwort or the seeds of nux vomica.*

In exceptional cases the cancerous nodules are much slower in growth, harder in texture, and more uniform in size. This form of the disease has been called scirrhus (*carcinoma fibrosum*); and it sometimes distorts the liver so uniformly and renders it so tough, that to sight and touch it exactly resembles the cirrhotic livers with the larger and less regular kind of "hob-nails." So close was the resemblance in a case under the writer's observation (1876) that it was a surprise when the microscope showed the true nature of the transformation (Guy's Path. Museum, prep. 1922[30]).

Whatever the form, the histological structure of the cancerous tumours of the liver is almost invariably the same, that of typical glandiform carcinoma. Cylindroma is occasionally seen, epithelial (corneous) cancer still less frequently, and most rarely of all, colloid carcinoma. Melanotic cancer is also rare, and is always secondary (*ibid.*, prep. 1937, *et seq.*).

In most of the cases formerly described as primary cancer of the liver the growth probably began in the gall-bladder, gall-ducts, or in the portal fissure.

When the disease is truly primary it sometimes assumes a remarkably infiltrating character. In a case of this kind under the writer's care in 1878 and 1879 the liver was enlarged to the enormous size of 200 ounces. There was no other trace of cancer in the body; and other remarkable features of the case were its long duration, its painlessness, and the youth of the patient, who was a boy of only twelve† ('Path. Trans.,' vol. xxxi, p. 125).

A liver affected with this diffused form of cancer has a peculiar appearance on section. The lobular markings are everywhere plainly visible, but they are coarser than natural. The substance of the liver is grey or white; all parts of the cut surface yield a milky juice, and the microscope shows that the cells in the lobules have the character of cancer-cells, although they are arranged in radiating columns, occupying the meshes of the blood-vessels, like the secreting cells of the healthy organ. Probably these elements are directly derived from the pre-existing epithelial cells, and not from connective-tissue corpuscles.

The presence of numerous and large nodules greatly increases the size of the liver, so that the largest livers observed are those affected with cancer. Two have been observed at Guy's Hospital which weighed each 18 lbs. In a case recorded in vol. xxiii of the 'Pathological Transactions' a liver, which was full of cancerous tubera, weighed 19½ lbs.; another case is there alluded to in which the weight was 24 lbs., and Dr Arthur Jones, of Northampton, met with a case in which the weight of 28 lbs. was reached.

Signs and symptoms.—Many cancerous nodules may be scattered through

* "Large white tubercles, in greater number near the surface of the liver than near the middle. . . . They consist of a firm, opaque white substance, and are generally somewhat depressed or hollow upon their outer surface."—MATTHEW BAILLIE, 1797.

† The distinction between primary and secondary carcinoma is often difficult, even in the *post-mortem* room. Some observers think that even the largest solitary masses, and the diffused forms of cancer of the liver, are very rarely primary. But my own impression is that such a view may be stated too absolutely. It is true that a *post-mortem* examination sometimes reveals the presence of a primary carcinoma in the intestine or the vertebræ of the os innominatum, which had been before unsuspected. But it is also true that in other cases no primary disease outside the liver can be discovered on the most careful examination. One source of fallacy may be mentioned, which is that cancer of the gall-bladder growing into the hepatic tissue has sometimes been mistaken for a primary cancer of the liver. The cavity of the gall-bladder may in such a case be so small that, lying in the centre of the tumour, it is easily overlooked.—C. H. F.

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the liver without enlarging it enough for its edge to be felt below the ribs. But when the tubera are grown larger they may reach such a size that they can be felt through the abdominal walls, or may be seen to rise and fall each time the patient breathes. They are generally firm to the touch, sometimes of apparently stony hardness; but occasionally they feel very soft, so that one might imagine fluctuation in them. Indeed, their centres may really become hollowed into cavities containing fluid, as in an instance which occurred at Guy's Hospital: a cancerous tuber formed a cyst that would have held a cocoa-nut; it was so near the surface of the organ that it might have yielded fluctuation; and it was filled with a clear straw-coloured liquid. Sometimes cancerous nodules can be felt to have a central umbilicus—a sign of importance, for it is not observed in any other affection.

Instead of several distinct nodules or tubera, cancer of the liver may be felt as a single large rounded mass, projecting from the right or left lobe downwards into the abdomen. Sometimes, again, a cancerous liver is enlarged without its shape being altered, even when it has reached an enormous size.

As above stated, cancerous tumours of the liver are often exceedingly vascular, and their vessels have very thin walls, so that hæmorrhage into the substance of the nodules is far from uncommon. According to Frerichs the extravasation of blood may be so copious as to give rise to clinical symptoms—a perceptible increase in the size of the tumour and decided anæmia. Sometimes, when a vascular cancerous growth is situated just beneath the surface of the liver, the serous membrane covering it gives way, and blood escapes from its substance into the peritoneal cavity. A remarkable instance of this once occurred in our pathological theatre, when a large clot covered the surface of the organ. One can seldom determine, in cases of this kind, what quantity of blood has exuded, for it is probably mixed with ascitic effusion; but it would seem that the fatal issue is sometimes more or less directly due to the rupture of the tumour, for patients in whom this has occurred have become collapsed some hours, or, in one case, three days before death.

It was shown by Frerichs that cancerous growths in the liver derive their vascular supply mainly from the hepatic artery, and that they receive very little blood from the portal vein. In proportion as they increase in size the trunk of the hepatic artery becomes enlarged, while the area of distribution of the portal vein is diminished. The growth, however, not infrequently penetrates into the interior of one of the branches of the last-named vessel, and may then extend along its channel so as to obstruct the flow of blood. This is one cause of *ascites* in cases of hepatic cancer; but it is sometimes the result of chronic peritonitis which started from the serous covering of the organ, just as pleuritic effusion follows cancer of the mediastinum or lung; or there may be multiple secondary nodules of the kind described above (p. 321).

Another symptom of cancer of the liver is *pain*, which is often severe, and generally accompanied by marked tenderness on pressure in the right hypochondrium. *Jaundice* is sometimes absent, or shows itself only when the case is about to terminate. In this respect there is a difference between cancer of the liver itself and cancer of the structures in the portal fissure, which is a frequent cause of jaundice. There is sometimes slight pyrexia, for which no other cause can be found after death—100° or 101° Fahr., and in one case (that of the boy mentioned on the last page) it reached 104·6°.

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Diagnosis.—Cancer of the liver, when at all advanced, is usually easy of detection. The liver is enlarged, painful, and irregular on its surface. There is sooner or later jaundice, and frequently ascites. The patient is at or beyond middle age, and usually shows signs of grave disease in loss of flesh, anæmia, and a sallow complexion. When such symptoms are present we should look for symptoms of gastric cancer, examine the breasts and uterus in a woman, the rectum and the testes in a man, with a view to discover the primary seat of the disease.

Sometimes, however, there is considerable difficulty in diagnosis, and this turns almost always upon the question between cancer and cirrhosis. We have seen (*supra*, p. 368) that in cases of cirrhosis the liver is often enlarged, and can be felt below the ribs; its surface may be uneven, it is often tender to the touch; jaundice is very frequently present (p. 343); and ascites may be moderate and late in making its appearance. Moreover intemperance in liquor does not preserve a man from cancer, and cirrhosis may develop itself in or even after middle age. The question, therefore, is sometimes extremely difficult, perhaps insoluble; and, as we have seen, even after death it can sometimes only be decided by the microscope (p. 385).

The *prognosis* is of course hopeless when the nature of the case is clear. The only chance for the patient is in the possibility of an error in diagnosis. Treatment can only be directed, and often with considerable success, to relieving distressing symptoms. It is remarkable how long such cases linger, and even revive for a time, when apparently at the point of death.

Sarcoma is excessively rare as a disease of the liver. When present it is probably always secondary, and often melanotic. *Cavernous angeioma* has been observed, usually as new growths the size of a marble. Wilks and Moxon regard it as non-malignant.

Cancer of the biliary passages.—The exact locality and extent of the growth vary widely in different instances. In many of those examined at Guy's Hospital its original seat appears to have been the head of the pancreas; in others its starting-point was the pylorus, or the first part of the duodenum, thence it gained the common bile-duct. In some it seems to have commenced in the walls of the gall-bladder, and to have passed downwards until it invaded the common bile-duct. In other instances the cancer has affected the glands about the portal fissure and thus obstructed the flow of bile, and then there has sometimes been primary cancer of some distant part of the intestine. The extent of the cancerous disease, again, is very variable in these cases: there may be nothing more than a small nodule, no larger than a hazel-nut, involving the walls of the common duct; or primary and true hard cancerous stricture, like that of the œsophagus or rectum. When this is the case, gall-stones are generally likewise present. Or all the parts in the portal fissure may be involved in an immense mass of cancer, which may extend to the peritoneum as scattered nodules, or lead to the formation of large and numerous secondary growths in the liver. In some of these cases also gall-stones have been found.

Malignant disease of the biliary passages is almost always true carcinoma. Sometimes, indeed, the growth looks hard and dry, and yields little or no juice on scraping. But in one case, although the growth in the portal fissure looked as if it were composed of a fibroid material rather than of true carcinoma, the liver contained large secondary nodules, the character of which was unmistakable.

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Obstructive jaundice is a constant result of this form of malignant disease, and constitutes one of its most striking and early symptoms.

When there is a mass of cancer about the portal fissure, or in the lesser omentum, the portal vein is almost always pressed upon, and ascites follows. Thus the association of ascites with jaundice is strongly suggestive of malignant growth outside the liver; indeed, with the exceptions of cancer in the substance of the liver and of cirrhosis, this is almost the only disease in which these two symptoms are found together.

Occasionally doubt arises between cancer of the gall-duct and enlargement of the liver from distension by an impacted calculus.

Clinically we may recognise three forms of hepatic cancer: the numerous nodules secondary to some other growth, the rare primary infiltrated form, and that just described, which begins with jaundice, and is commonly, though not always, followed by ascites and enlargement of the liver.

The remaining structural diseases of the liver are degenerative or adventitious. Like cancer, they do not as a rule interfere with the physiological action of the liver except by accidental mechanical pressure on its duct, and are for the most part of pathological rather than clinical interest; or if clinically important, it is because a recognition of their nature may lead to the diagnosis of a primary or concomitant lesion elsewhere. In all of them the liver becomes much larger than natural; and this increase in size is often the only indication of disease. In particular these affections are unattended with pain, and Murchison therefore conveniently grouped them together as "painless enlargements" of the liver.

Simple Hypertrophy.—The author once made a *post-mortem* examination of a case in which death occurred three weeks after an accident. The liver was found to project four inches below the ribs, and it weighed 130 ounces—fully double its normal weight. No morbid change could be discovered in the hepatic tissue, so that the case was set down as one of simple hypertrophy of the organ. This affection is recognised by writers on diseases of the liver, but at present nothing definite is known of its origin (p. 374).

In diabetes the liver is usually found larger than natural, sometimes considerably so, but it never approaches the bulk just described.

Enlargement of the liver without true hypertrophy occurs from chronic passive congestion in cardiac disease (cf. p. 60), and from distension of the bile-passages by long-continued obstruction of the common duct (p. 344).

Leuchæmic Enlargement.—In leuchæmia and splenic anæmia the liver often becomes considerably enlarged, owing to the overgrowth of the lymphatic tissue which forms the portal canals. The spleen is enlarged at the same time. This affection will be noticed in the chapter on anæmia.

The Fatty Liver.—Another condition in which the liver becomes enlarged, without pain or other marked symptoms, is that in which its cells are loaded with fat. In one of our cases of this kind the organ weighed 112 ounces, in another it weighed 155 ounces, or about three times its normal weight. The 'Pathological Transactions' contain a case in which it weighed twelve pounds. A cirrhosed liver often contains much fat, particularly when it is increased in size, and then the nodules on its surface

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can often be felt through the abdominal walls. But in primary idiopathic fatty degeneration the organ remains perfectly smooth. Its edge is somewhat thick and rounded. It is anæmic, and of a more or less yellow colour; but, as Rindfleisch remarks, one must not suppose that it has the same appearance during life, for it can be injected without the employment of any great force, so that the pressure of the blood probably suffices to overcome the resistance excited by the distended cells so long as the heart is beating. The organ is soft, and tears very readily beneath the pressure of the finger. Its specific gravity is diminished, sometimes to such an extent that it floats in water. When it is cut into, it greases the knife, especially if this be warmed. Fragments held in a spirit lamp will sputter, and then burn brightly.

The microscope shows that the accumulation of fat takes place within the hepatic cells, and especially in those which lie towards the periphery of the lobules. These often contain drops of oil so large as to obscure their walls, and an inexperienced observer might suppose that the cells had undergone destruction. This, however, is not the case. The oil can be extracted by ether, and the shrunken nucleated cells remain in their natural relation. In fact, the fatty liver of pathologists is only the fat-stored liver of the physiological absorption which follows every full meal; but what in health is intermittent becomes in disease constant and excessive. Possibly the Strassburg goose would lose his *foie gras* if allowed to go free.

As might be expected, a fatty liver can often be easily detected at the bedside. It may be fouud as low as the umbilicus, or even lower, and the smooth even surface and the soft doughy feel of the edge distinguish this from other enlargements of the organ. The deficiency of resistance may be so great that the liver slips away beneath the hand; one may then have great difficulty in feeling it, even though the parietes are perfectly soft and yielding, and percussion indicates that the organ is much increased in size. This very difficulty, however, points to fatty disease of the liver as the cause of the enlargement.

Ætiology.—The conditions under which the liver is apt to become loaded with fat are numerous, but they may be divided into two main classes, strikingly opposed to one another. In one of these an excess of fat is present in the body generally; in the other there is emaciation, often in an extreme degree.

The first kind of fatty liver occurs, along with general obesity, chiefly in persons who lead sedentary lives, and who eat large quantities of rich food, particularly if they also indulge freely in stimulants. This is evidently analogous to the affection that is artificially produced in geese by the purveyors of the *pâté de foie gras.* The birds are kept in a dark place, with but little space to move in, and are crammed with a farinaceous paste. The consumption of fat within the body is thus reduced to a minimum, while its formation is increased. It first accumulates in the blood, and then is deposited in the hepatic cells.

The other kind of fatty liver cannot be so easily explained. Cases of pulmonary phthisis are those in which it most frequently occurs. This was first noticed by Louis, who found it in one out of every three bodies of those who had died of consumption. At first sight one might be inclined to attribute this to deficient oxidation of fat from interference with the action of the lungs. But if this supposition were correct, the liver ought to become fatty in cases of asthma and of emphysema likewise, whereas, in fact, the other diseases in which it becomes so are such as only resemble

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phthisis in being attended with wasting, namely, cancerous affections, ulcer of the stomach, chronic dysentery, &c. One is therefore driven to assume that in the course of progressive emaciation the blood becomes in some way overloaded with fat, which is stored up in the liver. We may connect the fatty infiltration of the liver with the facts that the patient has generally been bedridden for a long time before death, and has been well fed, often with cod-liver oil. According to Larrey, it is possible by keeping geese shut up in close, hot cages, without food, to induce a fatty enlargement of the liver while the birds themselves become greatly wasted.

It is remarkable that the fatty liver of phthisis is more frequent in women than in men.

Symptoms.—A fatty liver produces neither pain nor jaundice. Many years ago Addison expressed the opinion that a symptom, suggestive if not pathognomonic of the affection, was a peculiar state of the skin, which he described as looking semi-transparent and pale, somewhat like polished ivory, and as feeling smooth, so as to resemble the softest satin ('Addison's Works,' New Syd. Soc., p. 102). This is observed in the form of fatty liver which accompanies emaciation. On the other hand, when the patient is the subject of obesity, the skin acquires a shining, greasy appearance, apparently due to an excessive secretion of fat by the sebaceous glands. It is said that when such persons are hot, the sweat is unable to wet the skin, and runs off in large drops.

Hebra noticed that habitual spirit-drinkers have usually a soft, smooth, and clear skin with free and active sebaceous secretion. It may be that this condition is only found when the liver also is fatty, or both may be the results of alcohol.

Some writers have thought that diarrhœa may depend on fatty degeneration of the liver. No doubt these conditions are often present together, but if not accidentally coincident, they are most likely both effects of one cause.

Significance.—In a case of phthisis or other wasting disease, the detection of a fatty liver does not affect the treatment, except that perhaps cod-liver oil and the like should no longer be given. Nor does it seriously influence the prognosis, since such cases are generally fatal, and the most that can be inferred is the fact, already sufficiently apparent, that the patient's nutrition is greatly damaged. If the same affection should be detected as a part of general obesity, it is this, and not merely the state of the liver which calls for interference.

Obesity in general.—This opportunity is the best that offers for the subject of obesity. No good insurance office will accept at ordinary rates the life of a man whose weight bears more than a certain proportion to his height. It is notorious that such persons bear even slight accidents badly, and succumb to illnesses that would be unattended with danger in healthy subjects. After death their tissues are found to be soft and flaccid, and to break down under pressure much more readily than usual; and decomposition often advances with undue rapidity. The omentum, the mesentery, and the subperitoneal tissue generally, are loaded with fat. The large size of the abdomen in fact presses up the diaphragm during life, and hampers the play of the lungs. The heart also is commonly covered with fat, and its substance is soft and lacerable, so that it readily tears, and has been compared to wet brown paper.

The most important part of the treatment of obesity consists in the regulation of the diet. Some years ago popular attention was strongly drawn to this subject by a pamphlet published by Mr Banting, the upholsterer,

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who, in less than a year, had reduced his weight from 202 to 156 pounds. He was at that time sixty-six years of age, and his height was five feet five inches. Before he began to diet himself he had great difficulty in stooping, was compelled to go downstairs slowly backwards, and used to puff and blow with every exertion, beside being liable to fainting. The articles which he avoided were bread, butter, milk, sugar, beer, sweet wines, and potatoes. He took a liberal supply of animal food. He says that when he had lost his excess of fat he felt better than he had done for twenty years, and the fainting fits altogether ceased.

Such a change of diet should not be made without supervision on the part of a medical man, for in some persons it may doubtless be attended with risks of its own. But the dangers which obesity brings with it far outweigh them.

Habits of early rising and of active exercise are useful in preventing the deposition of fat, but active exercise is beyond the power of those who are already corpulent. Even hard riding does not prevent increasing obesity. Liquor potassæ and other alkaline remedies have been recommended in the treatment of this condition, but they often prove altogether useless. The tendency is sometimes hereditary and insuperable, but walking, moderately restricted diet, avoidance of beer, and the free use of water, with occasional purgation and more frequent sweating, will in most cases succeed in diminishing the patient's bulk and relieving his discomfort.

THE MALARIAL LIVER.—Under the influence of repeated attacks of ague, the liver as well as the spleen may become enlarged so as to become palpable below the ribs. In such cases there is frequently some degree of jaundice present, and constantly the yellowish earthy pallor so characteristic of paludal cachexia (cf. vol. i, p. 346). The condition is apparently one of frequently recurring congestion leading to permanent enlargement. It may result from remittent fevers or (it is said) from residence in a malarious district, even when no febrile symptoms have followed. There is increase of connective tissue, constituting, according to some authors, a special form of cirrhosis. There is, apart from jaundice, deep brown pigmentation of the lobules.

THE LARDACEOUS LIVER.—Prolonged suppuration, syphilis, and perhaps other causes lead to the conversion of the liver, spleen, kidneys, and many other tissues, into a peculiar translucent material, which is known by the epithets lardaceous, waxy, albuminous, or amyloid. Its chemistry and pathology will be described in the chapter on Bright's disease.

A lardaceous liver often reaches a considerable size. In one case it weighed more than eight pounds, and Wilks records another in which it reached fourteen pounds.

One of its most striking physical characters is its greatly increased density. Wilks mentions an instance in which the specific gravity was found to be 1084. It is also extremely hard. Its cut surface looks dry and bloodless, smooth, shining, and translucent. It can be cut into thin slices much more readily than in health. The lobular markings are unduly distinct. Iodine gives its characteristic reaction, as does also methyl violet.

The microscope shows that the hepatic cells themselves are converted into lustrous shapeless masses of the lardaceous material. Those earliest affected are those which lie in the zone of each lobule intermediate between

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its centre and circumference. This position corresponds with the ultimate distribution of the hepatic artery in which the degeneration begins. After a time the change extends inwards to the centre of the lobule, and last of all outwards to its periphery.

Sir Dyce Duckworth has recorded cases in which the lardaceous liver became reduced in size. In one case, after reaching to the iliac fossa and nearly to the pubes, it became in fifteen months about half the size it was, and at the autopsy weighed only 117 ounces ('St. Barth. Hosp. Rep.,' vol. x, p. 57, 1874).

Symptoms of lardaceous disease of the liver are almost absent. As with fatty and leuchæmic enlargement, there is neither pain nor ascites nor icterus. It is recognised by its physical signs.

The liver often reaches down to the level of the umbilicus. During life the edge can generally be felt very distinctly, more readily than that of a fatty liver. It is firm and resisting. The surface of the organ is perfectly smooth, unless gummata or perihepatitis be also present. But such a combination is sufficiently common to be borne in mind, particularly in reference to the diagnosis of hydatids (p. 396).

The practical diagnosis of a lardaceous liver depends on our knowledge of its ætiology. We expect to find it in a case of disease of the bones or of the lungs, accompanied with long-continued suppuration, and particularly in syphilitic cases; and we are confirmed in our conclusion if we find evidence of the same degeneration affecting the spleen, the kidneys, or the intestines.

TUBERCLE OF THE LIVER.—It is not uncommon in cases of phthisis or other tubercular affection, and most common in children, to find the liver full of miliary tubercles, usually yellow and more numerous on the surface than elsewhere. They often accompany similar tuberculosis of the spleen and kidneys, probably appear not long before death, and have little or no clinical significance, although we have seen (p. 343) that cases of jaundice from this cause have been recorded.

Tubercular cirrhosis has also been described (p. 370), and some pathologists would perhaps have reckoned under this head a case recorded by the writer in a boy of thirteen ('Path. Trans.,' 1882).

But a large caseous tubercular mass softening into a pseudo-abscess or vomica, like the cavities of pulmonary phthisis or the tubercular growths of the brain or the kidney, is one of the rarest pathological curiosities.* Why it so seldom is seen is far from clear. Indeed, even the small number of such hepatic vomicæ which have been recorded probably include cases of actinomycosis (cf. vol. i, p. 383).

HYDATIDS OF THE LIVER.—In all the forms of painless enlargement of the liver that have hitherto been mentioned the organ is *uniformly* increased in size. In this respect they differ altogether from a hydatid tumour, which is a rounded elastic swelling, occupying only part of the liver. It may reach a considerable size before its presence is detected, and it often causes not the slightest inconvenience or discomfort to the patient.

Natural history and development.—It was stated in a former chapter (p. 278) that every tapeworm in the course of its development passes through a remarkable phase, in which it forms a bladder embedded in

* The hepatic *vomicæ* referred to by Celsus (ii, 8) were no doubt abscesses.

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the substance of one of the higher animals, and filled with a transparent fluid. Now, a hydatid of the liver really represents this stage in the development of a little tapeworm, which in its mature form inhabits the intestine of the dog, and is called *Tænia echinococcus.* When fully grown this measures at most four millimetres, or about the sixth of an inch in length, and consists of only three or four segments, of which the last alone contains developed sexual organs. It is very common in London dogs, and is often present in large numbers in their intestines. Its ova are discharged with the fæces of the host, and enter the human stomach, either in drinking-water, or on the leaves and stems of raw vegetables.

When an ovum of the echinococcus tapeworm has thus found its way into a human host, it at once enters upon a wonderful career of development. The first change appears to be due to the action of the gastric juice, which dissolves its shell and liberates the embryo as a larva, or *scolex.* This has six chitinous hooks; and therewith bores its way through the walls of the stomach, or small intestine. Its further course is not always the same; it may reach the serous surface, and either enter and develop itself within the peritoneal cavity, or perhaps strike across into one of the solid viscera. If, however, this were its usual course, it ought to be met with in the other abdominal viscera almost as frequently as in the liver. But the fact is that hydatids are far more commonly found in the liver than anywhere else. No doubt this is because the embryo, in piercing the wall of the stomach or intestine, generally falls into one of the rootlets of the portal vein, and is at once washed away by the stream of blood, and carried onwards through the main trunk until it is arrested in a capillary of the portal vein within the liver.

Having thus reached its destination, the scolex proceeds to develop itself into a hydatid. Perhaps it first bores its way out of the blood-vessel, and may travel some distance through the hepatic tissue, as the embryos of other tæniæ are known to do in the organs which they infest. Very soon, however, its movements are arrested. It acquires four suckers and more numerous hooklets, twenty-eight to fifty-two, arranged in two circles. It now grows larger, and, from being solid, becomes converted into a vesicle containing a transparent fluid. It also gives rise to certain changes in the tissues round it, apparently as a result of the irritation caused by its presence. It becomes surrounded by a layer of granular matter, and before long by a distinct membranous investment, consisting of connective tissue, and abundantly supplied with vessels. Henceforth the hydatid is always enclosed in this investment, which grows as it grows, and which may be properly termed its capsule. Thus the capsule of a hydatid is a structure formed from the human tissues, with which the hydatid or echinococcus itself lies in contact but has no organic connection.

Up to this point the development of the echinococcus is precisely analogous to that of the cystic stage of any other tænia—for instance, of a cysticercus. But the further steps are very different in the two creatures. The cysticercus, in order to complete its development, would only have to form a single "head" or "scolex" in its interior. This head would grow as a kind of bud or protrusion from one part of the interior of the cysticercus, and would gradually become provided with its two rows of hooklets and its suckers. But in most specimens of the echinococcus, instead of a single bud or protrusion, a number of them form at different times from the interior of the animal. And each of these buds does not develop into a

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head, but itself forms a cystic body which for a time remains attached to the spot where it arose by a pedicle, but soon becomes detached. It is then called a "daughter-cyst," while the original hydatid that encloses it is termed the "mother-cyst." Each daughter-cyst, again, may develop one or more "granddaughter" cysts in its interior. In this way the echinococcus becomes filled with a number of smaller vesicles of various sizes, which may amount to thousands. If any of them contain other smaller vesicles they are sometimes described as "pill-box hydatids," since their arrangement may be said to resemble that of a "nest" of pill-boxes. Sooner or later the little buds or protrusions cease to form detached vesicles, and develop into very small thin membranous sacs, the pedicles of which are persistent, and which are called "brood capsules," because they give origin to a variable number of "scolices" or "heads," each of which has its row of hooklets and its four suckers, and is capable, under favourable circumstances, of growing into a tænia. These scolices or heads have in England been commonly designated echinococci; and the use of the term accords both with its derivation (ἐχῖνος, hedgehog; κόκκος, grain or berry) and with the intention of Rudolphi, who invented it. But at the present day the term echinococcus is commonly applied to the whole animal, with its daughter-cysts, brood capsules, and scolices. It will be observed that a difference between a cysticercus and an echinococcus is that, whereas the former gives rise only to one scolex, and can therefore ultimately form only a single tapeworm, the latter may develop thousands of both.

Echinococci do not, however, necessarily pass through all the developmental changes above described. Sometimes they fail to produce scolices, and even daughter-cysts. They are then said to be "sterile hydatids" or "acephalocysts." The term acephalocyst was invented by Laennec, because he believed that scolices were never produced by the hydatid which infests the human subject, although he was aware of their presence in hydatids from the lower animals.* It is said that Bremser, in 1821, first discovered scolices in hydatids taken from the human body. Bright gave a drawing of them in the 'Guy's Hospital Reports' for 1837. Even after it was recognised that the hydatids of man usually contain scolices, they were still called acephalocysts, and the term may properly be applied to those hydatids which are really sterile. This is said to be more frequent in the case of hydatids infesting the brain than in those of any other organ.

Multiple hydatids.—Sometimes, but in the human subject very rarely, instead of budding internally to form daughter vesicles, the echinococcus produces them externally. In this way the liver may become riddled with hydatids, not contained in any mother-cyst, but penetrating its tissue in all directions, and even invading the neighbouring organs. A very remarkable case of this kind once occurred in Guy's Hospital, under the care of Dr Rees. A boy was admitted with what seemed to be effusion of fluid into the right pleura, and enlargement of the liver. But when the chest was punctured with a trocar, hydatids escaped. Ultimately he died, and it was found that the liver, diaphragm, and right lung were full of hydatids, which were budding externally in all directions.

Multilocular hydatid.—There is a remarkable form of hydatid—developed

* "There is no doubt that the hydatids in the livers of sheep are animalcules; they have been often seen to move when taken out of the liver and put into warm water."—Baillie, 1797. The discovery of the parasitic nature of hydatids is due to the naturalist Pallas in 1760.

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by this process of external gemmation—which is styled "multilocular" by Continental pathologists. It forms a solid globular mass in the liver, as large as a fist or a child's head. Its periphery is well defined, and it can be shelled out of the tissue in which it lies. But on section it is found to be divided by trabeculæ into a number of small cavities of irregular form, each containing a mass of gelatinous material which is made up of hydatid membranes pressed closely together, and small cysts containing scolices. The individual cysts are never larger than peas, and are often as small as millet-seeds. Virchow supposes that in this form of the affection the parasite occupies the interior of the lymphatic vessels. A multilocular hydatid tumour is always found to have undergone softening in its centre, and to be broken down into a suppurating cavity. Suppurative peritonitis and jaundice are also frequently present at the time of the patient's death. This form of hydatid tumour seems hitherto not to have been observed in England.*

Anatomy.—We must now return to consider the characters of an ordinary hydatid of the liver. This forms a more or less globular mass, varying in size from that of a walnut to that of a cocoa-nut, or even larger; the largest on record is said to be one weighing thirty pounds, which was observed by Luschka. If it is subjected to no pressure in its growth, its form is spherical; but if it meets with more resistance on one side than on another, it may be flattened or egg-shaped, or it may assume an hour-glass form. The extent to which a hydatid is embedded in the liver varies greatly in different cases, and entails very different clinical features. In some cases it would seem that the six-hooked embryo originally lay just beneath the serous covering of the liver; and the hydatid may then form a globular mass depending from its surface, and having so little obvious connection with it that one may find great difficulty in determining that the liver is really the seat of the tumour. In other cases a great part of the sphere formed by the hydatid may lie within the hepatic substance; and its presence may be only indicated by a rounded projection, the curve of which is often little noticeable if the cyst is large. Sometimes, again, a hydatid may reach both surfaces of the liver at once; and the original anterior edge of the liver may then be distinctly made out as a narrow ridge, passing obliquely downwards and to the right across the rounded tumour, which occupies the epigastric and hypochondriac regions. Lastly, a hydatid may be embedded entirely in the back part of the liver, or reach only that portion of its surface which is in contact with the diaphragm, and covered by the ribs.

Symptoms.—Manipulation of hydatid tumours of the liver through the abdominal walls gives different sensations in different cases. The tumour may be quite soft, and fluctuation may readily be detected, a wave being transmitted from one part of it to another; or it may be firm and tense, sometimes of stony hardness. In a certain proportion of cases a peculiar sensation may be elicited by percussion, to which Briançon first drew attention, and on which French writers lay great stress. It is termed the *frémissement hydatique.* The way to detect it is to place three fingers of the left hand upon the tumour, and then to tap the middle finger abruptly with the right forefinger. The other fingers of the left hand may then perceive a peculiar quivering sensation, which was formerly supposed to be due to

* Frerichs suggested that a specimen in the museum of Guy's Hospital, which is labelled "colloid cancer of the liver," might be a multilocular hydatid. But some years ago I carefully examined this specimen, and could not discover any trace of a parasite.—C. H. F.

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the vibration of the daughter-cysts contained in the hydatid, but which, it is now known, may occur with cysts in which there is nothing but fluid. It is far from decisive of the cyst being due to an echinococcus.

Diagnosis.—As a rule, the detection of a hydatid tumour, lying below the ribs, is not difficult. If the cyst should project far from the lower surface of the liver it may be mistaken for a distended *gall-bladder*, or even for hydronephrosis. Distension of the gall-bladder, without jaundice—the common bile-duct being patent—is, however, exceedingly rare; and in *hydronephrosis* the tumour fills the lumbar region more than would a hydatid; moreover, the colon is generally to be detected in front of it by percussion, whereas the downward growth of a hepatic cyst would push the gut backwards.

A tumour which is cystic, and embedded in the substance of the liver, can be nothing but a hydatid.

Simple *retention-cysts* containing serum are occasionally met with in the liver; but they appear never to reach such a size as would enable them to be detected during life, and are of merely pathological interest.

They sometimes occur in the broad ligament, as in a case noted many years ago by the writer. When occupying the substance of the liver they are sometimes very numerous, and in that case are probably not retention-cysts, but the result of vacuolation of the protoplasm of separate secreting cells, as was first stated by Dr Beale (*vide* 'Path. Trans.,' vol. vii, p. 234; see also vol. xxxii, pl. xvii, fig. 2).

When a hydatid is deeply embedded in the substance of the liver, so that it is but little raised above the surface of the organ, and yet causes it to project a long way down into the abdomen, there is often great difficulty in determining the nature of the case. In several instances when the diagnosis of a hydatid tumour was given, the enlargement proved to be due to lardaceous disease of the liver, and the circumscribed tumour to its being intersected by fibrous bands, in connection with *syphilitic gummata*. One diagnostic character of syphiloma of the liver is immobility of the organ during inspiration, due to adhesions of its surface, which are generally present. More or less pain and tenderness on pressure are also common symptoms in such cases. It once happened to the author to direct the performance of exploratory operations in two cases on the same day, and in each of them the tumour proved to be solid; it was probably in both cases a syphilitic and lardaceous liver. These patients did well; but another patient died from the effects of chloroform while undergoing an operation for a supposed hydatid of the liver, and in this case also the tumour was found to be of the nature just described.

In other cases there may be a difficulty in determining whether a tumour of the liver is a hydatid or a *cancerous growth*. The distinction must be based partly on the physical character of the tumour, partly on the presence or absence of general symptoms, particularly pain. It must, however, be admitted that pain is not invariably absent in hydatid disease. Frerichs gives a case in which a hepatic cyst was the seat of violent pains after every manipulation, and they ceased almost immediately after tapping. Moreover, the capsule of a hydatid may inflame and suppurate, and thus cause severe pain. The health of patients harbouring this parasite often appears to be perfect; but they may lose flesh to a certain extent, and may suffer much inconvenience from its pressure on neighbouring organs.

Sometimes, as has already been stated, an echinococcus growing in the

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liver, instead of forming a tumour that can be felt in the abdomen, may project from the convex surface of the organ, under cover of the ribs. If it should attain a considerable size it may then cause the lower part of the chest to bulge considerably, and the edges of the costal cartilages to form a much more open curve than on the opposite side of the body. The intercostal spaces over the swelling may feel more resistent than usual, and may even project beyond the level of the ribs. At the same time the lower part of the chest yields a dull note on percussion, and the case is very likely to be mistaken for one of *chronic pleural effusion*. Such an error may, however, be always avoided by careful observation of the limits within which the dulness on percussion and the enlargement of the intercostal spaces can alone be detected. In cases of hydatid tumour below the diaphragm the area of dulness is bounded above by a curved line, which descends as it approaches the spine posteriorly. In cases of pleuritic effusion the dulness reaches to quite as high a level in the dorsal region, close to the spine, as in the neighbourhood of the right nipple.

The author once saw a case with Mr Durham, which well illustrates this distinction. The patient, a young lady, had been sent to him by a physician, who considered her to be suffering from a chronic pleuritic effusion, the result of an attack of pleurisy two or three months before. The right lower ribs, in the lateral region of the chest, were bulging, the intercostal spaces were tense, and they seemed to yield a sensation of fluctuation. There was increased dulness on percussion over the same part; but in the back the physical signs were in all respects normal. In spite of the history we agreed that the case was one of echinococcus in the liver; and the aspirator at once proved this conclusion to be correct.

A collection of fluid in the right pleural sac might, indeed, be so confined by adhesions as to be undistinguishable from a hydatid in the liver. But this possibility need hardly be taken into account in a statement of the rule that where the physical signs indicate the presence of a collection of fluid limited to the lateral region of the base of the right chest, a hydatid tumour of the liver is present.

Moreover, whatever doubt there may be as to the nature of a cystic tumour of the liver, it is quickly set at rest by the chemical examination of the liquid removed from it by paracentesis with an aspirator.

The hydatid fluid possesses characters different from those of any other liquid that is met with in the chest or abdomen; they are not unlike those of the cerebro-spinal fluid. It is limpid or very slightly opalescent, its sp. gr. is 1007 to 1009, or a little higher; it contains no albumen, so that it does not coagulate either when boiled or on the addition of nitric acid.* There is only a trace of sodic carbonate and chloride. When a glass containing hydatid fluid is held up to the light one can often see floating in it delicate white bodies, so minute as to be only just visible, which rapidly settle to the bottom of the vessel. These are the clusters of scolices, either still enclosed in their brood capsules or (if the latter are ruptured) kept together by their common stalk. They form beautiful objects for the microscope, appearing as bodies of round or slightly elliptical form, with oval calcareous corpuscles scattered through their transparent substance, and each with its crown of hooklets and its four suckers usually retracted into the interior of the cystic body. Often they are still alive, and

* It is said that, like cerebro-spinal fluid, it always contains a minute proportion of grape-sugar, and succinate of ammonia has also been found in it.

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can be seen to move. It was formerly supposed that the scolices, or echinococcus heads, became detached from the main wall of the parent cyst in the course of their growth, and that they could swim about in the fluid. But this was a mistake; they are naturally fixed, and are only set free during the operation of paracentesis.

The discovery of a hydatid scolex, or of one of the indestructible hooklets, whether in the fluid or the solid contents of a cyst of doubtful nature, is of course conclusive, and the membranous wall of a hydatid cyst also possesses microscopical characters which are entirely different from those of any tissue of the human body. This is the case, at least, with the outer of the two coats of a hydatid, or, as it is technically termed, the "cuticula." It is made up of a number of very thin layers arranged concentrically. The smallest portion of it is seen under the microscope to be marked with delicate parallel lines, having a peculiar finely dotted appearance, which is perfectly characteristic. Another peculiarity which belongs to hydatid membranes is the fact that, when they are lacerated, the free edge always rolls itself up, so that the originally inner surface is outermost. Chemically they consist of a modification of chitin.

Events.—It is an interesting question whether there is any natural limit to the life of the echinococcus. No certain answer can perhaps as yet be given, although Reynal is said to have met with an instance in which a tumour of the neck which had existed forty-three years—from the age of seventeen to sixty—when punctured gave issue to an immense quantity of hydatids, all apparently living; and Budd recorded the case of a lady who died at the age of seventy-three, and who was believed to have had two hydatid tumours since she was eight years old. It is certain that in persons who have died at a much earlier age than this, dead hydatids are frequently discovered. Often their contents are deeply stained with bile, and Cruveilhier long ago suggested that the entrance of bile into the capsule by ulceration of some small bile-duct has in such cases been the cause of the death of the parasite. But this opinion, though since generally adopted, seems very doubtful, for two dead hydatids have been found by the author in the same liver, one of them containing bile-stained matters, while the contents of the other were colourless; and it seems unreasonable to attribute the death of the one to the toxic action of the bile, and to leave the death of the latter unexplained. Another supposition has been that hydatids die because their external adventitious capsule is too thick and resistent to allow of their due growth. The fibrous capsule of a dead hydatid is often of cartilaginous hardness, or in great part calcified.*

When a dead echinococcus is found in the liver at an autopsy, its capsule generally contains a putty-like substance, made up in large part of calcareous salts, and mixed with the gelatinous relics of hydatid membranes, which often glisten with cholesterine crystals. The putty-like substance is very like that which occurs in a dried-up abscess, and in all probability is transformed pus which had formed within the capsule of the hydatid—the cause of the death of the parasite or its consequence.

* It is asserted that the mother-cyst (although it may be full of closely packed daughter-cysts) does not appear to be folded, as though it had ever been of larger size. It is therefore imagined that the death of the hydatid was due to its being prevented from receiving the proper amount of nutriment for the supply of its multiplying daughter-cysts. But I have, in one instance of this kind (in which I paid attention to this point), found that the mother-cyst was much folded.—C. H. F.

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However changed the other parts of the parasitic growth may be, the microscope will always find the hooklets unaltered.

Suppurating hydatid.—Inflammation within the capsule of an echinococcus is not an uncommon occurrence, and one which is very important, since it modifies greatly both the physical signs of the affection and its symptoms. If the tumour can be seen or felt in the abdomen this becomes painful and tender and hot, and there may after a while be redness of the skin over it. The patient's health begins to suffer; he may have repeated attacks of shivering, and symptoms of hectic may show themselves. In some cases, however, inflammation of the capsule of a hydatid may apparently take place without marked symptoms.

Rupture of the cyst.—A living echinococcus may burst its capsule and pour out its contents, or a suppurating hydatid may ulcerate and open in various directions. In the former case suppuration of the cavity follows, and thus, unless the death of the patient has immediately followed, one cannot determine whether the creature was alive or not at the time when its capsule gave way.

A hydatid cyst sometimes, but very rarely, makes its way through the abdominal *parietes*. It is said that this may happen even to a cyst in which the parasite is still alive, with a discharge of clear water.

Another direction in which rupture may take place is into the *peritoneum* and this is often not a spontaneous occurrence, but the result of some injury to the abdomen, such as the patient's falling downstairs or receiving a severe kick or blow. Fatal peritonitis generally follows quickly upon an accident of this kind, but unless it is previously known that the patient had a hydatid tumour, it is of course impossible to say why the injury caused such severe symptoms. At least two cases, however, have been recorded in which rupture of a hydatid cyst into the peritoneal cavity seems to have taken place without the patient having been much the worse for it, although there was for some time afterwards fluctuation in the lower part of the abdomen, just as in ordinary ascites. In all probability the different results of rupture in different cases depend upon the circumstance that sometimes a large number of daughter-cysts and scolices are effused into the peritoneal cavity (particularly when the tumour is widely ruptured by extreme violence), but that in other instances only the hydatid fluid is extravasated, either from the hydatid cyst being sterile, or from the aperture being small and the escape of the contents gradual.

Much more commonly a hydatid cyst discharges its contents either into the *stomach* or *intestines*. The daughter-cysts are then vomited or discharged *per anum*; and sometimes air enters the tumour, which thus becomes tympanitic on percussion. The evacuations of membranous portions of hydatids in the fæces sometimes goes on for several weeks or longer; and in the majority of cases the patient ultimately recovers.

The rupture of a hydatid cyst into the *biliary passages* may be a cause of obstruction, and often of febrile jaundice (p. 343).

The hydatid cysts which discharge their contents in any of the directions that have been hitherto considered are generally connected with some part of the liver which is within reach of the ordinary methods of physical examination. But often the tumour bursts not downwards, but upwards into the chest; in these cases its seat is almost always in the upper and posterior part of the liver, so that very frequently no positive physical signs of its presence can be discovered either before or after its rupture.

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Sometimes a hydatid cyst discharges its contents through the diaphragm into the *pericardium;* sometimes into one of the hepatic veins within the liver, the daughter-cysts in the latter case passing straight into the right chambers of the *heart*, and plugging up the branches of the pulmonary artery. The clinical features in both cases are very similar, consisting in the occurrence of sudden death, or, at least, rapidly fatal syncope, in a person who perhaps has hitherto appeared to be in perfect health.

In rare instances, again, a hydatid tumour has been known to discharge its contents into one of the *pleural cavities* (generally the right), with the result of setting up a severe and rapidly fatal pleurisy. But far more frequently, when the diaphragm is perforated by a hydatid, the pleura has previously become adherent. The consequence is that the parasite makes its way into the substance of the *lung*, and sooner or later reaches a bronchial tube, into which it opens, so that its contents are expectorated. Cases of this kind are exceedingly interesting, and it is often a long time before their real nature can be made out.

Many years ago a boy, aged six, a patient of the late Mr Fagge, of Hythe, had suffered for about eighteen months a pain just outside the right nipple, and a constant hacking cough, for which all treatment was useless. He became exceedingly wasted, and was supposed to be sinking. One day his cough left him, and he became exceedingly prostrate, but next morning the cough returned, and he spat up a hydatid cyst and a quantity of pus. From that time he began to recover, and his cough gradually disappeared. Ultimately he died of another disease, and it was proved that the hydatid had originally come from the liver.

A young woman was once attending among the out-patients who had been expectorating hydatids for nearly a year when she first came to the hospital. Next week she had a most violent attack of coughing, which lasted three hours, and it seemed she would be choked. But at last she got rid of a large piece of hydatid membrane, which was apparently a part or the whole of the mother-cyst; for she coughed up no more hydatids, and to a great extent regained her health.

In a case under the late Dr Barlow's care, when the writer was his clinical assistant, a young woman was admitted to Miriam Ward with jaundice, enlarged liver and pyrexia. After a few days she was seized with pleurisy on the right side, and then with expectoration of blood and pus, under which she rapidly sank. No traces of hydatids were found in the sputum; but Dr Barlow supposed that a hydatid cyst in the liver had suppurated and perforated the diaphragm, so as to set up first pleurisy and then suppuration of the lung. This diagnosis was confirmed after death, the original cavity being found in the liver and a large hydatid cyst unruptured in the lung.

When portions of hydatid membrane from the liver are expectorated they are generally colourless, but sometimes are deeply stained with bile. In many instances the patient regains his former state of health when all the hydatids have been voided, and the capsule has contracted so as to close the cavity. This process, however, is not free from risk. In the case of a patient who had spat up hydatids some months before her death, and in whom hæmoptysis at last proved fatal, it was found at the autopsy that the blood had come from a branch of the pulmonary vein; this had become obstructed and dilated into a cylindrical tube as large as a lead pencil, and had afterwards opened into the original cavity of the hydatid cyst, although

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this cavity was now shrinking. Here the parasite had primarily been seated, not in the liver, but in the lung itself.

Prophylaxis.—Before considering what is the proper curative treatment of hydatid tumours, we must ask what measures can be adopted to prevent the echinococcus from entering the human body.

In London and Dublin hydatids are frequently found in the liver, but in Edinburgh this affection is exceedingly rare. In the United States, again, it is said to be very uncommon. On the other hand, in Australia it is far more frequent than in England, and among the inhabitants of Iceland it causes one seventh of the total mortality. Hydatids are said to be very seldom met with in the East Indies. In our own country, it is not likely that people will ever take precautionary measures against infection with hydatids beyond taking care when they eat raw vegetables to have them thoroughly cleansed.

According to our present knowledge, it is evident that this parasite would soon become extinct in all civilised countries if its cystic form were not liable to infest some other animal than man. Dogs acquire the *Tænia echinococcus* only by eating the flesh of some creature in which the scolex is embedded, and they can very rarely have the opportunity of deriving this from the dead human body. Sheep and pigs are believed to be the chief animals besides man which harbour hydatids. Consequently dogs should be prevented eating the offal of these animals, and should be excluded from all slaughter-houses. It has also been suggested that the floor of every kennel should be frequently scalded with boiling water so as to destroy any ova of the *Tænia echinococcus* that may have been voided with the fæces of the dog.

Curative treatment of hydatid tumours is believed at the present day to belong to surgery rather than to medicine. At one time it was thought that the internal administration of iodide of potassium, or even of chloride of sodium, would poison the intruder and lead to the disappearance of a hydatid cyst; but no drug can exert this power, and at the present day the operation of paracentesis is attended with little risk to the patient. An operation should therefore at once be performed when a hydatid is detected which is larger than a billiard ball, and which appears to be increasing in size.

The operations that may be adopted for the cure of the affection are the following: first, some of the fluid may be withdrawn by a grooved needle, a subcutaneous syringe, or an aspirator; secondly, one or two slender needles may be inserted into the cyst, and may either be left in it for ten minutes, and then carefully taken out again, or they may be connected with a galvanic battery, the current from which is allowed to pass through the tumour.

The operation last mentioned—that of *electrolysis*—was practised several times at Guy's Hospital by the author and Mr Durham, who published in the 'Medico-Chirurgical Transactions' for 1871 the reports of eight cases, in seven of which (if not in all) this plan of treatment was completely successful.*

* Two electro-gilt needles were used, which were introduced into the cyst at a distance of one or two inches from one another. Care was taken to observe that they had entered the same cavity, and could be made to touch one another. They were then attached to wires connected with the negative pole of a galvanic battery of ten cells, while the positive pole was made to terminate in a moistened sponge placed over the surface of the tumour at a little distance from the points of entry of the needles. The current was allowed to pass for about ten minutes, after which the needles were withdrawn.

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When this operation was first tried it was supposed that its success would be attributable to the decomposition of the saline liquid contained in the cyst, by which the hydatid would be killed. But further experience led to doubts. In several cases fluctuation could be detected in the lower part of the abdominal cavity a few hours after electrolysis; and in two instances a rash showed itself on the first or second day afterwards, resembling urticaria or scarlatina; a fact explained by a case of Dr McGillivray, in which urticaria followed the discharge of fluid from a hydatid cyst into the peritoneal sac. It therefore seemed probable that the operation of electrolysis led to a similar escape of some of the fluid, and that this was the real cause of the death of the parasite.

In fact, electrolysis is unnecessary, for the same result follows the introduction of the needles without any galvanic current, *i. e. acupuncture.* In two subsequent cases at Guy's Hospital this plan was adopted with successful results.

The other operation—that of *paracentesis*—was advocated by Murchison, and is now often performed with Dieulafoy's aspirator. If a large part of the hydatid cyst be outside the substance of the liver it may be an advantage thus to remove its contents; but if the cyst be enclosed in the liver the aspirator is likely to do harm. The withdrawal of a very little fluid is often sufficient to destroy the parasite, and to ensure the ultimate disappearance of the tumour. To do more is generally useless, and to exert forcible suction by an aspirator upon a cyst surrounded by solid tissue must involve some risk.

The fact is that almost any kind of mechanical interference with a hydatid cyst is capable of curing the complaint with safety to the patient. The question is what operation is least likely to be followed by suppuration of the cyst.

It very often happens that some weeks or even months after tapping the tumour is found to have regained its former size, or even to exceed it. This has generally been supposed to render necessary a repetition of the paracentesis, and when the liquid collects again it is tapped a third time, and so on. The liquid obtained by these later operations differs from hydatid fluid in containing more or less albumen. The augmented size of the tumour is due not to the continued life of the parasite, but to the effusion of serum within its capsule, and after a second or third operation this serum generally contains leucocytes in greater or less numbers, and so passes into pus. On the other hand, if the real cause of the enlargement of a hydatid tumour after an operation be recognised, and if further interference be carefully abstained from, the tumour sooner or later begins to decrease in size again, and after a time disappears. The rule, therefore, would seem to be that no second operation upon a hydatid cyst should be performed within twelve months, unless there be reason to fear that suppuration has already taken place.

Whether there is less danger of the occurrence of suppuration after acupuncture, or after paracentesis with a fine trocar, is perhaps uncertain; still in a series of ten cases treated by the former method (with or without electrolysis, which we have seen to be immaterial) there was not a single instance in which suppuration occurred, or in which any symptoms arose beyond those of very transient febrile disturbance. One patient was up and about the ward on the fourth day, and was discharged from the hospital on the tenth day. Such immunity from severe inflammation of the cyst is certainly

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rare. Dr Duffin has remarked that most of the patients on whom the operation of electrolysis has hitherto been performed were children, and that this may perhaps be the reason why no suppuration has followed; but hydatid cysts may suppurate under other conditions, even in children.

Another point that must not be forgotten in considering the relative result of acupuncture and paracentesis is the possibility that the cyst may have undergone suppuration, without any marked symptoms, before the operation. If this ever occurs, it would certainly make acupuncture dangerous; but we may probably exclude it when the patient is free from fever, and has not experienced the slightest pain or uneasiness.

Even when a considerable time has elapsed after the apparent cure of a hydatid tumour there still remains a liability to the occurrence of suppuration within it. Dr Wilks has seen more than one instance in which this has happened after a long interval. The retrograde changes which lead to the ultimate disappearance of such tumours are no doubt very slow in their progress. A patient of Dr Moxon's once lay for several weeks in the clinical ward in bed on account of a pain in the hepatic region, which had come on some time after the performance of an operation for a hydatid tumour. The tumour itself could no longer be detected, but the pain was intense and very obstinate. Doubtless some of the sensitive structures of the abdomen were subjected to traction by the shrinking of the capsule of the cyst.

Beside the operations that have been hitherto alluded to several others have been proposed and advocated: among them are the injection of ox-gall, iodine, or oil of male fern into the hydatid cyst; the introduction of a large trocar and the formation of a fistulous opening, or the penetration of the tissues external to the tumour by gradual stages, so as to allow of the formation of adhesions between the capsule of the cyst and the parietal peritoneum. But when these various plans were proposed, it was not known how safely and successfully hydatid tumours may be treated by the simpler methods above described.

When, however, suppuration has once occurred within the capsule of a hydatid tumour, or perhaps in any case if the tumour is very large, it should at once be opened with due antiseptic precautions, the cyst having been first fixed by sutures to the abdominal wall so as to obviate the risk of extravasation. A large trocar should be used, and if the tumour contains secondary cysts, as many of them as possible should be removed. A drainage-tube is then inserted, and the cavity is washed out every day. The obliteration of the cavity in such a case is necessarily a very slow process, and attended with much risk. But with proper management and good nursing, cases of this kind usually end favourably. An enormous multiple hydatid cyst, in a patient under the present writer's care in February, 1886, was opened in this way by Mr Jacobson with complete success, although innumerable echinococci continued to escape for many weeks.*

Hydatids in other organs.—Echinococci are seldom found anywhere than in the liver as their primary seat, and still more rarely have any clinical interest attached to them. Leuckart appears to think that for every three cases of echinococcus of the liver there may perhaps be one in some other organ, but even this estimate is probably above the mark.

* On the treatment of hydatid tumours in the liver, see an important discussion at the Clinical Society in December, 1887 ('Trans.,' vol. xxi).

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Davaine collected very carefully all published cases of hydatids occurring in various parts of the body, and he found that among 200 cases of this kind (those of the liver being excluded) there were about 40 cases of hydatids of the lungs, about 30 of the muscles and subcutaneous connective tissue, 30 of the kidneys, 26 of the pelvis, 20 of the nervous centres, 17 of the bones, and 10 of the heart.

When an echinococcus develops itself in one of the *lungs*, it is found as a rule in the base of the right lung, a fact no doubt due to the six-hooked embryo having penetrated into the organ from the liver by its own movements. Much more often, however, a primary hepatic cyst opens into the lung as above explained (p. 399). Thus in one way or the other pulmonary hydatids are almost always migrated parasites of the liver. Clinically, hydatid disease of the lung is scarcely likely to be suspected until one or more of the daughter-cysts have been expectorated. It produces hæmoptysis or purulent expectoration with fever, so that it has frequently been taken for phthisis.

Hydatids of the *brain* have occasionally been met with in the *post-mortem* room; the symptoms are undistinguishable from those of other cerebral tumours. It may be remarked that the capsule of a cerebral hydatid is exceedingly thin and delicate, and the cyst is sometimes acephalic (p. 394).

Echinococci have been found in the *heart*, where they may give rise to very varied symptoms, or sometimes to none at all.

The *spleen* is very rarely the seat of a hydatid tumour. In one case of the kind there was a large tumour in the left hypochondrium, but until an autopsy had been made, it remained uncertain in what organ the parasite was seated. Hydatids of the *kidney* will be mentioned again.

In some of the great cavities, such as the *pleura* and the *pericardium*, the echinococcus may grow to a considerable size, without any tendency to the formation of a capsule round it, the natural serous membrane seeming to take its place. This is said not to be the case with the *peritoneum*, in which hydatids are described as always having a proper capsule. However this may be, hydatid disease of the peritoneum has considerable clinical interest. One form of it is apt to be mistaken, even by the most skilful surgeon, for cystic disease of the ovaries; and an attempt has several times been made to remove such a tumour by ovariotomy. In another form of this affection a number of distinct globular tumours are found in different parts of the abdominal cavity, some of them having been developed in the omentum, and others in the interspaces between the different viscera.

Lastly, there is a very remarkable variety of hydatid tumour, which develops itself in the *pelvis*, between the bladder and the rectum in men, or behind the uterus in women. In these cases the six-hooked embryo doubtless gets into the serous cavity when it has penetrated the walls of the stomach, and rolls by its own weight into the most depending part of the peritoneal sac. The result is the formation of a tumour which may assume an oval form exactly like that of a distended bladder, and may occupy precisely the same situation. In a case of this kind which occurred in our wards it was supposed that the bladder was full, but of course the catheter failed to give relief. The patient died, but until the autopsy its real nature was not even suspected. Bright relates a similar instance, and several others have been placed on record by different observers; so that it may be laid down as a rule that whenever a fluid tumour is felt in this position, which cannot be reduced in size by the introduction of a catheter

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into the bladder, one should think of the possibility that an echinococcus may be present. The hepatic region should be carefully examined in such cases, and indeed whenever there is reason to suspect the existence of hydatids in any other part of the body; for very commonly the liver is also infested with echinococci.

A parasite belonging to the class Arachnida, and to the same order of mites as that to which the *Acarus* and the *Demodex* appertain, is occasionally found in the liver, but it is always dead and encysted. It is called *Pentastoma tænioides*, and has no clinical significance. We have frequently noted it at Guy's Hospital.

The rare presence of the liver fluke (a Trematode worm) in man has been already mentioned in the chapter on Entozoa (p. 462).

A few cases are on record of *Psorospermia* occurring in the human liver. These oval encysted parasites, probably a phase in the development of Gregarinidæ, are frequently found clustered into opaque white nodules in the livers of rabbits, and when extremely numerous produce emaciation and death. They have been recognised in the alimentary canal and liver or other glands, not only of mammals and fishes, but also in insects, mollusks, and worms, and are probably ubiquitous. They are rarely met with in man, and still more rarely produce symptoms; but possibly some cases of tubercles in the liver have really been examples of this disease. A remarkable case has lately been published by Mr Silcock in the 'Path. Trans.' (xli, p. 320). An excellent account of our present state of knowledge concerning these parasitic monozoa, with plates and ample references, is contributed by Dr Delépine to the same volume of the 'Pathological Transactions' (1890, p. 346).

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DISEASES OF THE KIDNEYS

FUNCTIONAL DISORDERS

Falstaff. Sirrah, you giant, what says the doctor to my water?
Page. He said, sir, the water itself was a good healthy water; but for the party that owned it, he might have more diseases than he knew for. I *K. Henry IV.*

Arrangement adopted—Polyuria and oliguria; high and low specific gravity—Diabetes insipidus—Renal inadequacy.
Reaction: alkaline urine—Urinary pigments: urobilin and indican.
Lithic acid and lithates—Oxalate—Phosphatic deposits—Cystine and calcic sulphate—Ammoniacal urine—Bacilluria.
Hæmoglobinuria—General characters—Toxic form—Infantile form—Paroxysmal form—its relation to malaria and to Raynaud's disease.

In dealing with the affections of the kidneys we will begin with their functional disorders, and afterwards pass on to the forms of nephritis comprised under the title Morbus Brightii, of which a common symptom is albuminuria: then will follow suppuration of the kidney and pyelitis, with effects of obstruction by calculi or otherwise, affections characterised by the presence of blood or pus in the urine.

The concluding chapter will treat of tubercle and new growths of the kidney, and of renal parasites.

Diabetes, a disease in which the urine contains sugar, but which is *not* in any other sense a disease of the kidneys, will be treated separately.

The present chapter deals with certain morbid conditions of the urine which are independent of any organic lesion of the kidneys, and are characterised by some alteration in its *quantity*, *density*, *colour*, or *reaction*, by the formation of *precipitates* or deposits, by the occurrence of putrefactive changes, or by the appearance of *hæmoglobin* in solution.

In the sixteenth and seventeenth centuries the inspection of the urine was a regular part of the physician's office, as is proved by many passages of the Elizabethan dramatists and many scenes of the great Dutch painters. But the examination was as useless as the Chinese examination of the pulse, until modern chemistry made in some degree clear the causes of the colour, turbidity, or transparency of the secretion. At present, though many important gaps in our knowledge remain, we know the principal normal and abnormal constituents of urine, and can determine their relative amount. In particular, we know what conditions belong to the physiological changes of the body, and what are purely chemical and take place after the urine is voided.

There are many morbid conditions of the urine, such as the absence of chlorides in pneumonia (vol. i, p. 993), and the presence of bile-pigment in

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jaundice (p. 338), or of leucin and tyrosin in acute yellow atrophy of the liver (p. 377), which require no further account in this place. Our present task is to give an account of the changes in the urine which are primary pathological processes rather than symptoms of organic disease.

Among these morbid conditions there is theoretically a line of distinction which it would be very desirable to draw clearly. In some the urine contains an abnormal material, or a normal material in undue quantity, as the result of a morbid process in organs remote from the kidneys. In others the formation of a deposit is due merely to a relative excess or deficiency of acidity in the urine, or to the occurrence of fermentative changes in it after it has been secreted. Unfortunately, however, one cannot carry out this division completely; for in many cases, as, for example, with regard to oxalate of lime and lithic acid, there is the greatest difficulty in determining whether the total urine does or does not really contain too much of the materials which are thrown down as deposits. Nor is it possible in every case to be sure whether the abnormality of the urine is due to derangement in another organ, *e. g.* the liver, or in the kidney itself, or to chemical changes in the urine after secretion. Nevertheless the order adopted will as far as possible bring out first those affections in which the urine is faulty from the time of its formation, and afterwards those in which its morbid condition is a subsequent event.

Changes in the quantity and specific gravity of the urine.—Between the amount and the density there is an inverse proportion. In health, when the urine is very abundant, it is always pale and watery; when it is scanty, it is dark and of high specific gravity. Among diseases, diabetes is the only one in which pale and abundant urine is of great density; and a diminished flow of urine is not likely to be accompanied with a low specific gravity except when the kidneys have undergone extensive destruction in advanced Bright's disease.

In health, the daily amount of urine ranges in different persons and under different circumstances from forty to fifty ounces or more; it is liable to great variation from day to day, and probably there are some perfectly healthy people who habitually void either considerably larger or considerably smaller quantities. The specific gravity of the collected twenty-four hours' urine generally varies between 1018 and 1022; but in cold weather, or after drinking water freely (whether the pure element or in any form of liquor), single specimens will yield a much lower specific gravity, 1005 or less.

Physiology teaches that the quantity and the density of the urine depend mainly upon the activity of the blood-current in the renal glomeruli. According to Ludwig the determining factor is the pressure of the blood within the vessels of the tufts; according to Heidenhain it is rather the rapidity of its passage through them. The chief point in favour of the latter theory is the fact that when in experiments on animals the outflow of blood through the renal veins is checked, the urine becomes scanty and of high specific gravity; but Cohnheim shows that this fact is perhaps not so completely inconsistent with Ludwig's view as might at first sight appear.

Clinically the distinction appears to have but little significance. The only local cause of obstruction of the renal veins is thrombosis of these veins, or of the inferior cava above their mouths. Thrombosis of the renal veins is not very infrequent as a complication of lardaceous and other forms of

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Bright's disease, and Dr Moxon recorded in the 'Guy's Hospital Reports' for 1869 two cases in which it was associated with injuries to the lumbar spine. But in the former class of cases the existence of lesions in the renal cortex makes it impossible to determine the effect of the thrombosis upon the characters of the renal secretion; moreover, as the obstruction is probably developed very slowly and gradually, collateral channels have time to enlarge and can carry on the circulation. In both of Dr Moxon's cases the arteries were plugged as well as the veins, so that the urinary secretion was of course entirely suppressed. Bartels has related in 'Ziemssen's Handbuch' a case in which the inferior vena cava was closed by a thrombus from the point where it passes along the groove in the back of the liver downwards; in that instance, however, the urine, which contained blood and albumen, was secreted in fair quantity, and was of sp. gr. 1011 to 1013.

When the systemic venous circulation generally is obstructed, as in cases of heart disease and of pulmonary emphysema, the urine is almost constantly found to be scanty and of high density. This accords as well with Ludwig's as with Heidenhain's theory, since the pressure in the systemic arteries is, in these conditions, always lowered. When, under the influence of digitalis, the blood-pressure in the glomeruli can be brought up to a sufficient point, it is surprising how greatly the flow of urine increases.

Prout thought that he recognised a disease, which has since been termed *azoturia*, the fundamental symptom of which was an increase in the excretion of urea. In cases which have been placed under this category the flow of urine has been generally excessive; but it is very doubtful whether they have been rightly interpreted. Urea is a powerful diuretic, but its quantity varies greatly with the amount of nitrogenous food; moreover, the specific gravity of the urine depends on the salts as well as on the urea excreted, beside the activity of the skin and lungs, and the amount of water taken in.

An opposite state of urine, in which, while the quantity is augmented, the density is reduced, is of frequent occurrence. We shall find it to be an important symptom of certain forms of Bright's disease, in which there is an abnormally high arterial tension. But it is also seen as an independent condition.

Diabetes Insipidus.*—In this disease the patient passes enormous quantities of urine, exceeding even those that are voided in saccharine diabetes itself, for which it is pretty sure to be mistaken until chemical analysis shows that no sugar is present. The specific gravity constitutes another marked distinction between the two affections, for in diabetes insipidus it is often scarcely above that of water, and seldom reaches higher than from 1003 to 1007. The daily secretion of urine may range from fifteen to thirty and even forty pints. It is clear, and almost, if not quite, colourless; it has a faintly acid reaction, but early undergoes the ammoniacal fermentation. It of course contains a very small proportion of solid matters,

* *Synonyms.*—Pseudo-diabetes — Polydipsia — Polyuria — Hydruria — Diuresis. The Greek word διαβήτης (*i. e.* a siphon, so called from its resemblance to a pair of compasses, from διαβαίνω, to straddle) was applied by Aretæus and Galen to the condition in which whatever a man drinks runs through him as through a siphon. Most of such cases were probably true saccharine diabetes, others were chronic Bright's disease. The discovery that in many of them sugar is present was made by Thomas Willis in 1670, and from that time *diabetes insipidus* has been distinguished from *diabetes mellitus*.

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but nevertheless the total daily amount of urea excreted appears to be rather excessive than diminished.

One abnormal constituent, *inosite*, or muscle-sugar ($C_6H_{12}O_6$), has been sometimes detected in the urine; but its presence is probably accidental, for it is often absent; and, on the other hand, it sometimes occurs in saccharine diabetes, in Bright's disease, and in healthy urine. Since inosite is to be found in small quantity in the heart and other muscles (as also in the lungs, liver, spleen, and other organs), its excretion in the urine may be the result of the excessive transudation of water through the tissues; and Strauss is said to have discovered it in the urine of three healthy persons, who, for the purpose of experiment, had drunk a large quantity of water.*

Symptoms.—A tormenting thirst is one of the main symptoms. At one time it was imagined that this might really constitute the essential feature of the disease, which should therefore be properly regarded as a "polydipsia" rather than a "polyuria." But experience has shown that the patients always pass more urine than healthy persons who drink the same quantities of fluid; moreover, when in a case of diabetes insipidus the amount of drink is restricted, the urine does not fall in the same proportion, and the tissues become dry.

In some instances the general health remains wonderfully good. Sir William Roberts cites the case of a farm labourer, aged fifty-one, who had been affected for twenty-four years, drinking from thirty-two to thirty-six pints of water daily, and voiding urine in proportion, and who yet remained able to do all kinds of hard work, such as threshing and mowing. And another case is recorded of a woman who bore eleven children while suffering from the disease. It is especially noted that the farm labourer's skin was moist, and that he perspired freely when at work. As a rule the skin is obviously dry and harsh; and Roberts himself had under his care a boy who, although rosy and plump, had a dry skin and tongue.

Most patients complain that their rest at night is disturbed by the frequent desire to micturate. Other symptoms are, according to Roberts, a painful dryness and heat of the mouth and fauces, pains in the loins and in the epigastrium, an indifferent or sometimes a voracious appetite, enfeeblement of bodily strength and of mental vigour, irritability of temper, and abolition of the sexual functions. Senator, in 'Ziemssen's Handbuch,' states that the temperature of the body is slightly lowered, possibly from the large quantity of water that is swallowed having a cooling effect. Enforced abstinence from drink aggravates most of the symptoms; the skin then becomes hot, there is an intolerable sense of sinking, or intense pain at the pit of the stomach, and at last the intellect becomes impaired. Sir Thomas Watson relates the case of a boy aged eleven, who was limited during twenty-four hours to drinking a pint and a half of fluid, and who nevertheless passed ten and a half pints of urine. That he absorbed water from the air seemed to be clear from the result of weighing him at short

* Inosite occurs in many unripe fruits (as French beans, whence it has been called *phaseolo-* or *phaséo-mannite*). It is crystalline, soluble in water, and sweet like glycose, but does not reduce copper or rotate the polarized ray. Scherer's test for inosite in solution consists in treating with nitric acid, evaporating cautiously to dryness, moistening the residue with ammonia and solution of chloride of calcium, and evaporating again, when a rose colour makes its appearance. This test only succeeds with nearly pure inosite; and therefore to detect it in urine Gamgee recommends Gallois' test of evaporating and treating with solution of mercuric nitrate and evaporating again, when a rose colour appears which fades on cooling and returns with heat ('Phys. Chem.,' i, p. 338).

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intervals. The polydipsia and diuresis lasted for three years, and the boy died of "scrofulous tubercles" in the brain and lungs.

Diagnosis.—Diabetes insipidus is not a common disorder. In London hospital practice it is decidedly rare; and with regard to published statistics there is a doubt whether they are not more or less vitiated by the inclusion of cases in which Bright's disease would have been found present if an autopsy had been made. Roberts, indeed, avowedly places in his collection of seventy-seven cases three which ended fatally, although in each of them the kidneys were affected with a marked degree of atrophy in association with hydronephrosis, *i. e.* "consecutive Bright's disease." It is clearly important to distinguish from diabetes insipidus all cases in which the polyuria is a symptom of an organic lesion of the kidneys. But it is possible that in some of the cases in question (two of which were published by Sir Peter Eade, of Norwich) the hydronephrosis may have been a secondary result of the frequent micturition, just as we shall find hypertrophy of the bladder may be a consequence of saccharine diabetes.

The clinical diagnosis of diabetes insipidus must therefore always be somewhat uncertain in persons advanced in years, for renal cirrhosis comes on insidiously and without much albuminuria. Probably, however, mistakes might be prevented by careful observation of the state of the arterial tension, which in diabetes insipidus appears to be lowered rather than excessive. Even in young subjects one must not overlook the possibility of the presence of hydronephrosis from calculous disease in childhood.

Ætiology.—Among Roberts's cases there were seven in which diabetes insipidus was said to have begun in infancy, or from the time of birth, fifteen in which it began between the ages of five and ten years, thirteen between ten and twenty years, sixteen between twenty and thirty years, fifteen between thirty and fifty, and four between fifty and seventy. Males bore to females the proportion of five to two.

In a few instances there was a well-marked history of the occurrence of the disease in several members of the same family; the most striking example of this seems to be one recorded by Lacombe, in which a mother, her three sons, her daughter, her brother and his children were affected in turn. Trousseau believed that diabetes insipidus is not uncommonly seen in persons whose parents had suffered from diabetes mellitus, or from albuminuria. In some cases it has been attributed to exposure to cold or heat, drinking cold fluids when heated, intemperance, muscular efforts, or mental emotions. A more probable exciting cause is a blow or fall upon the head, or an organic lesion of the brain. As regards the traumatic cases, Roberts remarks that in some of them the polyuria has set in with its maximum intensity on the very day of the accident, but in others not until after the first loss of consciousness had passed off, or a few days later, or even in one case not until the time of subsidence of severe nervous symptoms at the expiration of six months. In some of the cases associated with cerebral lesions there have been tuberculous or other tumours, occupying various positions,* but in one instance projecting into the fourth ventricle from its floor.

Such observations possess special interest in consequence of the fact that an affection like diabetes insipidus can be produced experimentally in animals by injuries to certain parts of the nervous apparatus. Bernard first showed that this effect followed puncture of the floor of the fourth ventricle at a point a little above the glycosuric centre. There is also experimental evidence as

* See Dr Alexander Hughes Bennett's case, 'Brit. Med. Journ.,' Feb. 24th, 1883.

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to the production of an excessive flow of urine by irritation of the cervical sympathetic and by lesions of the spinal cord. The probability seems to be that the immediate cause of the affection is a dilatation of the renal arteries, from defect of the controlling action of their vaso-motor nerves. So far as is yet known, there are no nerve-fibres influencing the renal function which are secretory (in Heidenhain's sense, *i. e.* trophic, affecting the secreting epithelium). It is worthy of mention that, in a case observed by Külz, diabetes insipidus was accompanied by spontaneous persistent ptyalism (the patient spitting from twelve to eighteen ounces of saliva daily); for this is another symptom that has been produced in animals by puncture of the floor of the fourth ventricle.

Prognosis and treatment.—The course of diabetes insipidus varies widely in different cases. Roberts cites one instance in which it set in with absolute suddenness: the patient, a woman aged thirty-four, went to her work one morning at six o'clock in her usual health; two hours later she was seized with intense thirst and diuresis, which continued from that time.*

In those cases which follow injuries to the head the affection commonly subsides in the course of a few weeks or months, but cases are on record in which it had been of six or seven years' duration. And of the non-traumatic cases beginning from infancy, some are stated to have run on for fifty years or more. When there is a cerebral tumour, this is of course almost sure to prove fatal in a comparatively short time. Otherwise diabetes insipidus does not in itself appear to have much tendency to destroy life; for the patient generally succumbs to an intercurrent malady, such as phthisis or pneumonia. In one of Roberts's cases the patient, a man aged sixty-two, is said to have suffered for twenty years from excessive thirst and diuresis.

It is a remarkable fact that the occurrence of some inflammatory or febrile disease has in several cases led to the temporary, or even permanent, subsidence of diabetes insipidus. Thus one patient, who had suffered from it for eighteen years, recovered completely after an attack of acute rheumatism; and another after an attack of pleurisy, treated by a blister which suppurated for thirty-five days. Roberts suggests that it might be worth while in future cases to try the effect of a large blister applied to the back of the neck, or to the epigastrium.

Among medicines the most useful seems to be valerian, which was prescribed by Trousseau in enormous doses, two and a half drachms of the extract daily, or even more. To one patient he gave nearly an ounce, and in the course of four months recovery took place. Roberts relieved a boy under his care with the valerianate of zinc, increasing the dose until it reached twenty grains a day. Dr Dickinson has found benefit result from codeia. Ergot is another remedy that has been used with more or less success. The constant galvanic current has been recommended by some German observers. One pole may be applied to the loin on one side near the spine, and the other to the corresponding hypochondrium for five minutes; and then they may be transferred to the opposite side of the body in the same

* I once saw a patient at Dulwich, who had several distinct but very short attacks of what appeared to be diabetes insipidus. I have unfortunately preserved no notes, but my impression is that each attack lasted a day or two, and I remember that he passed enormous quantities of urine, and became for the time exceedingly prostrate and exhausted. I think that this recurred at intervals of some weeks.—C. H. F.

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manner; or, as Külz advises, the positive pole may be placed upon the nape of the neck, and the negative pole, first to the loins for four minutes, and then to the epigastrium for the same period of time.

Renal Inadequacy.—Under this name Sir Andrew Clark has described ('Brit. Med. Journ.,' i, 1883) a class of cases of which the main feature is that the kidneys appear unable to excrete more than the normal daily quantity of urine (from forty to fifty ounces); while even this is of low specific gravity (1002 or 1003 to 1008), and is deficient in urea (not containing more than 2 per cent.), though the amount of uric acid may be natural. Even if these patients drink freely of water they do not pass a larger quantity of urine; and a liberal diet with a full allowance of wine is obviously injurious to them. The urine in such cases is devoid of albumen, and contains no casts. Although Sir Andrew Clark admits it to be possible that the kidneys are on the way towards chronic Bright's disease, he says that when he has had an opportunity of making an autopsy the organs have appeared to be healthy.

The patients are generally ailing, without being definitely ill. They are apt to take cold, and do not get rid of the cold easily, and are also liable to be attacked with pneumonia, pleurisy, or pericarditis, without apparent reason. They recover slowly from even slight injuries, and they do not bear surgical operations well—a fact noticed independently by Sir James Paget. They complain of *malaise* and weakness, and unfitness for work; they sleep badly, are subject to headache, and suffer from nervousness. Sir Andrew Clark does not say anything about the state of the arterial tension in these cases. He describes patients so affected as ultimately developing a condition very like myxœdema, with pink and white faces, a dry, puffy skin, a slow articulation, and a somewhat staggering gait.

The main points in the plan of treatment which he advises for them are a very sparing diet and careful management of the skin. He allows for breakfast bread and butter and an egg; for the midday dinner not more than half a pound of meat with vegetables, and afterwards some pudding; about six or seven o'clock bread and butter again, with an egg.

Reaction of the Urine.—Urine has normally an acid reaction, which is probably due to the presence of the acid phosphates of soda and potass.* The greater acidity of the urine when a healthy man lives on a meat diet, and of carnivorous compared with herbivorous animals, is explained by the acid phosphates and sulphates derived from muscle. The same applies to the acidity of urine during pyrexia, when the muscles waste and the excretion of urine is increased.

The degree of acidity of the urine (that is of the whole collected through twenty-four hours) is commonly expressed in terms either of the

* It has been surmised that the organic acids of urine—especially hippuric and uric acids—may be wholly or in part in a free condition, and may take some share in causing the acid reaction. The behaviour of urine towards various organic colouring matters (especially towards congo-red, which is rendered of a deep blue or a violet colour by highly dilute solutions of free acids, including hippuric acid) is such, however, as to disprove this hypothesis. Other facts point in the same direction, as the non-precipitation by urine of a solution of sodium hyposulphite, and the impossibility of separating the hippuric acid of urine by simply agitating it with ether (*Gamgee*).

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dried carbonate of soda required to neutralise it, or of an equivalent weight of oxalic acid. Roberts found that in a healthy man it amounted on an average to about fourteen grains of the carbonate; but there are wide variations, the range during a period of nineteen days being from six to more than twenty-three grains. Writers who give it in terms of oxalic acid say that it corresponds to about thirty grains.

At different periods of the day, however, the reaction of the urine is by no means uniform. After each of the principal meals it becomes for a time decidedly less acid, and is often alkaline. This now well-known fact was originally pointed out by the late Dr Bence Jones. One cause is the formation of gastric juice, which coincides with increase of alkalinity in the saliva, and perhaps the pancreatic juice. Another cause of diminished acidity of the urine is the absorption into the blood of bicarbonates produced by the digestion of the citrates, tartrates, malates, &c., derived from the food. Consequently it is likely to be well marked after meals consisting largely of fruits and of other substances in which salts of the vegetable acids are abundant. In medical practice the effect of food upon the reaction of the urine is seldom noticed except by accident, because what is passed from the bladder is generally a mixture of the secretions formed by the kidneys over a considerable period of time. But one sometimes finds a specimen alkaline when it is passed in one's consulting-room in the course of the morning by a patient who has been to the closet after his breakfast; and the urine passed by candidates for insurance between one and two o'clock, after lunch, is very often neutral or alkaline.

In some abnormal conditions the urine passed throughout the whole twenty-four hours is found, when collected, to have an alkaline reaction, not due to ammoniacal fermentation of the urine after it is secreted. Quincke has observed this in patients with chronic vomiting, as the result of dilatation of the stomach. A like condition of the urine has also been noticed in patients who have the stomach regularly washed out.*

Under other circumstances a persistent alkalinity of the urine from fixed alkali is not common. But Bence Jones observed such cases, and so have Hutchinson and Roberts. Some of these patients were anæmic, chlorotic, dyspeptic, or phthisical; but others were apparently in health. Often the urine is alkaline for two or three days together, and then acid for a time, becoming again alkaline later on. But sometimes it remains steadily alkaline for weeks.

With vegetarians, as with herbivorous animals, an alkaline state of the urine is natural.

Urine which is alkaline from fixed bases is commonly turbid when passed, and on standing throws down certain of its solid constituents, chiefly phosphates of the alkaline earths. This deposit will be further considered below with the other urinary sediments (p. 424).

In the *treatment* of cases in which the urine is habitually alkaline from fixed alkali the main thing is to improve the general health by change of air to the sea-side or to a mountain health resort, exercise short of fatigue, and other measures tending to the same end. The direct administration of acids is found to have very little effect.

* This cannot, as Quincke supposed, be due to loss of acid, for the acid of the gastric secretion is neutralised by the bile and pancreatic secretion before it can be reabsorbed. Probably, as Dr Gamgee suggests, it is due to absence of absorption of the acid phosphates and sulphates of the food.

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URINARY PIGMENTS.—With regard to the substances that give to urine its various shades of colour in health and in disease there is still much uncertainty and confusion.

Urobilin.—According to Salkowski and other writers, the principal urinary pigment is a substance to which Jaffé first gave the name of *urobilin*, *hydrobilirubin* of Maly ($C_{32}H_{44}N_4O_7$). It is amorphous, sparingly soluble in water, soluble in alkalies and in alcohol, and it gives peculiar absorption lines in the spectrum; a green fluorescence is produced by its ammoniacal solution on the addition of chloride of zinc. Vierordt, however, has shown that this cannot be the only colouring matter, inasmuch as the spectrum of urine is not identical with that of a solution of urobilin; the normal pigments are more than one. And Huppert, in his eighth edition of the well-known work of Neubauer and Vogel, declares that the real pigments of normal urine are still unknown. Urobilin, he states, is not present as such, but in the form of a chromogen, which yields it on the addition of mineral acids or by other oxidising methods, sometimes by exposure to air. In certain pathological conditions urobilin exists in the urine in a formed state; and the quantity that can be extracted is far greater than natural, though it still amounts to only from $\frac{1}{32}$ to $\frac{1}{16}$ in a thousand parts of the fluid. Urobilin can be derived from bile-pigment, or from hæmatin or hæmoglobin, by a process of deoxidation (Hoppe Seyler); and it can be oxidised to choletelin. MacMunn distinguishes between normal and febrile urobilin by the aid of the spectroscope; and believes that urobilin is not identical with reduced bilirubin and its products in the intestines, choletelin, or stercobilin.

There is, however, every reason to believe that the colouring matters of the urine (including urobilin and its chromogen) are derived more or less directly, and probably by a process of reduction, from the hæmoglobin of the blood.

It is especially in febrile urine, and in that passed by patients with obstruction of the venous circulation, that urobilin is found in excess. But it must be remembered that such urine is generally scanty, so that the increase may not be so great as it appears. Salkowski says that constipation seems not to augment the urobilin in the urine. In jaundice there is often a great excess, which, however, can be recognised only after the bile-pigment has been precipitated and removed. The urine may likewise be found loaded with urobilin before an attack of jaundice and after it has passed off.

Indican, &c.—The fact that *indigo-blue* is sometimes present in the urine was noticed many years ago by Prout, and afterwards by other observers, some of whom showed that this colouring matter in many cases made its appearance only when the urine had been exposed to the air. Schunck, of Manchester, first recognised in urine the constant presence of *indican*, a colourless material, which he had also discovered in plants, and which readily passes into indigo-blue by oxidation. More recently, however, it has been found that the indican of urine is not identical with vegetable indican; according to Baumann, it is an indoxyl-sulphate of potass ($C_8H_6NKSO_4$). Jaffé in 1872, and Baumann and Brieger in 1879, observed that indican could be made to appear in the urine of animals in large quantity by feeding them with indol (C_8H_7N), or by injecting that substance under the skin. Now, indol is formed within the intestine in dogs, and to some extent in man, as the result of a change in albumen induced by the pancreatic ferment. The absorption of it from the intestine may therefore be fairly supposed to give rise to the presence of indican in the urine; and Jaffé has in fact detected indican in very large quantity in cases of obstruction

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of the small intestine or of strangulated hernia, and also in dogs after ligature of a loop of small intestine. But it is quite another question whether this is the sole, or even the usual source of a morbid excess of indican in the urine. Senator ('Centralblatt,' 1877) and Heninger ('Deutsches Archiv,' 1879) have investigated the conditions under which such an excess is met with. The former finds it in states of inanition and wasting, such as arise from cancer of the stomach, gastric ulcer, multiple lymphomata, phthisis with diarrhœa, or granular disease of the kidneys. The latter insists that the excess is especially marked in cases in which wasting is dependent upon affections of the intestinal canal. He observed it not only when there was constipation, but also when diarrhœa was present, and even in cases of *cholera nostras*. On the other hand, in cases of "catarrhal" (idiopathic or "simple") jaundice, and in cases of cirrhosis of the liver, the amount of indican in the urine was always small. The general result of these observations appears to be that the recognition of indican in the urine is at present useless from a practical point of view. It is often found in large quantity in urine which is pale and contains little formed pigment.

The test for indican, as given by Jaffé, is to add to the urine an equal volume of hydrochloric acid, and then to pour in drop by drop a solution of chloride of lime, shaking the fluid well, and adding no more of the chloride after a greenish colour begins to appear. If any considerable quantity of indican is present, a blue colour will soon show itself; and if the quantity is very large, indigo-blue will be deposited in flocculi. A dilute solution of bromine may be used instead of the chloride of lime. The blue pigment may be afterwards extracted by agitating with chloroform or ether; and in this way the amount of it may be roughly estimated.

According to Brieger, a still more frequent constituent of human urine is an allied substance, skatoxyl-sulphate of potass, which is derived from *skatol* (C_9H_9N), this being (like indol) a product of the decomposition of albuminous substances within the intestine. Both indol and skatol have a strong fæcal odour. Jaffé's test with hydrochloric acid and chloride of lime gives a reddish-violet instead of a blue colour when the skatoxyl-sulphate is present.

The urinary pigment, which gives a pink or red colour to deposits of lithates, has been called *uro-erythrin*. It is amorphous, not very soluble in water, but dissolved by hot alcohol, and when dry turned green by Liq. Potassæ. Uro-erythrin is sometimes spoken of as identical with what Dr Golding Bird called "purpurin." Probably, however, he included under that term several substances which are now described as distinct, since his test for it was to add hydrochloric acid to the hot urine, when he obtained a colour "varying from a delicate lilac to the deepest crimson."

Adventitious pigments.—In conclusion, it must not be forgotten that urine may show colours which are due to the administration of drugs. For example, rhubarb colours the urine a deep gamboge-yellow, which is changed to red by the addition of ammonia. After operations under the carbolic acid spray, patients often pass urine of a dingy, sometimes almost black colour; this is due to the excretion of pyrocatechin, $C_6H_4(OH)_2$, and its subsequent oxidation. Senna communicates a brownish, and logwood a reddish tinge. Santonin gives a conspicuous orange-yellow colour to the urine if alkaline, a rich golden-yellow if acid.

A curious point observed by the late Dr Moxon is, that in persons who are taking iodide of potassium the addition of nitric acid to

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the urine produces an orange-coloured zone, the appearance of which is characteristic.

URINARY DEPOSITS.—We have already spoken of leucine and tyrosine as precipitated from the urine in cases of acute atrophy of the liver, and we shall in a future chapter discuss the subsidence of urinary casts, epithelium, pus, and blood.

The urinary sediments now to be described are saline or other inorganic compounds which separate from their solution in the urine. They are of clinical importance from one or both of two different points of view: either as indications of disturbance of the chemical processes in some other part of the body, or else as involving the risk of the formation of gravel or calculus within the urinary organs. They are (1) lithic acid and the lithates, (2) oxalate of lime, (3) earthy phosphates, (4) cystine, (5) sulphate of lime.

*Lithic acid and lithates.**—In urine having the normal acid reaction lithic acid exists in the form of acid salts (probably, as Bence Jones believed, quadrurates) of soda and other bases, which at the temperature of the body are fairly soluble. But as the fluid cools, these are often precipitated. Very slight changes may disturb the balance between them and other saline ingredients, so as to separate the lithic acid, which is then thrown down, inasmuch as it requires a very large quantity of water (14,000 parts in the cold) to hold it in solution. According to Roberts, the quadrurates, under the circumstances to be presently stated, form with water biurates (of soda, potash, or ammonia) and uric acid; the biurate in the presence of acid phosphates (of soda and potash) is reconverted into quadrurate, and thus more uric acid is thrown down.†

Lithic or uric acid ($C_5H_4N_4O_3$).—As a deposit from urine, this substance appears in the form of crystalline grains which have a reddish colour, so that they often look almost exactly like cayenne pepper. They commonly lie loose at the bottom of the fluid, but sometimes they adhere to the sides of a glass vessel, or may float in a film upon the surface. Their colour is not proper to the acid itself, for this, when derived from other sources, is colourless; it really belongs to urinary pigment, for which uric acid seems to have a strong attraction, and which is consequently carried down with it. Dr Lionel Beale says that he has three or four times seen colourless crystals of uric acid deposited from urine which happened to contain hardly a trace of colouring matter.

The form of uric acid crystals is primarily that of a rhombic prism or lozenge, but they present a great many varieties of shape. Sometimes they are very short and thick cylinders; sometimes they form rods which seem to have rectangular extremities; sometimes they appear as flat plates, pointed, oval, or of a halberd shape. Very often they form large stellate aggregations, and sometimes fan-shaped masses. Dr. Ord has stated ('Med.-Chir. Trans.,'

* Lithic acid was the name given by its discoverer Scheele in 1776, and was used by Prout and Bence Jones. Uric acid (Harnsaüre) and urates are later synonyms and less distinctive.

† Scherer many years ago asserted that during the first few hours after being voided, urine, as a rule, undergoes what he terms an "acid fermentation." This, however, is not now believed to be the case. By Voit and Hoffman ('Bayerische acad. Sitzungsbericht,' ii, p. 79) it is maintained that the acid phosphate of soda (the cause of the acid reaction of urine) gradually takes away from the uric acid more and more of the bases with which it is combined, so that an appreciable diminution in the acidity of the urine may be produced in this way, inasmuch as the uric acid, being deposited in a solid form, is no longer capable of affecting the reaction.

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lxiii) that the deviations in form which those crystals present from the regular four-sided or six-sided plates that are seen when the pure acid is crystallised from water, depend upon the presence of mucus and of colouring matter, which substances favour Rainey's "molecular coalescence" rather than crystallisation, so that by a kind of compromise the resulting crystals, instead of having sharp angles and straight sides, are more or less rounded off, and also tend to cohere together in masses having a common centre. He has also found that the presence of albumen in the urine still further modifies the form of the crystals, rendering them small and thick, with their angles more or less nearly equal, so as to give them the barrel- or cask-shape. The association with sugar, on the other hand, tends to produce flat and elongated crystals, which may have the typical hexagonal shape that is otherwise so rarely seen in specimens derived from urine.

A microscopical deposit of lithic acid is sufficiently distinguished from any other that occurs in the urine, by being crystalline and by its yellow colour.* Its weight, its red colour, its solubility to the naked eye in alkalies, and insolubility in dilute acids are equally characteristic. Moreover, unless the quantity be very small, the well-known murexide test can be easily applied.

Mixed amorphous lithates or urates.—The commonest of all urinary deposits consist of a loose pulverulent substance, which varies in tint, but is of a deeper colour than the urine from which it is derived. It is often spoken of as brick-coloured, or "lateritious" (*later*, a brick). It generally settles quickly, leaving the urine above almost clear, but sometimes it remains a long while diffused. Not infrequently, if the urine has been put aside while still warm, different strata of the deposit in the same glass have different colours—fawn-coloured, orange, brick-red, pink, or purplish. A part adheres to the side of the vessel as a sort of film or bloom, which is not easily cleaned off unless a little liquor potassæ is used.

With the microscope this precipitate is seen to consist of minute granules, which are coarser or finer, and more or less opaque, according to the closeness of its aggregation. All doubt as to its nature may be removed by applying heat. As soon as the turbid urine is warmed, it becomes bright and clear; and even when albumen is present, there is seldom any difficulty in obtaining a satisfactory result, for the lithates dissolve at a far lower temperature than that at which albumen coagulates.

The lateritious deposit was formerly spoken of in this country as urate of ammonia, while German writers described it as a urate of soda. In reality it consists of a mixture, in different proportions in different cases, of the urates of soda, potass, and lime, to which that of ammonia is only exceptionally added. Moreover, the quantity of uric acid in it is largely in excess of that which would correspond even with the acid salts of these bases (for uric acid is bibasic), being in fact about twice as much (quadrurates), and making up 80 or 90 per cent. of the whole precipitate. Roberts says that this loosely combined acid can be separated from the acid lithates by warm water (which must of course be used sparingly), or even by cold water, with which the deposit is to be repeatedly washed upon a filter.

Crystalline lithates or urates.—It is a curious fact that urate of soda is never deposited from urine in those needle-shaped crystals with which we are familiar in gouty concretions, and which may also be readily obtained artificially from solutions of the salt. In some cases, however, it

* This yellow colour is due to urobilin (or whatever be a better name for the urinary pigment); pure uric acid and urate of soda are white.

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forms opaque globular masses from which project spiny crystals, straight or curved. These "hedgehog" or "thorn-apple" (*Stechapfel*) bodies occur in putrid ammoniacal urine. But they are also sometimes seen in patients (especially children) who are suffering from febrile disorders. Their occurrence probably depends upon the urine being very scanty and concentrated, and being detained in the bladder. In one case a child three years old was suffering from fever, and had passed no water for three days: while Roberts was examining the abdomen, the child cried, and urine began to flow: the first that came was turbid and of a gamboge-yellow colour, containing the spiny masses; then, after about an ounce had passed, there followed several ounces of clear fluid.

Clinical significance.—In medical practice, deposits of lithic acid and of lithates have to be looked at from two points of view.

First, there is the question whether they are likely to be the cause of calculi, or of lumbar pain, hæmaturia, and other symptoms of pyelitis.

As regards free uric acid, much depends upon whether or not it is precipitated soon after the urine is passed from the bladder. As Roberts remarks, if the crystals are seen before the urine cools, there is always a risk that they may also be formed within the urinary passages. Even if the deposit takes place within three or four hours it is certainly not natural, though it hardly requires special treatment. But if it does not occur until after twelve hours or longer, it has no pathological significance whatever.

Amorphous urates cannot in themselves produce irritation of the kidneys or bladder, since they appear never to be precipitated so long as the urine is of the temperature of the body. But the hedgehog crystals of lithate of soda are perhaps less innocent.

Secondly, there is the question how far deposits of lithic acid or of lithates indicate a disturbance of the chemical processes by which nitrogenised substances in the blood or in various organs are prepared for excretion by the kidneys. We cannot too strongly insist that the formation of such precipitates is not in itself a proof that the quantity of uric acid is excessive. In the case of the amorphous urates a great deal depends upon the *temperature* to which the urine falls when it cools. It does not appear that there is any other cause than the lower temperature for the fact that deposits of these substances are so much more frequently observed in winter than in summer. Again, whatever diminishes the *amount of water* excreted by the kidneys increases the likelihood that urates will be precipitated. This seems to be the reason why amorphous urates are so often seen in healthy persons after violent exercise and after profuse sweating, and also in patients who are suffering from any disease, such as rheumatic fever, attended with perspiration in its whole course, or who are passing through the crisis of an attack of pneumonia when there is excessive perspiration. Again, the degree of *acidity* of the urine is also of importance. Most observers say that urates are never deposited except from urine which is acid, but according to Salkowski the reaction is occasionally found to be neutral. In the case of free uric acid, the degree of acidity of the urine is the most essential factor of all in determining its precipitation. In addition to a high degree of acidity, Roberts mentions poverty in chloride of sodium and other mineral salts and low amount of pigmentation, as conditions which favour deposit of uric acid, independent of its abundance in the urine.

Practically we may probably assume that cold after the urine has been passed, and acidity with concentration before its being passed, are the most

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common causes of deposit of the mixed lithates; and that a high degree of acidity is the most common cause of the appearance of crystals of lithic acid.

To determine the exact significance of deposits of lithic acid or of lithates we need quantitative analyses. A good method is to precipitate with hydrochloric acid, to collect the uric acid upon a filter, and to weigh it; a variable amount of the uric acid remains in solution and must be allowed for.* Salkowski and Ludwig introduced a method which depends upon the formation of urate of silver; this is said to be more accurate, but there are difficulties in carrying it out correctly. Dr Pavy has advocated ('Med.-Chir. Trans.,' lxii) the use of the ammoniated cupric solution, which he employs for the estimation of sugar. Dr Haycraft has since introduced a modification of the silver method, which will be found described in the 'Journal of Anatomy and Physiology,' vol. xx, p. 695. It is more rapid, but less accurate.

The total quantity of uric acid excreted on mixed diet by healthy persons in the twenty-four hours is not large; as a rule, it is from five to eight grains. The proportion between the uric acid and the urea is usually as one to fifty or as one to sixty.

It is remarkable that uric acid crystals are very common in the urine of children, even infants at the breast; and calculi of uric acid are frequently found in the bladder of boys. The explanation of this fact is not obvious, nor the equally curious one that these calculi, though common in hospital patients, are rare in private practice.

Liebig supposed that lithic acid is formed by oxidation out of the same materials as urea, and represents a stage in the formation of the latter substance in which the oxidising process is as yet incomplete. This hypothesis, however, has never been proved. The most obvious way of testing it seems to be that of adding uric acid to the food, and determining whether or not the amount of urea excreted afterwards undergoes an increase. Wöhler and Frerichs tried this experiment, and came to the conclusion that the uric acid was mainly converted into urea; other observers have since repeated it with a like result. Salkowski found in dogs that allantoin appeared also to be formed out of the acid; but as this must itself have arisen by a process of oxidation, he regards it as rather tending to confirm than to upset the view that uric acid constitutes a step in such a process. The same may be said likewise of the facts which seem to show that oxaluric acid and oxalic acid may be formed in the body out of lithic

* The details of this process and of the silver process next mentioned in the text are given as follows by Dr Gamgee:—"In order to determine the amount of uric acid, 200 cubic centimetres of the filtered urine (from which any albumin which may be present has been separated by boiling) is treated with 10 or 15 c.c. of hydrochloric acid of specific gravity 1·12, and set aside for forty-eight hours.

"At the end of that time the uric acid is collected on a small weighed filter; the filter and uric acid are carefully washed, and the washings added to the filtrate. The filter and uric acid are then weighed, and by subtracting the weight of the former that of the greater part of the latter is found.

"The combined volume of the urine and the washings of the filter is now carefully ascertained, and for each 10 c.c. 0·00048 grm. (nearly half a milligramme) of uric acid is added to the amount already directly obtained by weighing.

"Salkowski and Ludwig's method consists in precipitating a known quantity of urine with a specially prepared ammoniacal solution of silver. The precipitated urate of silver is collected on a filter, washed with ammoniacal water, and then decomposed by boiling it with a special solution of sodium sulphide, silver sulphide being precipitated whilst all the uric acid is obtained as urate of soda in solution. From the latter, after concentration, the uric acid is precipitated by means of hydrochloric acid."

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acid. In birds lithic acid is derived from the same substances (aspartic acid, glycin, leucin, and asparagin) which in mammalia pass into urea. Urea itself, when given to birds, is said to undergo conversion into lithic acid; this, at any rate, indicates how close is the relationship between the two bodies. But according to the latest authorities the formation of lithic acid is separate from that of urea throughout, and is not a stage in the process.

Clinically, several sets of facts have been adduced in support of the view that lithic acid may be excreted in excess as the result of a deficient supply of oxygen, and take the place of urea; but this explanation seems to be open to question in every case. For example, as to the "uric-acid infarcts," which are seen in the kidneys of newly born infants, Cohnheim remarks that he has observed them especially in strong healthy children who had breathed well, whereas they were often absent in cases in which there was pulmonary atelectasis, bronchitis, or broncho-pneumonia. So, again, many observers have shown that there is in leuchæmia a marked increase of uric acid in the urine, both absolutely and relatively to the urea. This has been attributed to the deficiency of red discs as oxygen carriers. Pettenkofer and Voit, however, found ('Ztschft. f. Biol.,' v) that in a case in which the excess of lithic acid amounted to 64 per cent., the absorption of oxygen and the evolution of carbonic acid from the lungs were normal. Lastly, Bartels some years ago ('Deutsches Arch.,' 1865) endeavoured to show that in various affections attended with insufficient aëration of the blood the amount of lithic acid in the urine is increased in proportion to that of the urea, the proportion rising from the normal rate of one to fifty or sixty up to that of one to thirty-five, or even of one to twenty-five. But experiments made on animals by Senator and others have for the most part failed to confirm these observations; and of late it has been shown by Fränkel, and also by Fleischer ('Virch. Arch.,' 1882), that a constant effect of dyspnœa is actually to augment the secretion of urea. Possibly the over-activity of the respiratory muscles plays a part in bringing about this result, and at any rate we may agree with Salkowski that the conditions in dyspnœa are far too complicated to allow of our attributing solely to deficiency of supply of oxygen the changes in the urine that may be associated with it.

But it is especially in relation to disorders of the chylopoietic viscera that the meaning of deposits of uric acid or of urates has to be considered by practical physicians. Roberts made during seven days a series of observations on a person who dined at 2 p.m. and afterwards took no solid food till the next morning. He found that during the period when the urine was alkaline after the meal—which was from about 4 p.m. till 7 p.m.—the quantity of lithic acid excreted in each hour was three times greater than it was from 9 p.m. till 11 p.m., or later on during sleep. The proportion which the acid bore to the rest of the urinary solids, and even to the water, was likewise greater, though, being alkaline, the urine of course threw down no deposit of lithates. The food taken while these observations were being made was very simple. Consequently it seems fair to conclude that the effect of rich food and of frequent meals in increasing the excretion of uric acid would be even more decided. And, as a matter of fact, many persons who habitually live plainly, and who ordinarily pass clear urine, find that the occurrence of a lateritious deposit is an inevitable consequence of any considerable indiscretion in diet; while other persons, whose rule it is

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to indulge in the pleasures of the table, secrete during a large part of every day urine that becomes turbid with urates as it cools. It is true that such deposits do not in themselves prove an excessive excretion of lithic acid, but, if we also take Roberts's observations into consideration, we may probably infer that there is this excess. At any rate, no theoretical doubts can do away with the practical significance of this symptom, especially if it is associated (as we so constantly find it to be) with a foul tongue, yellow conjunctivæ, an irritable temper, and all the other signs of lithæmia (p. 333). Of course, even if we admit that the amount of lithic acid contained in the urine in such cases is absolutely increased, it does not at all follow that it is increased relatively to the urea. That substance itself may be increased likewise, though, as it is readily soluble, there is nothing to show it. But what seems to prove conclusively that deposits of lithates are not merely an indication of excessive nitrogenous elimination as the result of the ingestion of undue quantities of nitrogenised food is, that among the articles of diet which are most apt to be followed by the appearance of such deposits in the urine are some (such as sweet things, port wine and champagne) which are themselves non-nitrogenous. It is difficult to resist the conclusion that such substances are capable of disturbing the balance of the chemical changes which nitrogenous foods (and also probably the products of the waste of nitrogenous tissues) undergo in the liver or elsewhere in the body, and of causing an excess of uric acid to be formed. Parkes found, it is true, that no excessive excretion of uric acid occurs as the result of the experimental addition of either sugar or starch or alcohol to the food. But, in all probability, the limits of health were not overpassed in the observations upon which these statements were founded, as they are in the cases of lithiasis just referred to.

Dr Haig has found that the excretion of lithic acid and lithates is increased by taking alkalies and diminished by acids; the explanation apparently being that in the former case the lithic acid or acid lithates in the liver and spleen are rendered more soluble, and thus carried off in larger quantity to the kidneys ('Journ. of Physiology,' vol. viii, p. 211).

The presence of *colouring matter* and chromogens in the urine in excessive quantity is generally held to afford corroborative evidence of lithiasis beyond that which is afforded by the mere presence of the uric acid or of the urates. The deeper the tint of a lateritious deposit occurring in a non-febrile patient, the more surely is it generally regarded as a proof of lithæmia; and the same conclusion is drawn from the formation of pigment in large quantity on the addition of mineral acids to urine. The late Dr Golding-Bird, for example, was convinced that the presence of what he termed "purpurine"—detected by the violet colour which followed the addition of hydrochloric acid to urine and heating—was a proof of "derangement of the hepatic function." He thought it was sometimes a valuable clinical aid to diagnosis in cases in which there was an abdominal tumour of doubtful nature, or in which it was uncertain whether ascites was due to hepatic disease or to chronic peritonitis. The same significance applies to the zone of pigment of various hues that is so commonly observed when urine is poured upon nitric acid. This seems to depend upon the presence, in different proportions, of the chromogen of urobilin, indican (or rather indoxyl-sulphate), and skatoxyl-sulphate of potash (p. 415). The tint is sometimes crimson, sometimes purple, sometimes bluish black. The darker purple is usually seen in cases of marked constipation.

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Oxalate of lime ($CaC_2O_4 + 4Aq$).—This substance produces in the urine a cloud like that caused by mucus, and so little noticeable in an ordinary vessel that a patient's attention is seldom, if ever, attracted by it. Roberts, however, says that when the deposit is allowed to form in a conical glass vessel it presents appearances which are quite characteristic. The sides of the glass become marked with fine lines, running transversely or obliquely, and making it look as if it were scratched; these are due to the crystallisation of the salt along minute irregularities left on the surface of the glass when wiped. In the urine itself there is near the bottom of the vessel a soft pale grey mucous-looking sediment, and above this a snow-white denser layer with an undulating but sharply limited surface. If a drop of the deposit be taken up with a pipette, and placed under the microscope, the oxalate is generally seen to form small colourless octahedral crystals, beautifully transparent and lustrous. So transparent are these octahedra that all their facets and angles are commonly visible at the same time. They have a flattened shape, the principal axis being much shorter than the other two. In size they vary considerably; the late Dr Golding-Bird found that the length of the sides of different specimens ranged from $\frac{1}{5600}$ to $\frac{1}{750}$ of an inch. They usually lie upon one pole, and then have the appearance which is aptly compared to that of a square envelope. But in different positions they may seem to have a rhombic outline, or even one which is rectangular or hexagonal. They are really octahedra, two four-sided pyramids set base to base. Other modifications in their shape are due to flattening of their lateral edges. This gives them a dodecahedral form, so that they consist of a rectangular prism with a pyramid on each summit. It is also said that half-crystals are sometimes seen—pyramids on a square base. Much more rarely oxalate of lime assumes a non-crystalline character; it is then said to appear in the form of "dumb-bells." Recent observers, however, have shown that the real shape of these bodies is that of a flattened, rounded disc, with a central depression in each surface; it is when these discs lie on the side that they look like dumb-bells. They, too, vary in size. According to Dr Golding-Bird the long diameter ranges from $\frac{1}{1420}$ to $\frac{1}{500}$ of an inch; their short diameter from $\frac{1}{2500}$ to $\frac{1}{750}$ of an inch. There was at one time doubt as to the chemical nature of the dumb-bells. Dr Bird was disposed to think that they differed in composition from the octahedra, and suggested that they might consist of the oxalurate of lime. But this question has been *finally* set at rest by the observations of Dr Ord, who has shown that when oxalic acid and lime come into contact in the substance of a mass of gelatine, *both* forms are obtained. The assumption of a discoid, rather than of a crystalline character, is doubtless due to the influence of colloid substances. Dr Beale has sometimes found dumb-bell crystals in the interior of renal tube-casts.

Chemically, oxalate of lime may be distinguished by its being insoluble in potash and in acetic acid. The shape of the octahedra, however, is in itself a sufficient proof of their nature; it is only the dumb-bells which *might be* mistaken for like bodies of a different composition.

The origin of oxalate of lime is still unknown. There is no doubt that *it* may in part be derived directly from the food. Many vegetables and fruits contain this salt; not only rhubarb (which is so largely eaten in the spring in England) and sorrel (which on the Continent forms a common article of diet), but also, to a less extent, spinach, cauliflower, asparagus, tomatoes, apples,

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and grapes. Buchheim and Piotrowski found that from 10 to 14 per cent. of the quantity of oxalate ingested could be recovered in the urine.

Auerbach detected oxalate of lime in the urine of dogs when fed entirely on animal food; so that probably oxalic acid may be also formed within the human body as the result of chemical changes. But as yet there is no certainty as to its source. English physicians of a former generation were inclined to think that it arose out of sugar; its presence in the urine in a conspicuous form has sometimes been observed in connection with diabetes; but this can hardly be said to have much bearing on its origin. A more probable view is that it is derived from uric acid by a process of oxidation with which chemists in the laboratory are familiar. Schunck's discovery, that oxaluric acid in minute quantity is a normal urinary constituent, is generally held to confirm this view, inasmuch as that substance forms a step in the process in question. But it seems not yet to have been positively shown that the ingestion of uric acid is followed by an increased excretion of oxalic acid.

Clinical significance.—Prout and Golding-Bird regarded the recognition of the oxalate of lime in the urine as affording a key to the right explanation and to the successful treatment of a group of symptoms which they enumerated, and of which the chief were a constant pain or sense of weight across the loins, irritability of the bladder, incapacity for exertion, impairment of sexual power, dryness of the palms of the hands and soles of the feet, a painful susceptibility to external impressions, nervousness, hypochondriacal depression, and emaciation. But in the urine of many patients who have such symptoms no oxalate can be found, while in that of many other patients who are quite free from them it is present in abundance. Again, Fürbringer has shown that no conclusion as to the quantity of the salt contained in the urine can be drawn from the fact that crystals are discovered in the urine. Healthy persons, according to Schultzen ('Reichert's Archiv,' 1868), pass about a grain and a half in the whole of the twenty-four hours; in a morbid condition, in cases of jaundice, he found as much as seven and a half grains. But, from what is known with regard to uric acid, it might well appear that this increase is too small to allow of its leading to disturbance of the general health.

However this may be, the chief clinical importance of oxalate of lime in the urine depends upon the risk of its deposition while the secretion is still within the body. It may furnish a clue to the probable presence of a calculus formed of the same substance, or to that of minute agglomerations producing lumbar pain, hæmaturia, or pyelitis. All of these effects will be discussed further on. At present it only remains to state the conditions under which the salt is apt to be deposited.

It has long been known that, in some cases at least, the octahedral crystals are formed slowly in the urine *after* it has been voided. They have been found to be both more numerous and larger in urine that has stood for a time than they were when it was first passed. Dr Rees even maintained in his 'Croonian Lectures' for 1856 that all the oxalic acid arose out of uric acid in the urine itself, especially when heat was applied to it for testing purposes. This, however, seems to be very doubtful; and it may be noted that Neubauer ('Ztschrift. f. anal. Chem.,' 1868) found that even on adding oxalurate of ammonia and chloride of calcium to urine no oxalate of lime was formed, the only change being the conversion of the oxalurate into carbonate of ammonia when the fluid putrefied and became alkaline.

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Moreover, Voit and Hoffman seemed to have satisfactorily accounted for the gradual separation of the oxalate of lime from urine which had for a time held it in solution. Neubauer, as far back as 1856, showed that this salt can be kept dissolved by a solution of the acid phosphate of soda. Now, it appears that when urine is allowed to stand, the urate of soda in it becomes acted upon by the acid phosphate of soda, so that an acid urate of soda, and ultimately free uric acid, are formed while the phosphatic salt loses its acidity. Consequently the conditions under which alone the oxalate of lime can be held in solution are no longer present, and it crystallises out. Obviously, therefore, the fact that octahedra are discovered in a patient's urine is in itself no reason for supposing that there is danger of the deposition of oxalate of lime in the renal pelvis or in the bladder. And it would seem to follow that the danger must be less in proportion as the urine is more highly acid. Almost all observers, however, are agreed that oxalate crystals are most often found in very acid urine; Salkowski alone (influenced possibly by theoretical considerations) speaks of them as being present chiefly in urine that is only just acid, neutral, or faintly alkaline. They are sometimes seen in association with deposits of urates or of uric acid; and radiating crystals of phosphate of lime are not uncommon in urine containing oxalates, even, as Dr Beale says, when it is acid.

Oxalate of lime is insoluble in hot and cold water, in potash, and in acetic and dilute hydrochloric acids. Only strong hydrochloric acid, with the aid of heat, decomposes this very stable salt.

From what has been already stated it is evident that *treatment* is by no means necessary for all cases in which oxalate of lime is discovered in the urine. Dyspepsia and the effects of dyspepsia must be dealt with in the same way as if no such deposit were present. As a rule the nitro-hydrochloric acid does more good than alkalies. Cold sponging, the use of a flesh-brush, exercise on horseback, change of air to the sea-side or to an elevated health resort, are each of service. The food should be light, digestible, and varied. Leube, however, cites Cantani as having found that the urine was rendered free from the oxalate by an exclusive meat diet. Patients who have had lumbar pain or hæmaturia should be cautioned against eating rhubarb. To prevent the deposition of the salt within the urinary passages the best plan seems to be that of maintaining the naturally acid state of the urine; but Roberts says that in some cases the urine has temporarily ceased to contain the crystals when it has been rendered freely alkaline.

Earthy phosphates.—The amorphous phosphate of lime ($Ca_3P_2O_8$) is by far the most abundant constituent of the "white gravel" which is thrown down in alkaline urine. It collects as a flocculent deposit, always much paler than the supernatant liquid, and thus distinguishable from the fawn-coloured or brick-red amorphous lithates. It also forms an iridescent film upon the surface, the cause of which is said to be the escape of carbonic acid from the urine. Mixed with this is occasionally found another phosphate of lime ($CaHPO_4 + 2Aq$), which is crystalline. It forms rods or needles, smaller at one end, so as to be club- or bottle-shaped, and generally grouped together into stars, rosettes, fans, or sheaf-like bundles. Still more rarely present is the phosphate of magnesia ($Mg_3P_2O_8$), which forms elongated plates with oblique ends; it seems only to have been recognised as a precipitate by two observers, Tollens and Stein. It is a soluble salt, so that it is not likely to be precipitated unless present in very large quantity; and,

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moreover, there must be no ammonia in the urine, since that base, if present, unites with the phosphate of magnesia to form another salt, the well-known "triple phosphate."

All phosphatic deposits are dissolved by acetic acid, a test distinguishing them from other urinary precipitates.

Carbonate of lime seems to be frequently mixed in small quantity with the amorphous phosphate of lime; sometimes it appears in small spheroids.

Not infrequently urine when warmed in a test-tube becomes cloudy and opaque, as if it contained albumen; but this is owing to a separation of phosphate of lime that had hitherto been retained in solution. A drop of acid (nitric, hydrochloric, or acetic) decides the point by dissolving the cloud.* This precipitate has generally been attributed to the heat driving off carbonic acid from the urine. But Salkowski has shown that this cannot be the case, since when the turbidity is but slight the urine often becomes clear again on cooling. Dr Walter Smith ('Dublin Journ. of Med. Sci.,' 1883) has since taken up this subject, and has arrived at the conclusion that the precipitation of phosphate of lime by heat depends upon "a nice adjustment of the proportions and basicity of the phosphatic salts in the urine." He supposes that when the fluid at the ordinary temperature contains the dicalcic phosphate ($Ca_2H_2P_2O_8$), held in solution by the presence of other salts, the effect of heat may be to resolve it into tricalcic phosphate ($Ca_3P_2O_8$) and monocalcic phosphate ($CaHPO_4$), the former of which is insoluble; cold, on the other hand, may lead to the inverse change and to a disappearance of the precipitate. Urine which thus deposits its phosphates on being heated does not always do so on neutralisation by acids; indeed, it is sometimes alkaline or neutral to begin with.

If the test-tube is heated until all the precipitate has fallen, and this is allowed to subside, the clear supernatant urine will, after being decanted off, again deposit a cloud of earthy phosphates on being heated, and this may be repeated several times—another proof that they are not held in solution by carbonic acid.

Clinical significance.—The presence of a phosphatic deposit in urine alkaline from fixed alkali may be regarded as a matter of no consequence, so far as concerns the formation of concretions within the urinary passages, since the amorphous phosphate of lime which forms the bulk of the precipitate has scarcely any tendency to cohere into solid masses. Only the very rare calculi which consist entirely of this substance can have had their origin in such a state of the urine.

Nor is there any ground for supposing that visible precipitation of phosphates, whether spontaneous or as the result of heat, is an indication that these salts are being excreted in excess. To determine that, it would be necessary to make an exact quantitative analysis of the twenty-four hours' urine. Such analyses have been often made, but with the most meagre results, so far as their clinical value is concerned. There is therefore no antecedent probability in favour of the view, which at one time was entertained by good observers, that a "phosphatic" state of urine is dependent upon an undue disintegration or waste of the nervous tissues. And certainly direct evidence in support of such a view is altogether wanting.

Triple or ammonio-magnesian phosphate.—In ammoniacal urine the earthy

* This fallacy was unknown to Prout and to Bright, and nearly led the latter to abandon his hypothesis of the connection of albuminuria with dropsy and disease of the kidneys. It was detected by Drs Barlow and Rees ('Guy's Hosp. Reports,' 1836, p. 401).

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phosphates are as insoluble as in that which is alkaline from carbonates of soda and potash, and white gravel is therefore precipitated.

The phosphate of lime ($Ca_3P_2O_8$), with a little of the corresponding magnesian salt, comes down in the form of amorphous granules, not as dumb-bells or crystals. But along with these salts are also thrown down brilliant crystals of another salt, the phosphate of magnesia and ammonia ($MgNH_4PO_4 + 6Aq$), formerly spoken of as "triple phosphate." The form of these crystals is that of a triangular prism with bevelled ends; but they are liable to a great many modifications by planing-off of their edges and angles, and sometimes their sides become hollowed out. Some of the prisms may be so short that their bevelled ends meet one another on one edge; they then look not unlike the octahedra of oxalate of lime. The addition of acetic acid at once distinguishes them; it dissolves all phosphatic deposits.

The precipitate of mixed phosphates (sometimes with lithates of soda and ammonia) does not carry down urinary pigment with it, like the amorphous lithates which occur in acid urine; and therefore preserves its white colour. Beside the deposit, there is often on the surface of the urine an iridescent film containing the same elements.

An important character of the mixed phosphates thrown down as the result of the ammoniacal fermentation is their liability to undergo concretion into a mortar-like mass; this forms a large part of many calculi, and it may also collect upon the end of a catheter left in the bladder, and upon the surface of the mucous membrane itself.

Cystine ($C_3H_5NSO_2$).*—In 1810 Wollaston discovered, in analysing a urinary calculus, that it was composed of a peculiar substance, to which he gave the name of "cystic oxide." Subsequently the same substance, which is now known as cystine, was found to occur as a light flocculent deposit from the urine. It looks very like a fawn-coloured sediment of amorphous urates, but with the microscope it is seen to be crystalline, consisting of hexagonal tablets, which Roberts describes as having an iridescent, mother-of-pearl lustre, and as being often chased on the surface by lines of secondary crystallisation; there may also be thick rosettes of great brilliancy.

Acetic acid throws down from urine exhibiting this deposit an additional quantity of cystine, which had remained dissolved; and sometimes, perhaps, might reveal its presence in urine which contained it in too small an amount to yield a spontaneous sediment. But the occurrence of cystine in any form is very rare.

Urine which deposits cystine is usually faintly acid, and is described as having "a honey-yellow colour, an oily appearance, and a peculiar sweet-briar odour." The deposit is instantly dissolved by caustic ammonia; when this evaporates the hexagonal crystals reappear, but mixed with them there may also be highly refracting prisms, lying singly or forming stars, which are never seen in the urinary sediment.

The formula for cystine corresponds with sulphamido-pyruvic acid, pyro-racemic or pyruvic acid being derived from lactic acid. From a chemical point of view, the most remarkable fact about it is the large amount of sulphur that it contains, nearly 26 per cent. of its weight. When urine

* Not seven atoms of hydrogen, as usually given (Dewar and Gamgee, 'Journ. Anat. and Phys.,' vol. v).

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holding cystine in solution decomposes, it evolves sulphuretted hydrogen, so that it blackens the vessel in which it stands by its action on the lead in the glass. It is also said that the silver in the pockets of persons passing cystine in the urine may be similarly discoloured.*

The absolute weight of the cystine excreted daily is probably never considerable; for it is a light, though comparatively bulky, deposit.

It is a curious circumstance that cystine has several times been observed in the urine of persons related to one another as brothers or sisters. It is more often seen in males than females, and in children or young adults than in those who are older, but Roberts met with it in a patient aged fifty-seven. No condition of general ill-health is associated with the formation of this substance, and the only risk that appears to attach to it is that of the development of concretions or calculi. Some patients go on voiding it for many years continuously; in some cases it disappears from the urine for a time and then returns; ultimately it may cease altogether.

The treatment that has been advised is the administration of nitro-hydrochloric acid, or tincture of perchloride of iron, but it is doubtful whether either is of use.

Xanthin, which appears as a very rare form of calculus, has not yet been certainly observed as a urinary deposit (p. 511, *note*).

Sulphate of lime ($CaSO_4 + 2Aq$).—Considering that both sulphuric acid and lime are normal constituents of the urine, it is remarkable that the not very soluble compound which they form is so rarely deposited. As a matter of fact it has only been recognised in two instances, first by Valentiner ('Ctrlblatt.,' 1863) and afterwards by Fürbringer ('Deutsch. Arch.,' xx). It formed a bulky white sediment consisting of long needles and prisms with oblique ends, arranged in sheaves and rosettes. Valentiner's patient was an anæmic boy, Fürbringer's a wasted man affected with paraplegia. The conditions required for the production of such a precipitate appear to involve something beyond the mere presence of a moderate excess of sulphuric acid and of lime in scanty urine, for in these respects there was no change in Fürbringer's case, when after about three weeks it gradually ceased to appear; and as the urine was highly acid he was inclined to think that there was a deficiency of the alkaline bases. It is worthy of remark in this connection that calcic sulphate is more soluble in cold than in hot water.

Ammoniacal Urine.—As is well known, urine left exposed to the air soon undergoes a change which renders it turbid and offensive to the smell, and which may be regarded as putrefactive. This change takes place earlier in hot than in cold weather. It is accelerated by the presence of pus or blood or mucus, and still more by admixture with even the smallest quantity of urine already putrid. It occurs much less rapidly in urine which is concentrated and highly acid than in that which is pale and watery and of a faintly acid, or neutral, or alkaline reaction.

The same ammoniacal decomposition may take place within the urinary

* The composition of cystine forms an obvious point of resemblance between it and taurine, one of the elements of the bile. The amount of sulphates appears not to be much less, if at all, in urine containing cystine than in that which is healthy; but whether there is any change in the quantity of the organic sulphuretted compounds that exist in healthy urine seems at present to be unknown.

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passages while the urine is still unpassed, and may become a source of severe inflammation.

Decomposed urine may be recognised by its penetrating fœtid odour, an odour which is unfairly termed urinous (since it is quite unlike that of healthy urine), and the immediate cause of which has not been exactly ascertained. It also gives off carbonate of ammonia, the pungency of which may irritate not only the nose but the conjunctivæ, if the vessel containing the urine be held close to the face. This carbonate of ammonia arises from the decomposition of the urea, thus: $CH_4N_2O + 2H_2O = (NH_4)_2CO_3$. It gives to the urine an alkaline reaction, consequently the change which produces it is called the alkaline or ammoniacal fermentation. That the alkaline reaction is due to this cause, and not (as in other cases) to the presence of fixed alkali, can be easily determined. A piece of reddened litmus paper, or of turmeric paper, may be suspended in the mouth of a vessel over the urine but without touching it, when it will be found slowly to change its colour. Or the paper, after being dipped in the urine, may be left exposed to the air to dry, in which case the change in its colour that at first occurred will presently disappear. It must be remembered, however, that urine which was originally alkaline from fixed alkali may afterwards become putrid: in that case it will change the colour of paper suspended over it, but nevertheless paper dipped in it will continue to show an alkaline reaction after being dried.

Another character of urine that has undergone this change is its turbidity, a condition which filtering will not remove. The microscope shows that its cause is the presence of innumerable minute plants, occurring singly, or two or more in chains, or in a group with mucus as zooglœa. It is by these microphytes—*Micrococcus ureæ*—that the ammoniacal fermentation is brought about, probably immediately by a ferment which they secrete, just as alcoholic fermentation is brought about by the yeast plant, *Torula cerevisiæ*.*

Urine placed in perfectly clean vessels, and guarded from the approach of organisms from without, can be kept free from this change for an indefinite length of time; and the same is the case in the body. Some observers have maintained that mere stagnation of urine in the bladder, or in the pelvis of a kidney, suffices to render it putrid, especially if it contains mucus or cast-off epithelium; but that this is not the case has been proved by experiments on animals, for when the neck of the bladder or ureter has been ligatured the urine has remained acid.

In too many cases the surgeon has introduced the microphytes into the bladder by using catheters which had not been cleaned with the scrupulous care necessary to render them "antiseptic." This danger was pointed out by Traube in 1864. The case which drew his attention to it was that of a man who had apparently had a distended bladder for two years, but whose urine was clear and acid when it was first drawn off by a catheter, whereas the next day it was turbid, and within six days it became alkaline, ammoniacal, and slightly fœtid.

It is, however, perfectly true that stagnation of urine in the bladder favours its decomposition. Cohnheim says that fluid containing bacteria may actually be injected into the bladder of a healthy dog without ill effects,

* It is not certain whether the *Micrococcus ureæ* of Pasteur and Cohn and the bacterium of Leube are identical with the micrococcus cultivated by Dr Wm. Robert Smith and described in the 'Quart. Journ. of Micr. Sci.,' January, 1887, p. 371.

See also a note by Mr A. S. Lea in the 'Physiological Journal' in 1885, vol. xi, p. 10.

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because they are all expelled the next time that the animal micturates. And in men, when this change has once occurred in the urine, nothing tends so much to keep it up as an inability on the part of the bladder to empty itself completely; so that some of the putrid fluid is always left behind to induce the same process in that which is afterwards secreted. It is a notorious fact, too, that formerly, when catheterism was constantly practised without any precautions against the introduction of septic matters, the urine seldom became ammoniacal unless either the bladder was paralysed or the urethra in some way obstructed.

In some cases the putrefactive change takes place in the urine when no instrument has been passed. An abscess may have opened into some part of the urinary passages, and brought bacteria with the pus. Or bacteria may have found their way along the urethra from outside, possibly through a layer of mucus which may have been left upon the surface of the mucous membrane in the act of micturition, when the urine contains much mucus. Or possibly they may have come from the blood through the renal glomeruli.

One effect of the occurrence of ammoniacal fermentation, whether in the body or out of it, is at once to precipitate the earthy phosphates, chiefly tribasic calcic, and ammonio-magnesian phosphates, together with some magnesian phosphate (p. 426), and also occasionally lithates of soda and of ammonia. The lithate of soda appears as round or irregular masses, from which project spiny crystals, straight or curved, like the "morning star" of mediæval warfare; the lithate of ammonia as opaque, globular bodies, or very small, slender dumb-bells, which may lie across one another, or be united into a rosette.

The precipitate consisting of these various substances is usually white, but Roberts has known the urate of ammonia in putrid urine to possess a beautiful violet hue.

It is often of great consequence to determine whether the occurrence of the alkaline fermentation is limited to the urine which has already entered the bladder, or whether the bacteria which produce it have also made their way up into the ureters and into the renal pelves. Sometimes this may be made out by very carefully washing out the vesical cavity, and by drawing off a few minutes later the first few drops of urine that collect. If this is found to be acid the change takes place only in the bladder. How quickly the reaction may be reversed is well shown when there is extroversion of the bladder, so that the orifices of the ureters are exposed to view. In a case of this kind Dr Rees, many years ago, found ('Croonian Lectures on Calculous Disease,' 1856) that the urine as it flowed out from the orifices possessed its natural amount of acidity; but when reddened litmus paper was applied a quarter of an inch lower down, so as to test the urine after it had passed over that short distance of mucous membrane, its blue colour was restored. This result, however, he attributed, not to ammoniacal fermentation (for which there was not sufficient time), but to the admixture of alkaline exudation from the reddened and inflamed surface.

In most cases, before putrefaction of the urine within the bladder begins, cystitis is already present; and even when that is not so its occurrence is not long delayed. Urine which has become ammoniacal acts as a powerful irritant, though it is not certain whether the inflammation is set up directly by the bacteria, or by the carbonate of ammonia that results from their presence. The exudation of a large quantity of pus follows. This,

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however, does not retain its usual characters, but is converted by the ammonia into a viscid, semi-transparent, mucoid material, which often blocks the urethra, and causes the patient severe pain. In a vessel it collects at the bottom, and coheres so firmly that when the urine is poured out it does not separate, but glides away as a gelatinous mass, which hangs from the vessel like a liquid rope. The cystitis is often intense, leading to "diphtheritic" infiltration and sloughing of large tracts of mucous membrane. The inflammatory process often extends along the ureters, and reaches the kidneys, as will be pointed out further on.

In this way ammoniacal fermentation of the urine becomes indirectly the cause of death. It is therefore a matter of the highest importance to prevent its occurrence, and to check it whenever it shows itself. The strictest antiseptic precautions should be observed whenever a catheter is introduced, and this should never be done without necessity, when there is likely to be subsequent difficulty in emptying the bladder. If the change has already taken place before the patient comes under observation the bladder should be washed out regularly with warm water containing borax and boracic acid. Roberts says that the urine may sometimes be restored to its natural condition by making the patient drink large quantities of fluid at regulated intervals, so as to keep the bladder washed out by the renal secretion. We have often kept the urine sweet and acid, or restored it after it had become ammoniacal, by giving salicylate of soda or benzoate of soda in ten or fifteen grain doses three times a day.

Ammoniacal urine without sepsis.—It would seem that an ammoniacal state of the urine, at the time when it is voided from the bladder, is not in itself a proof of the occurrence of putrefaction. At least Sir William Roberts says that he has observed this in two cases of advanced Bright's disease without there being any evidence of delay in evacuation, and without any part of the urinary passages being afterwards found inflamed at the autopsy; he does not mention whether bacteria were present. Graves gives two cases, in each of which the urine, although free from any smell of putrescence, contained a large quantity of carbonate of ammonia, even when it was passed soon after being secreted. In one (a case of dropsy) the fluid was tested for urea, which was found absent; the other was a case of fever.

Non-septic bacilluria.—Again, the presence of bacteria in the urine is not in itself proof that the ammoniacal fermentation has taken place, or is about to take place in it. Roberts, in the 'Transactions of the International Medical Congress,' held in London in 1881 (p. 157), has related several cases in which the urine when voided was opalescent, and full of micrococci and moving rods. In two of the cases it had a disagreeable odour. But it was acid in reaction, and showed no greater (and perhaps less) tendency to decomposition than healthy urine. Moreover, in the course of about twenty-four hours the bacteria sank slowly to the bottom, leaving the liquid itself clear, and it remained in that condition and retained its acidity for several days afterwards. The organisms seemed to be incapable of multiplying in the urine, even when it was kept at blood-heat. Roberts was therefore inclined to think that their original seat of development was in some part of the mucous membrane of the urinary tract. Some of the patients had had catheters introduced at a former period of their lives; in others this had not been the case. Two only were women, and one of them had no symptoms beyond a slightly increased frequency of micturition, especially

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at night. The others were men, and they suffered from scalding pain in passing water, with frequent desire to micturate, symptoms which in one instance had lasted thirteen, and in another seven or eight years. In one case there were severe intermittent attacks, as of acute cystitis. The administration of salicylate of soda, in doses of twenty to thirty grains twice or thrice daily, usually proved effectual; the urine became free from bacteria within a few days, and all the symptoms disappeared.

A similar case occurred in the clinical ward of Guy's Hospital a few years ago. A boy who was under the writer's care for another disease, without any symptoms of renal or vesical disorder, passed urine which contained rod-shaped bacteria, when examined immediately after it was voided. The urine was not ammoniacal, and in all other respects was normal.

Sarcinæ.—Several observers have recognised the presence of sarcinæ in the urine, sometimes in sufficient numbers to form a greyish-white amorphous deposit. They resemble those derived from the stomach, except that they are smaller. They may occur in either acid or alkaline urine. The patient is generally troubled with vesical or renal symptoms, but in some instances these have been probably attributable, not so much to the growth of the sarcinæ as to some concurrent disease, such as stricture or enlargement of the prostate. In a case recorded by Munk the sarcinæ were abundant in the summer, but almost wholly disappeared in the winter, notwithstanding that the patient, being paraplegic, was bedridden. Dr Bateman, of Norwich (cited by Dr Beale), met with a case in which the urine on four separate occasions contained sarcinæ for a few days at a time.

Kyestine (from κύησις, gestation) is a name given to a product of decomposition which shows itself occasionally in urine on the second or third day. It was first observed in cases of pregnancy, and was supposed to be peculiar to that condition; but this is now known not to be the case (Dr J. B. Hicks, 'Guy's Hosp. Reports,' 1861). It forms a thick scum upon the surface, and consists of the ordinary products of ammoniacal decomposition, crystals of ammonio-magnesian phosphate, vibrios, and bacteria.

In some cases urine, with kyestine in large quantity, is said to resemble chyluria, for the urine is then "milky" throughout; but no albumen or fat is present.

HÆMOGLOBINURIA.—The urine may be reddened by the colouring matter of the blood, and yet no red discs be found in it. This condition must be carefully distinguished from hæmaturia, where blood-corpuscles are present; which will be considered in the chapter on calculi, tubercle, and cancer of the kidneys, for these diseases are its most frequent cause.

Characters.—Urine containing hæmoglobin varies in its tint in different cases. It is sometimes of a pinkish hue, usually dark red or chocolate-brown, and occasionally almost black. It is often compared to strong tea or to porter. It may be clear and transparent when passed, but as it cools it is apt to throw down a thick sediment of lithates having a chocolate colour. Or the hæmoglobin itself may form a more or less abundant precipitate. With the microscope there is often only a granular *débris* to be seen, but in some cases a few shrivelled or altered blood-discs are visible. It must be remembered that the hæmoglobin may appear in the form of rounded reddish-yellow drops, looking not unlike red discs, but variable in size and

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arranged in rows like the beads in a necklace. It may also be moulded into casts of the renal tubules, which have an opaque granular appearance and a reddish-brown colour; but with few, if any, exceptions, there is no true hyaline "cast" of fibrin enclosing the pigment. Sometimes, but very rarely, it assumes the form of crystals. Oxalate of lime crystals are not infrequently met with, and their presence is in certain cases so constant that it seems likely to be more than accidental (see a paper by Mr Fullerton in the 'Lancet' for October, 1890, p. 709).

When urine containing hæmoglobin is heated, it yields a coagulum which has a brownish-red colour, and is described as differing from the ordinary coagulum of albuminuria in floating upon the surface of the fluid, instead of subsiding quickly to the bottom. This coagulum is said to be formed solely by the proteid of hæmoglobin itself, no serum-albumen being present. Cohnheim speaks of a case in which Roux found that there was enough iron in the urine to correspond, according to that view, with all the albumen.

If there is any doubt as to the nature of the colouring matter, the chemical tests of hæmoglobin are applied; or with the spectroscope a characteristic appearance is obtained. This consists in the presence, not only of the well-known absorption bands of oxyhæmoglobin in the yellow and in the green between the solar lines D and E, but also of a third broad band in the red between the lines C and D, but nearer to C. This third band belongs to a modification of hæmoglobin, called methæmoglobin, generally believed to be intermediate in oxidation between oxyhæmoglobin and reduced hæmoglobin.

There is no doubt that hæmoglobin is set free in the blood before its excretion by the kidneys, inasmuch as Marchand has shown that in poisoning by chlorate of potass (which we shall find to be one of the causes of hæmoglobinuria) the blood itself yields a spectrum showing the band between C and D. Moreover, blood has been taken by cupping from patients with hæmoglobinuria, and after clotting, the serum has been seen to be red instead of yellow.

Morbid anatomy.—In fatal cases of hæmoglobinuria the kidneys are found of a deep chocolate colour, with radiating striæ of a still darker tint. With the microscope the renal tubes are found to be completely plugged with masses of hæmoglobin. These have also been shown by Dr Bridges Adams to be present in the Malpighian capsules—a point of some importance as showing that the hæmoglobin is excreted through the glomeruli rather than the epithelium of the tubes. In one instance Hofmeier further noticed that the spleen had a peculiar reddish-brown appearance on section, and that the medulla of the femur in its upper half was brown. Ecchymoses have also been present in the mucous membrane of the stomach and intestines.

Pathology.—Hæmoglobinuria is probably always the result of a disintegration of some of the red discs within the circulating fluid, or at least of the escape of their hæmoglobin from their "stroma." It is not due to any primary alteration in the structure, nor even to perversion of the functions of the kidneys. The causes of the change in the blood are various, so far as we know.

Transfusion of blood from one kind of animal to another—as when, for example, lamb's blood is thrown into the veins of a dog—will produce hæmoglobinuria in the dog; the explanation being apparently that the foreign red discs are broken up by the blood-serum of the animal into which they are introduced.

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Cohnheim states that hæmoglobinuria is a common symptom after extensive burns, if they are not too rapidly fatal, though this symptom has attracted less notice from surgeons than from Lichtheim and other pathologists who have made experiments upon animals.

Hæmoglobinuria is said to have been observed in Germany as a result of heat-stroke. It has been recorded by Immermann as a complication of enteric fever during a relapse ('Deutsches Arch.,' xii), by Heubner in a case of scarlet fever on the twentieth day (*ibid.*, xxiii), and by Stolnikow in one of ague ('Petersburg. med. Woch.,' 1880). Hæmoglobinuria in its paroxysmal form, is frequently the result of exposure to cold; it probably has relations to malaria in many cases, and to other forms of vaso-motor disturbance in many more. It is also caused by various poisons.

Toxic form.—Among the ingesta which have been known to cause it, either in animals or in man, may be mentioned hydrochloric acid, sulphuric acid, chlorate of potass, nitro-benzol, naphthol, and carbolic acid. Eitner has recorded a fatal case ('Berl. klin. Woch.,' 1880) in which it was set up by the inunction of pyrogallic acid in a patient affected with severe psoriasis.

In several instances it has been due to the entrance of *arseniuretted hydrogen* gas into the air-passages. Eitner recorded four cases due to this poison, in which the sufferers were a professor of physics and three of his students. The professor had two attacks of hæmoglobinuria, separated by an interval of some days, and resembling the paroxysmal form of the affection in all respects except perhaps in having a rather longer duration. It was not until other persons were found to have suffered from like symptoms that their real nature was suspected. He and his pupils had been repeating Tyndall's experiment of inhaling hydrogen gas, for the purpose of showing that it alters the pitch of the voice; and the zinc used in generating the hydrogen was impregnated with arsenic.

From a clinical point of view, *chlorate of potass* far surpasses in importance all the other toxic causes of the affection. Hofmeier collected ('Deutsche med. Woch.,' 1880) no fewer than twenty-seven cases, all but four of which proved fatal. Some of the patients took the salt by mistake for sulphate of soda or for some other saline aperient. But for most of them the chlorate of potash was prescribed, and the mischief arose either from the dose being too large or too frequently repeated, or else from their swallowing large quantities of a solution intended only as a gargle. In children it would appear that from one to two drachms in the course of twenty-four hours is a dangerous quantity; in adults perhaps from two or three drachms upwards. In many instances the disease for which the chlorate of potass was ordered has been diphtheria, and one cannot help fearing that in many other cases the salt may have produced like effects without their real cause having been suspected, the state of the urine having been attributed to diphtheritic nephritis, and the severe constitutional symptoms regarded as indicative of the "collapse" which sometimes proves so rapidly fatal in diphtheria.

The symptoms of toxic hæmoglobinuria differ in intensity, but seem always to belong to a common type. In the most marked and severe cases the patient is seized with a rigor; vomiting and diarrhœa then set in; he becomes collapsed and cyanosed, falls into a state of stupor, and so dies.

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Infantile hæmoglobinuria.—Spontaneous hæmoglobinuria occasionally occurs as an epidemic among infants. Winckel has related ('Deutsche med. Woch.,' 1879) a most remarkable outbreak, which occurred in 1879, in the lying-in hospital at Dresden, where, between March 20th and April 29th, twenty-four newly born infants were attacked by it, of whom twenty-three died. The symptoms were in every instance similar; the child, generally about the fourth day after its birth, became suddenly cyanosed and collapsed, with cold extremities; and it usually succumbed within thirty-six hours. There was seldom any diarrhœa or vomiting, but the skin had in many cases an icteric tinge. The respiration and the pulse were very rapid; the temperature in the rectum was scarcely raised. At many points the superficial veins became visible as dark lines; when they were incised no blood escaped, but on pressure a brownish-black fluid of syrupy consistence could be squeezed out. The urine was of a brown colour, and exhibited the usual characters of hæmoglobinuria. Death was preceded by convulsions. On *post-mortem* examination the kidneys were dark brown, and their papillæ showed plugs of hæmoglobin. The chief other morbid appearance was an enlargement of the mesenteric lymph-glands and of the spleen, the latter being tough and of a brownish-red colour. Every effort was made to discover the cause of this affection, but without success. Winckel described it as *Cyanosis infantilis icterica perniciosa cum hæmoglobinuriâ.* There can be little doubt that its essential character was an intense and rapid disintegration of corpuscles within the blood-vessels. But why this should have occurred in a number of young infants in succession—themselves apparently healthy at birth, the offspring of healthy mothers, and in a hospital where no like disease had been observed before—remains a mystery.

PAROXYSMAL HÆMOGLOBINURIA.*—In marked contrast with this fatal infantile form of hæmoglobinuria is one to which adults are also liable, and which recurs over and over again without danger to life or severe disturbance of health.

The first account of this remarkable disorder as distinct from hæmaturia was (as Dr Wickham Legg has shown in a valuable paper in the tenth volume of the 'St Barth. Hosp. Rep.') an article by Dressler, published in 'Virchow's Archiv' as long ago as 1854, under the title "Intermittent Albuminuria and Chromaturia." In this country Dr George Harley, Dr Dickinson, and Dr Hassall each published typical cases in the same year (1865). Dr Harley's single and Dr Dickinson's four cases appeared in the forty-second volume of the 'Med.-Chir Trans.;' Dr Hassall's in the 'Lancet' (ii, 368). A case was described by Sir William Gull in the 'Guy's Hospital Reports' for 1866 as "intermittent hæmatinuria," by which name it has been since commonly known; but the best name for it appears to be "paroxysmal hæmoglobinuria," used by Dr Pavy in 1866 and 1868. Greenhow, Druitt, and many others at home and abroad have since written on the subject. Lichtheim published three cases in 1878 (No. 134 in 'Volkmann's Sammlung') under the title "periodical hæmoglobinuria."

Ætiology.—In some few instances no cause can be discovered. Two such cases occurred in Guy's Hospital during the height of summer, in June, 1876, and in August, 1880. In neither case were there any subjective

* *Syn.*—Intermittent chromaturia (Dressler)—Winter hæmaturia—Intermittent hæmaturia—Paroxysmal hæmaturia—Hæmatinuria—Methæmo-globinuria.

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sensations, except pain in the loins, associated with the discharge of urine, which was almost black with hæmoglobin.

In the great majority of cases, however, the affection is due to one particular exciting cause, namely, *cold*. Sometimes the degree of cold that causes an attack (especially the first attack) is excessive, or, at any rate, far beyond that to which the individual is accustomed. A London physician, a friend of the author, observed hæmoglobinuria for the first time when he was one day fishing in Scotland in a biting wind, and his next seizure occurred some months later while he was skating without a great coat. But he subsequently became liable to the affection when the provocation appeared inadequate to produce such a result, as, for example, after standing four or five minutes at a railway station on a foggy morning, or riding two or three miles in a hansom cab. In some instances it is stated that all but the early attacks have been independent of cold. Thus a patient of Sir William Roberts said that he was just as bad in the summer as in the winter. As a rule, however, the affection ceases entirely during the warm part of the year, returning in the cold season, perhaps for many years in succession. That this is entirely a question of temperature was shown by Rosenbach, who, by means of a cold foot-bath, brought on an attack during the summer in a person liable to the disease. Roberts relates two cases, in each of which there were as many as three daily attacks. But commonly they recur at much longer intervals and quite irregularly.

They are most apt to take place in the *morning*, when the temperature of the body is lower than in the afternoon and the evening. It is especially when the patient is exposed to cold directly after breakfast, before the meal has been digested, that hæmoglobinuria is apt to show itself; and a cup of hot beef-tea on first waking may prove an effectual preventive. An outbreak never comes on during the night, when the patient is warm in bed. Bodily or mental exhaustion, as from sexual intercourse, or from study late at night, favours the occurrence of an attack. A like influence appears to be exerted by the free use of wine at dinner, probably from the recoil after its stimulant action, as well as from its relaxing the cutaneous vessels. Hæmoglobinuria is often seen in persons whose fingers are apt to "go dead" —*digiti mortui*—cases closely allied to Raynaud's disease (p. 439).

The previous occurrence of *ague* has been noted in some instances, and it seems probable that this may sometimes be a more or less direct cause of hæmoglobinuria, as it is of splenic leuchæmia. The writer had an instance of an elderly man with ague and enlarged spleen, who passed hæmoglobin in his urine during the cold stage of each paroxysm.

Syphilis was present in a case observed by Ehrlich; and a patient who was under the author's care in the clinical ward of Guy's Hospital in 1882, and who did not improve under the usual remedies, was subsequently cured by Dr Moxon with mercurial treatment. In that instance the spleen was much enlarged. Dr Legg met with a similar case.

Fleischer has recorded ('Berlin. klin. Woch.,' 1881) an instance in which a soldier was attacked every time he marched for an hour or two, although no other kinds of bodily exercise had a similar effect.

The disease is much more common in men than in women. It is most apt to occur in young adults, and may continue up to the age of forty or fifty; but Dressler's patient was a boy between ten and eleven and Dr Greenhow's a child only two years od, while Dr Druitt was above fifty when he was first attacked.

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Seven cases of this remarkable disorder have been under the present writer's care. They all occurred in men—the youngest fourteen years old, the eldest above sixty, and the remainder between nineteen and thirty-three. Two were pretty clearly of malarial origin, associated in one patient with dysentery during the Zulu campaign, and they were both benefited or cured with quinine; two were complicated by gangrene of the ears or fingers and toes. One case occurred in a schoolboy three times at intervals, the first after bathing, and the third attack was marked by high fever.

The *symptoms* of paroxysmal hæmoglobinuria vary in different cases. One of the most constant is a feeling of languor and weariness, with a disposition to yawn again and again. With this there may be a feeling of chilliness, so that the patient is inclined to huddle up by the fire. The fingers and the toes—some or all of them—may turn white and dead. A graphic account of the disease was given by the late Dr Druitt in the 'Medical Times and Gazette' for 1873; it is now no secret that he was himself the patient. He describes his palms and soles as becoming "cold, wet, blue, and cramped, like those of a cholera patient." At other times he felt numbness of the right foot and of the left hand, without coldness; or his nose, or some part of his cheek, would become first pale, then red, then purplish, and at last almost black. In several instances the ears not only turned livid during the seizures, but failed to regain their natural appearance afterwards, so that a reddish-brown eschar formed along the edge of the helix, leading to a permanent loss of its substance.

While these symptoms are developing themselves, and until they have entirely passed off and been succeeded by a feeling of warmth and comfort over the whole body, there may be nothing to show that the secretion of urine is other than normal. There is often no desire to micturate. In a case which the author had many opportunities of watching, it was sometimes only after the lapse of several hours that any water was passed, when the patient has almost forgotten that he had been chilly; so that he was quite surprised to find it like chocolate or porter. But in other cases the bladder is irritable, and Dr Druitt says that on some occasions he suffered great pain in the bladder, and was obliged every half hour to void urine, which was of a bright scarlet colour. Sometimes, but not always, there is pain in the back, or radiating across the abdomen or down the thighs. Retraction of the testes has also been recorded.

The urine, beside methæmoglobin, often contains a few red discs, more abundant leucocytes, blood-casts, and sometimes crystals of calcic oxalate.

The patient's temperature during a seizure of hæmoglobinuria is, as a rule, normal. Dr Druitt says that during the course of his illness (which had then lasted six years) he had severe attacks of remittent pyrexia, in which the thermometer would rise to about 103° in the evening, and fall to 100° in the morning; during these periods jaundice was always present, but the urine never contained any blood-colouring matter. His pulse generally fell to 55 or 50 when the hæmoglobinuria was about to occur. In one of the writer's cases the patient, a robust youth of nineteen, had pyrexia reaching 103° on one, and exceeding 104° on another occasion, with a pulse of 90 to 100 during the attacks.*

One of the most remarkable features of the disease is the rapidity with which, after a seizure, the urine regains its normal characters. The patient

* The second attack had been recorded by Dr Penny, Medical Officer to Marlborough College, and was also accompanied by pyrexia.

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may once or twice, or oftener, have voided a fluid like porter; and that which he passes an hour or two later is perfectly clear and pale. Even in Roberts's two cases, in which there were two or three attacks in the course of a single day, colourless urine was secreted in the intervals. It is also curious how completely the appetite is unaffected; a patient who had a well-marked attack in the morning may at one o'clock eat a hearty dinner as usual, sometimes even before he has discharged from his bladder the dark urine formed during the attack.

Pathology.—It has now been ascertained that the starting-point of paroxysmal hæmoglobinuria (as we found in its other forms) is disintegration of a certain—probably a small—proportion of red discs in the circulating blood. Microscopical examination of the blood does not always show any marked change in it, as the writer and several other observers have found. Küssner, however, took blood with a cupping glass ('Deutsche med. Woch.,' 1879) from a patient on six occasions during a seizure, and each time found that the serum was of a ruby-red colour, whereas at other periods it had the normal yellowish appearance. A still more striking experiment has since been performed by Ehrlich (*ibid.*, 1881). Having under observation a woman who was liable to the disease, he bound an elastic ligature round one of her fingers, which he placed for a quarter of an hour in ice-cold water, and afterwards for the same length of time in tepid water. Such an experiment on a healthy person produces no change in the blood, but in the patient in question it caused the red discs to break down in considerable numbers. When a drop of blood from the finger was placed in a capillary tube and allowed to coagulate there, the serum was distinctly seen to be reddened; and under the microscope the blood was found to contain "phantom discs"—stromata or *œcoids* of discs that had more or less completely lost their hæmoglobin—though there were also many normal corpuscles, as well as pœcilocytes and microcytes. The disease therefore seems to originate in an undue sensitiveness to cold on the part of certain of the red blood-discs.

In some patients, however, there may be observed a symptom which at first sight seems inconsistent with this view of the disease. It is that attacks of chilliness, which fall short of the degree of severity necessary to bring about an excretion of hæmoglobin, render the urine albuminous. This is a fact which we have verified on many occasions, the patients being perfectly free at all other times from any indication of Bright's disease. It was markedly the case in the youth of nineteen whose case was mentioned above; and indeed was observed in the earliest case published, that by Dressler in 1854.

Probably the explanation is that when the disintegration of red discs occurs only to a moderate extent all the hæmoglobin which is set free splits up into globulin and hæmatin. We may then suppose the albumen (or globulin) to be incapable of remaining as a constituent of the serum, and to be excreted as such by the kidneys. Indeed, it is certain that even in ordinary cases of hæmoglobinuria a part of the hæmoglobin is decomposed, so as to form a pigment resembling bilirubin, if not identical with it. The proof of this is the sallow bilious appearance which is invariably presented by patients who frequently suffer from the disease, and which is sometimes apparent during or after a single severe attack.

In not a few cases hæmoglobinuria follows or is replaced by hæmaturia, and occasionally both conditions are accompanied by the symptoms of acute

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nephritis, as in a remarkable case recorded by Dr Donald Hood in the 'Lancet' (Oct. 4, 1890).

According to Dr Noel Paton's evidence the presence of hæmoglobinuria is in some cases accompanied by very large increase in the excretion of urea as well as by increased secretion of bile.

Prognosis.—Paroxysmal hæmoglobinuria has never been known to destroy life, nor does it appear to entail very serious consequences, though one can hardly doubt that if the attacks were allowed to recur frequently, and for a great length of time, the patient's health would ultimately break down. The author once saw chronic Bright's disease in a man who had previously suffered from hæmoglobinuria, and had lost the margins of his ears from it. The attacks may continue for many years in succession, or they may cease after a few weeks or months. Dr Druitt had suffered for six years when he published the account of his case; in another recorded instance the disease ran on for eleven years. In twenty cases collected by Roberts there was no fatal issue, and twelve were well when their cases were published.

Treatment.—During an attack the essential requisite is warmth. If necessary the patient may be put to bed. Probably the best thing to drink is a basin of hot soup. When there is severe pain in the back, it may be relieved by the application of mustard, or by dry cupping, or (as Dr Druitt found) by the internal administration of extract of hyoscyamus.

But the important object is to prevent the recurrence of the disease. The utmost care should be taken to avoid exposure to cold in the early part of the day, and in such circumstances as may be shown by experience to be injurious in the particular case under observation. The meals should be so arranged that there may be food in the stomach at the time when a cold journey is to be made, or when any unaccustomed task is to be gone through that may cause nervousness and anxiety. The clothing must be warm; flannel underclothing, fur-lined boots and gloves, wash-leather waistcoats, and cork soles are all useful.

The question of removal to a warm climate during the winter should in severe cases be seriously considered; Dr Druitt himself went to Madras, where he escaped the disease almost entirely. During the summer a bracing air is probably advisable.

Throughout the twenty-four hours the habits should be so arranged that the body shall become as little exhausted as possible. The patient should not spend his evenings in heated rooms, nor should he devote them to arduous mental work, but should go to bed early. He should have nutritious food in the latter part of the day, but little or no alcohol; for its effect is an immediate stimulation that rapidly passes off.

The one medicine that seems to have a marked effect in warding off the attacks is *quinine*. It must be given in full doses. It sometimes proves perfectly successful, so that the patient becomes again able to live his usual life without fear of his complaint returning. But in severe cases, like that of Dr Druitt, no permanent benefit can be obtained by it, even when it is used in such quantities as to cause singing in the ears and other disagreeable effects. Salicine, the tincture of iron, and arsenic, may each be of service in their turn. Chloride of ammonium is said to have done good in one instance. If syphilis is present, iodide of potassium or the bichloride of mercury will sometimes work wonders.

Raynaud's disease.—There can be little doubt that paroxysmal hæmo-

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globinuria in its most characteristic form, is closely related to the local asphyxia with symmetrical gangrene of the extremities, which was described by the late Dr Maurice Raynaud in 1862, and which, in its most severe forms, is known by his name.*

The age of the patients, the coldness of the extremities, and the mortification of the ears, or fingers, or toes, are very suggestive of this relation; and in typical cases of Raynaud's disease hæmaturia or hæmatinuria has been frequently observed. Curiously enough, in the twenty-five cases on which his monograph is based (five of his own and twenty collected from previous writers), no mention is made of the state of the urine. There are, however, points of difference: of Raynaud's twenty-five cases, twenty occurred in women and only five in men. Some of the most severe cases of hæmoglobinuria show no tendency to gangrene, and the patient's circulation is perfect in the intervals between the attacks. On the other hand, some of Raynaud's cases are not accompanied by any change in the urine, or the change is to true hæmaturia. At present it is perhaps best to keep together the cases of hæmoglobinuria (and even of hæmaturia) which are decidedly paroxysmal and unaccompanied by gangrene, and to reserve the title of Raynaud's disease for the continuous and severe cases described by him.

* "De l'asphyxie locale et de la gangrène symmétrique des extrémitiés" ('Thése de Paris,' 1862). See a case of Prof. Billroth's in the 'Wiener med. Wochenschft.,' 1878, reported in the 'Lond. Med. Record' of that year, p. 343; also Dr Southey's papers in 'St. Bartholomew's Hospital Reports,' vol. xvi, and in the Clinical Society's 'Transactions' for 1883; Dr Thos. Barlow's communication in the same volume, with the sequel to his cases in 1885 (vol. xviii, p. 307), and Dr T. C. Fox's two cases (*ibid.*, p. 300). Weiss, "Über Symmetrische Gangrän."

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BRIGHT'S DISEASE

"Crescit indulgens sibi dirus hydrops
Nec sitim pellit nisi causa morbi
Fugerit venis et aquosus albo
Corpore languor."
HOR., *Carm.* II, ii.

History—The three chief forms—Additional varieties—Common characters of the disease—Albuminuria: its detection and estimation, pathology, and significance—Casts: their structure, varieties, and meaning—Dropsy: its pathology, its primary and secondary form—Serous and other inflammations—Retinitis and retinal hæmorrhage—Anæmia—Changes in the heart and arteries: the renal pulse, hæmorrhage—Uræmia: eclampsia, coma, amaurosis, &c.; vomiting; dyspnœa; theory of uræmia.

Parenchymatous Nephritis, acute and chronic—causes—anatomy— three stages—symptoms—event and prognosis.

Lardaceous Nephritis—chemistry—anatomy—symptoms—diagnosis—causes.

Cirrhosis of the Kidney—pathology—causes—anatomy—symptoms and clinical aspects—Consecutive renal cirrhosis—its relation to local disease of the bladder, urethra, and uterus—Hypertrophic cystic degeneration.

Relation of the several forms described—Distribution—General prognosis and treatment of Bright's disease.

DROPSY, as anasarca and ascites, was well known to the ancients;* it was associated with the effects of drink and afterwards with disease of the heart; but renal, as distinct from hepatic and cardiac, dropsy was only recognised about ten years after Laennec's great discovery, although its characters must often have been noted, as in the quotation at the head of this chapter.

In 1827 Dr Richard Bright published, in his 'Reports of Medical Cases,' the fact that in many cases of dropsy there are well-marked lesions of the kidneys, and that the urine is albuminous. His discovery was of the connection between the clinical symptoms of dropsy and albuminuria and the anatomical fact of renal disease. That dropsy is often attended with the presence of serum in the urine had been pointed out a short time before by Dr Wells of St Thomas's Hospital (the author of the 'Essay on Dew'), by Dr Blackall of Exeter, and Dr Osborne of Dublin; in the previous century it had been noticed by Cotugno and by Cruikshank. But notwithstanding that Wells and Blackall each made autopsies in which they found the kidneys "remarkably hard," they both regarded the presence of disease in these organs as accidental. Blackall's hypothesis was that serum was excreted by the kidneys because it was vitiated, possibly from having already formed part of the dropsical fluid, and having been thence reabsorbed

* See the quotation, p. 305, from Aretæus, who flourished in the first century. He there distinguishes *tympanias*, or "windy dropsy," from *ascites*; and a general swelling of the body with white, thick, cold phlegm (*phlegmatias*), from *anasarca* (ὕδρωψ ἀνὰ σάρκα), which is defined as a liquefaction of the flesh into a thin watery humour ('Morb. Chron.,' II, i). Celsus (probably contemporary) writes much to the same effect, that there are three kinds of dropsy: "primum *tympaniten*, secundum *leucophlegmasiam* vel *ὑποσαρκα* (not anasarca), tertium *ἀσκίτην* Græci nominaverunt" (lib. iii, cap. 21).

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into the blood. To Bright belongs the full credit of first showing the real and constant relation of disease of the kidneys both to dropsy and to albuminuria, and his name is therefore justly associated with it throughout the world.

After the publication of his first cases and drawings, two wards at Guy's Hospital were devoted to further investigations into the new disease, and Dr Bright, with the help of Dr Barlow and Dr Owen Rees, made the further observations which were printed and illustrated in the first volume of the 'Guy's Hospital Reports,' and in the fifth of the second series.

Its divisions.—A difficulty which has stood in the way of all subsequent writers is that the lesions of the kidneys described by Bright vary greatly. He himself and his fellow workers admitted three forms of the affection, leaving it an open question whether or not they should be regarded as separate diseases.

The three anatomical types of morbus Brightii recognised by Barlow, Rees, and Wilks, were the *large red kidney*, the *large white kidney*, and the *small red kidney*.

Rayer, who first followed Bright's footsteps in France, divided what he called *néphrite albumineuse* into a much greater number of varieties. In 1851, Professor Frerichs, then of Breslau, propounded the doctrine that the diverse appearances presented by the kidneys belong to the successive stages of a single morbid process. This view, however, was refuted two years later by Dr Wilks, who showed in a paper in the 'Guy's Hospital Reports' (second series, vol. viii) that, under the name of Bright's disease, there are included at least two independent affections differing in their causes, in their mode of onset, their symptoms, and their course—"the large white kidney with considerable dropsy," and "the hard contracted kidney, often destitute of symptoms"—and added a summary of twenty-three cases of the former, and thirty-three of the latter affection.

Wilks reasoned that since anasarca is particularly associated with the "large white kidney," one ought, if that were an early stage of the "small red kidney," to obtain a definite history of there having been at a former time an attack of dropsy in each case in which the small red kidney was found after death. Frerichs, however, had not brought forward a single instance in which there had been this sequence of events, nor were any furnished by Wilks's own experience. The statement that the large white kidney does not pass on into the small red kidney may perhaps be put too absolutely; but in the main it is undoubtedly correct. Moreover, it is not uncommon to find in the bodies of those who have died of various diseases kidneys showing all the stages of the morbid process by which they gradually became shrunken; and none of these are marked by enlargement. As for the other points of distinction pointed out by Wilks, it may be briefly stated that, whereas the large white kidney often results from scarlet fever, or from exposure to cold, the small red kidney is not traceable to either of these causes, but in many cases to gout or to plumbism; that whereas the former occurs in children and young adults, the latter is rare under five-and-thirty, and is not frequent before the age of fifty; that the former is often abrupt in its onset and acute in its course, while the latter always begins insidiously, and is very slow in development; and lastly, that although either may be attended with changes in the heart and arteries, such changes are far less marked in the former than in the latter kind of Bright's disease, which often appears clinically under the mask of cardiac symptoms, or of apoplexy due to rupture of an artery in the brain.

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It would, of course, add to the force of these considerations if it could be shown that two distinct pathological processes are concerned in the production of the two forms of Bright's disease, or even that the same process attacked in the one form one anatomical element of the kidneys, in the other form another. But the pathological process is, in both cases, inflammation; and, in both, it always falls short of suppuration. Virchow, in his 'Cellular Pathology,' made the ground of distinction between the two forms that in the large white kidney the tubular epithelium, in the small red kidney the interstitial tissue, was mainly affected. Later observations, however, have shown that this distinction is far from being absolute; and therefore the two names proposed by Virchow, "parenchymatous nephritis" and "interstitial nephritis," can no longer be regarded as perfectly applicable. In both forms the tubes are affected; in both there is intertubular exudation; in both the cortex is the seat of the lesion: the distinctions are rather in the origin, course, and result than in the nature of the process, or even in its seat. Virchow's distinction is general, not absolute; the histological origin in both forms is obscure, and mixed cases occur. But it remains true that the tubular parenchyma of the cortex is swollen in the large smooth kidney, whereas in the small rough kidney it is contracted and atrophied as in other chronic interstitial inflammations.

Both clinically and pathologically we must therefore admit the fundamental unity of Bright's disease, together with the broad and important differences just stated between its two principal forms. As physicians, we are quite justified in regarding them as distinct affections when we find that they differ in their causes, in their symptoms, and in their clinical course. Some German writers describe separately "acute Bright's disease," "chronic Bright's disease," and "contracted or granular atrophy of the kidneys." Such a division, however, keeps apart cases that should be brought together, and it brings together cases that should be kept apart. An acute renal affection may be the result of scarlet fever, or of cold, or of pregnancy; but if it does not soon end either in recovery or in death, it becomes chronic, and may ultimately pass into a stage of contraction and of atrophy. All such cases should be held to belong to a single form of Bright's disease; and since an affection of the tubal epithelium is a more or less constant feature, we may call them *tubular*, or *parenchymatous nephritis*. On the other hand, Ernst Wagner's "contracted or granular atrophy" includes two sets of cases that are essentially distinct; one is the advanced stage of the affection just mentioned, the other is the originally chronic affection which produces the "small red kidney." It is to the latter process alone that the term *interstitial nephritis* should be applied, or its synonym, *cirrhosis of the kidney*.

These two chief forms of Bright's disease, however, are by no means the only ones. That in some cases the primary lesion is a *lardaceous degeneration* in the arterioles and in the glomeruli of the kidney has long been known, and of late it has become apparent that such cases are more numerous than was formerly suspected. It is true that the lardaceous affection always becomes complicated sooner or later with parenchymatous or with interstitial lesions, or with both. But inasmuch as its causes differ from those of Bright's disease in general, and as there are also differences in its symptoms and course, it is desirable to keep it apart.

There are two other subordinate forms: one is the affection commonly known as *cystic disease of the kidneys;* the other, which has more recently been recognised, is in its origin secondary to lesions of the renal pelvis or

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of the lower urinary passages, so that it may be termed "*consecutive Bright's disease.*" Both are varieties of chronic interstitial degeneration.

It might be thought that the recognition of so many independent forms of Bright's disease would render it advisable to drop the common designation. But, first, many important characters belong to them all alike. Secondly, in clinical practice one is not infrequently in doubt as to which form is present. Thirdly, we often meet with transitional forms:—the acute tubal nephritis is passing on to, but has not reached, the chronic stage of the large white kidney, as red hepatisation passes into grey; the granular atrophic organ is overtaken by an acute tubal inflammation, as a bronchial catarrh supervenes on chronic cirrhosis of the lung; lardaceous disease usually complicates tubal nephritis, or is complicated by it, but sometimes it is seen in a granular contracted kidney.

A satisfactory definition of Bright's disease is very difficult. It should not include temporary symptomatic albuminuria, such as accompanies the venous congestion of heart disease, or the secondary albuminuria of cholera, or the febrile albuminuria of erysipelas, diphtheria, or typhus. It should not include any cases in which transitory albuminuria occurs without there being reason to suppose any organic change in the kidneys. And, lastly, it should not include cases in which the presence of albumen is only due to that of blood, of pus, or of hæmoglobin. Taking these reservations into account, we may perhaps say that by Bright's disease is meant primary diffused nephritis, attended with persistent albuminuria; and its divisions we will treat as follows:

I. *Acute and chronic tubal or parenchymatous nephritis.*—This, as a rule, is attended with general dropsy. The onset is sudden, when scarlet fever or cold is the cause, but in other cases it may be more gradual. In the acute stage the kidneys are swollen and red; when the disease has become chronic they are whitish yellow, and sometimes very large. Finally, they may shrink and become granular. The urine is at first dense, high coloured, scanty, containing much albumen, tube-casts, and often blood. Later on, it may be pale, with a variable amount of albumen. Retinitis and uræmia are of frequent occurrence. In advanced cases the heart becomes hypertrophied, and the systemic arteries are thickened. This division includes the large smooth red and the large smooth white kidney, which are two stages of one pathological process, and also a few cases of the small contracted kidney.

a. Glomerular nephritis is the name given by Klebs to the form of acute diffuse inflammation which follows scarlatina. The glomeruli are often most affected, but not always, and the tubules also share in the change, and even the intertubular spaces; so that it is best regarded as only an anatomical variety of acute diffuse nephritis.

The following varieties of nephritis with albuminuria are not accompanied by dropsy, and are best not included under Bright's disease:

β. Febrile diffuse nephritis is a secondary condition common in all cases of prolonged pyrexia. The kidneys are swollen and full of blood from active congestion ,and on microscopic examination the epithelium of the tubes is in a condition of "cloudy swelling." This state is common to other secreting glands, and, though it produces albuminuria, subsides as the primary febrile state passes away. It appears never to lead to chronic disease of the kidney. It is present occasionally during scarlatina (independently of post-scarlatinal nephritis), and frequently in typhus, enterica, erysipelas, diphtheria, pneumonia, and other fevers.

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γ. *Congestive nephritis* is the result of long-continued passive or venous hyperæmia of the kidneys, usually the result of chronic disease of the heart. The organ at first only "hard," becomes gradually "coarse," the capsule somewhat adherent, and the section of the cortex blurred. Casts as well as albumen appear in the urine, and finally the kidney would probably atrophy.

II. *Lardaceous disease of the kidneys.*—This is caused by protracted suppuration or by syphilis. The kidneys give a characteristic reaction with iodine. Gradually they become very large, pale yellow, and waxy looking, as the result of the supervention of parenchymatous nephritis. Finally, they may shrink and become granular. The urine is excessive in quantity, pale, and contains much albumen; but when there is also acute nephritis, it may be scanty and high coloured. General dropsy is frequent. Cardio-vascular changes, retinitis, and uræmia are seldom observed. This is the form of Bright's disease discovered by Virchow after the two main types had been made out.

III. *Cirrhosis of the kidneys, or red granular atrophy.*—This is a slow and insidious affection, of which the chief known causes are gout, lead-poisoning, and probably drink. It is very rare in early life, but begins to occur towards forty years of age, and beyond this it is frequent up to seventy. It gradually destroys the renal cortex until this may not be more than a line in thickness; the surface of the organ remains of a red colour, but it becomes very uneven and granular. The urine is abundant, clear, pale, of low specific gravity; it contains only a small quantity of albumen, or there may be none at all for days together. Marked general dropsy occurs only when the affection becomes complicated with parenchymatous nephritis. Cardio-vascular changes are constantly developed, and reach an extreme degree. In many cases the patient dies with the symptoms of heart disease, including obstructive dropsy, which affects the dependent parts of the body. Cerebral hæmorrhage is another frequent cause of death. This division includes the great majority of cases of the small contracted red kidney. The two following sub-forms belong to the same category:

α. *Consecutive Bright's disease.*—This is seen as the result of such affections as stricture of the urethra, stone in the bladder, prolapsus uteri, compression of the ureters by an abdominal tumour, calculous pyelitis, and scrofulous disease of the kidneys. The kidneys become tough, hard, and whitish; they may be either of normal size or shrunken, either smooth on the surface or puckered by cicatrices, or granular. The general symptoms and the characters of the urine are those of renal cirrhosis.

β. *Cystic disease of the kidneys.*—The main peculiarity of this affection is the presence of innumerable cysts of various sizes. Commonly, the kidneys are small and contracted, but sometimes they are sufficiently large to be felt as abdominal tumours during life. In its clinical features it resembles renal cirrhosis; but it is sometimes congenital, and due to malformation.

Before entering upon a separate and detailed description of these different forms of Bright's disease we must consider certain symptoms and effects which belong, in a greater or less degree, to all of them in common:—Albumen and tube-casts in the urine, dropsy, and serous inflammations, retinitis, anæmia, cardio-vascular changes, hæmorrhages, and uræmia.

1. *Albuminuria.*—The coagulable constituent of the urine in Bright's disease is a mixture of two proteids, *serum-albumin* and *serum-globulin* or "paraglobulin," both of which are naturally present in the liquor sanguinis. They can best be separated by Hammarsten's process of saturating

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with crystallised sulphate of magnesia; this precipitates the paraglobulin, but leaves the serum-albumin in solution. Estelle found ('Revue Mensuelle,' 1880) that in certain cases of albuminuria in which he investigated the point, sometimes the whole of the so-called albumen, and, as a rule, all but one third of it, was really paraglobulin.

Subsequent investigations by Dr Halliburton of King's College, and by Dr Noel Paton of Edinburgh, seem to have proved that the proportion of globulin to serum-albumin differs greatly. It may exceed the latter, and sometimes is only present in traces. Globulin is somewhat more diffusible than serum- or egg-albumin, and is precipitated by carbonic acid gas, as well as by all the reagents which coagulate albumen.

There is no reason to suppose that the proportion of serum-albumin to globulin in the urine is of pathological or clinical importance.

A third proteid, the more diffusible *peptone*, is occasionally present in the urine, uncoagulable by heat, but recognised by the biuret test—a rose (instead of the albuminous purple) tint with cupric sulphate and soda.

Albumose (hemialbuminose or pro-peptone) and metalbumin have been found in the urine in certain cases, but the presence of these proteids has at present no diagnostic significance.

Acid albumen appears to be never present in urine, and alkali albumen only after ammoniacal decomposition has taken place.

Tests.—The oldest method of detecting albuminuria is by *heat*. The best way of applying it is to fill two thirds of a test-tube with urine and then to hold it near the bottom, while the upper part of the liquid is gently warmed over a spirit lamp. When the urine is acid, any albumen that may be present is precipitated. The temperature at which this occurs depends, first upon the amount of albumen (if it is very small, no change is perceived until the boiling-point is reached; if it is large, an opaque coagulum forms at about 150° F.); secondly, on the reaction of the urine (when alkaline a higher temperature is required, or the albumen may remain dissolved at boiling-point); and thirdly, on the amount of neutral salts, which if very small, raises the temperature requisite for coagulation.

For urine which is turbid with lithates this test is peculiarly applicable. The existence of opacity from this cause shows that there is sufficient acidity. The first effect of the heat is to redissolve the urates, and to make the fluid transparent; presently the albumen begins to appear and renders it again cloudy. In a long column of fluid all three conditions may be seen at the same time; at the bottom, a part which is cold and turbid; above it, one which is warm and clear; still higher, one which is hot and opaque. In alkaline urine heat may produce no change, although albumen is present; but one may avoid the risk of overlooking albuminuria by adding a drop of acetic acid, just enough to acidify the urine before beginning to warm it.*

If the urine is neutral or faintly acid, the application of heat frequently produces an opacity which looks like that due to albumen, but really con-

* If the amount of acetic acid be at all excessive, it may itself prevent the heat from throwing down any small quantity of albumen that the urine may contain, unless the urine is also rich in salts. The safest way, therefore, is, besides the acetic acid, to mix with the urine about one sixth of its bulk of a concentrated solution of common salt, or of magnesian or sodic sulphate. On heating the liquid after treating it in this manner, the albumen is seen to come down. Salkowski says that the test so applied is not only absolutely conclusive, but unsurpassed by any other in delicacy. Very dilute acetic acid will also precipitate globulin, but redissolves it in excess by changing it to syntonin.

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sists of a precipitate of phosphate of lime (p. 425). By adding a little acetic acid, we can redissolve the phosphates, and thus distinguish them from albumen. If urine which throws down the phosphate of lime when warmed also contains albumen, the opacity will not be removed by acetic acid.

A second valuable test for albumen in urine is Heller's *nitric acid* test. The best way is to pour a moderate quantity of strong acid into the tube, to hold it in a slanting position, and then to let the urine slide gently down until it floats on its surface. If no albumen is present, the two fluids are separated by a more or less deeply coloured layer, from oxidation of chromogen (p. 414). If albumen is present it forms an opalescent zone, of greater or less thickness, at the line where the acid and the urine meet. When the amount is exceedingly small, the zone may appear only after the interval of a minute or two. It is made more conspicuous by holding up the test-tube against a dark background, as, for instance, the sleeve of one's coat. Employed in this way, nitric acid is a very delicate test for albumen, and the most certain of all.*

In urine of high specific gravity, lithates are sometimes precipitated by the acid; but they first appear, not at the line of junction of the two fluids, but nearer the surface of the urine, the turbidity gradually spreading downwards. If there is any doubt, it may be removed by gently warming the test-tube, when urates will at once disappear. In concentrated urine nitrate of urea will crystallise out, but this takes time, and is quite unlike the dull white cloud of albumen. In the urine of patients who are taking copaiba or cubebs a resinous substance is excreted which is made more opaque by nitric acid, but not in a well-defined zone. The application of heat diminishes the opacity from this cause, and the odour should warn one.†

Many other tests for albumen are known besides the two already given. Acetic or citric acid with *potassium-ferrocyanide*, is a convenient one and free from most fallacies, but less delicate than either heat or nitric acid. Dr Pavy has invented pellets of citric acid and the ferrocyanide which form the best portable test. In the absence of the former the ferrocyanide would act perfectly if, as Dr Cavafy suggests, a little vinegar were added.

Dr George Johnson has strongly advocated the use of *picric acid* in saturated solution. One advantage which it possesses over nitric acid is that it can be carried about without the risk of damage if spilt or of giving off corrosive fumes. On the other hand, picric acid, like heat, fails to precipitate albumen in alkaline urine; and like nitric acid, it may precipitate lithates or lithic acid. It precipitates peptones (which are redissolved by heat), and it causes a cloud in the urine of persons taking quinine in full doses, which is also cleared by heat. Lastly, it precipitates mucus.

Dr Oliver, of Harrogate, has brought out a series of paper slips, saturated with picric acid, potassio-mercuric iodide, tungstate of soda, and other solutions which precipitate albumen, and these tests are certainly as portable as could be wished.

* Sir Wm. Roberts has shown that the readiness with which albumen is precipitated by nitric acid is to some extent affected by the presence of other dissolved matters. The proof is that if two samples of the same albuminous urine be diluted, the one with successive quantities of pure water, the other with the same quantities of healthy urine, the former continues to yield an opaque zone with nitric acid, after the latter has ceased to do so. The same observation was made independently by Dr Grainger Stewart. It depends on the solubility of all proteids being related, not only to the temperature and reaction of the solvent water, but also to the presence of neutral salts.

† I remember a man under treatment for gonorrhœa, who came out with the copaiba rash, and was supposed to have scarlet fever with his urine full of albumen!—C. H.F.

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Tincture of galls was used by Dr Rees as a sensitive precipitant of proteids in urine; so is metaphosphoric acid, and also a solution of mercuric iodide in iodide of potassium, but this is too sensitive, for it precipitates not only peptones and other proteids, but also mucus.*

On the whole, nitric acid is the most trustworthy of all tests, and if used by the contact method, and with a few minutes' grace, as delicate as is desirable; heat with acetic acid is still more delicate and nearly as certain; picric acid is quickest and most convenient, but it is not more delicate than heat, it is less easy to get a good contact action than with nitric acid, and, while it never fails to detect albumen, it may show a cloud when it is not present. The other proposed tests are either inconvenient, untrustworthy, or *too* delicate, *i. e.* they show such mere traces of proteid as may be the result of a little globulin from epithelial cells, pus, or mucus corpuscles, &c. Moreover, we must remember that we have a vast accumulation of facts as to albumen which refer to "urine coagulable with heat and nitric acid," and albumen means a different thing if it is defined by reaction to new and uncertain tests.

Quantitative estimate.—To determine with absolute accuracy the amount of albumen in the urine takes up much time; it has to be precipitated from a known bulk of the fluid, washed, dried, and weighed. In clinical practice, however, there is no sufficient object to be gained by this troublesome procedure. Sir William Roberts in 1876 proposed a method which is far easier, and which appears to yield sufficiently satisfactory results. It consists in diluting the urine with water until it almost ceases to give a reaction with nitric acid, the point fixed being that at which the opalescent zone at the junction of the two liquids begins to be visible between thirty and forty-five seconds after the addition of the acid to the urine. To calculate the number of grains of albumen per fluid ounce of urine all that is necessary is to multiply the figure 0·0034 by the number of dilutions with an equal bulk of water that the urine has undergone.

An easier plan, but one that yields only comparative results, is to take a column of urine of definite depth in a test-tube, and, after precipitating all the albumen in it with heat or with nitric acid, to let it stand until the coagulum has sunk to the bottom, forming a layer the depth of which can be expressed as a fraction of that of the urine, a half, or a quarter, or one sixth, as the case may be. Vogel, however, found that the space occupied by the same quantity of albumen might vary widely according as it happened to be thrown down in larger or smaller masses; and it is also influenced by the specific gravity of the urine, the range of error from these causes being as much as from 30 to 50 per cent. (Esbach).

A mixture of 10 grammes of picric and 20 of citric acid to the litre has been devised by Prof. Esbach, which, when added to urine, precipitates any albumen present quickly and completely, and the precipitate falls more perfectly than that of picric acid or heat alone. After twenty-four hours it may be read off in a graduated tube, and the percentage of albumen may thus be calculated. For comparative estimates of the albumen passed in a given case this method is quite sufficient; but it fails when the amount is very small; and when it is very large. the urine must be diluted before using the test, and the result corrected accordingly.

* With respect to the relative merits of tests for albumen in the urine, including his own, of acidulated brine, see Sir William Roberts's valuable criticism in the 'Discussion on Albuminuria,' at Glasgow, in 1884, p. 16. Also the report of a committee published in the 19th volume of the Clinical Society's 'Transactions.'

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Dr Johnson and Dr Grainger Stewart find that Esbach's method gives results nearly the same as those obtained by precipitating and weighing. In fact, however, the precise amount of albumen is not very important, and may almost be left to rough estimation.

The actual weight of albumen contained in the most bulky coagulum is but small. Accurate analysis seldom gives more than 5 per cent., even when the urine becomes solid when boiled.

Theory of albuminuria.—The first question in the inquiry why albuminuria occurs in Bright's disease is why normal urine contains no albumen. What prevents its escaping from the blood with the water, urea, and salts which transude in the glomeruli? Only one answer to this question seems possible, namely, that it is kept back by the epithelial layer which covers the capillary tufts; and, as Cohnheim remarks, it is interesting to notice that the vessels of the choroid plexuses, which also yield a non-albuminous fluid, are the only ones that have a similar investment.*

That when albumen appears in the urine it escapes through the glomeruli is rendered probable by Nussbaum's experiments on frogs ('Arch. f. Phys.,' 1878), in which animals these structures have an arterial supply distinct from that of the renal tubes; he ligatured the glomerular arteries, and found that after this operation egg-albumen, injected into the stomach or into the blood, no longer passed into the urine, as it does when the circulation of the kidneys is undisturbed. Again, Ribbert ('Centralblatt,' 1879), having set up an artificial albuminuria in rabbits with egg-albumen, excised the kidneys, and placed them directly in alcohol, so as to coagulate *in situ* the albumen in their interior; he then found that the spaces within the Malpighian capsules always contained coagulum as well as the tubes. It is still possible that albumen may transude through the tubal capillaries also; but in that case it would naturally find its way, not into the urine, but into the lymph-spaces between the tubes. Hence, even when albuminuria is dependent upon obstruction to the blood-flow through the systemic veins, it is not perhaps directly due to an escape of serum through the tubal capillaries as the result of increased pressure.

The earliest theory of albuminuria was that of Dr Owen Rees (lately revived by Prof. Semmola, of Naples), according to which it depends upon a chemical change in the albumen of the liquor sanguinis, enabling it to pass through the walls of the glomeruli (cf. p. 440). Stockvis ingeniously disproved this notion by the direct experiment of injecting albuminous urine from patients with Bright's disease into the veins of animals, when he found that the albumen did not, as a matter of fact, escape with the urine that they passed.

The same observer failed altogether to obtain experimental corroboration of the idea, formerly common, that hydræmia may be a direct cause of albuminuria. A low specific gravity of the blood probably obtains in most cases of anæmia, but albumen is absent from the urine.

Nor does there seem more reason to attribute albuminuria to the increased presence of neutral salts in the blood; this would increase osmosis through a membrane, but not transudation through endothelium. The same criticism applies to diminished alkalinity of the liquor sanguinis, of which, moreover, there is little evidence. Experiments on animals do not support

* A theory was once current, and received the high authority of Ludwig, according to which albumen was supposed to be present in the transudation from the glomeruli, but to be taken up again and restored to the blood by the epithelial cells of the convoluted tubes; but this was refuted by the observations of Posner ('Virch. Arch.,' 1880).

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any of these chemical or physical theories of albuminuria, nor that which ascribes it to increased blood-pressure; whereas they do support its being a result of diminished arterial pressure, or of cutting off the flow of blood from the kidneys for a time, or of increased pressure in the renal veins.

The most probable immediate cause of albuminuria is neither chemical nor hydraulic, but structural, or, to be more precise, the occurrence of some nutritive change in the epithelium covering the glomeruli, which renders it no longer capable of resisting the passage of albumen.

Whenever a full stream of arterial blood is not kept up through the capillary tufts their epithelium is liable to be damaged, so that it can no longer fulfil its normal function. The instance which Cohnheim adduces as most obviously supporting such an opinion is that of the albuminuria which follows the suppression of urine during an attack of cholera (vol. i, p. 231). This, he maintains, is precisely analogous to the albuminuria which can be experimentally produced by temporary obstruction of the circulation through the renal artery, and which lasts for hours or even days after the obstruction is removed. Another cause of deficiency of blood-supply to the glomeruli may be an impeded outflow through the veins of the kidneys; for Ribbert has shown that after arrest of the circulation through the renal artery the cells of the glomerular epithelium become obviously swollen and altered in appearance.

According to this conception of the pathology of albuminuria, it has no essential relation to the state of the blood-pressure in the arteries of the kidneys. The prevalent doctrine, that nothing favours the escape of albumen so much as an increase of (systemic) blood-pressure, was the conclusion at which Stockvis arrived as the result of his elaborate investigations, and it was adopted by Bartels. It appears, however, to rest on no good evidence, experimental or pathological. The occurrence of albuminuria as the result of venous obstruction certainly lends it no support, because the pressure in the glomeruli is probably thus diminished rather than excessive, in consequence of the enfeeblement of the heart's action, which arises at an early period in such cases; and Runeberg goes so far as to maintain that albuminuria is always dependent upon *deficiency* of arterial blood-pressure. It is the *quantity of urine* passed which is increased by a high blood-pressure in disease just as in health.

A conclusive pathological argument against albuminuria depending on increased blood-pressure is that it is least in constancy and degree in those cases of advanced Bright's disease in which the blood-pressure is the highest.

It is undoubtedly to changes in the glomeruli that the albuminuria of Bright's disease is principally due, though, perhaps, the renal tubes may also have a share; whether this is more likely to be the case when their basement membrane has been exposed as the result of exfoliation of the epithelium must at present be regarded as doubtful.

"*Physiological*" *albuminuria.**—If now we pass on to discuss the conditions under which albuminuria occurs, we find, in the first place, that it is seen in many persons who are, so far as can be ascertained, in good health, and whose kidneys appear to be perfectly sound. It is only within the last few years that this fact has been clearly ascertained. Leube tested ('Virch. Arch.,' 1878) the urine of 119 soldiers, and found albumen in the urine passed in the morning by five of them, and in that passed at midday after a march by no fewer than nineteen; the urine passed in the evening was

* *Syn.*—Functional, intermittent, or cyclical albuminuria.

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never albuminous. Capitan found albumen in the urine of nearly 45 per cent. of French soldiers. Fürbringer (*ibid.*, lxxi; and 'Ztschft. f. klin. Med.') tested the urine of sixty-one children, and detected albumen in seven cases, always in the latter part of the forenoon. In other instances the presence of albumen in the urine of healthy persons has been traced to some definite cause. Thus Dr George Johnson has recorded ('Clin. Soc. Trans.,' vol. vii) several cases in which it was temporarily produced by cold bathing; the same observer ('Brit. Med. Jour.,' 1879, ii) alludes to other cases in which it followed active walking exercise, an instance of which also occurred to a medical friend of the author's in his own person. In another case which came under the editor's notice, temporary albuminuria followed a day's hunting. Fürbringer met with a case in which it was more than once brought on by distress of mind. Dr Moxon related in the 'Guy's Hospital Reports' for 1878, several cases in which albumen was from time to time discoverable in the urine of boys and young men who were generally anæmic, listless, and languid; with all in whom he was able to trace the further progress of the affection, it sooner or later passed off, usually in the course of a few months. Sir William Gull had previously observed the same condition, also in young persons, and associated with similar symptoms. In some of Dr Moxon's cases, "albuminuria of adolescents" (as he terms it) was associated with oxaluria. Ernst Wagner speaks of having seen similar cases in anæmic and weakly girls. Dr Dukes, of Rugby, shortly afterwards stated that he had seen ten cases in boys of thirteen to seventeen, in whom albuminuria had occurred as the result of cold, exertion, or excitement, but subsided when they were kept in bed and on a milk diet ('Brit. Med. Journ.,' November 30th, 1878). Dr Pavy has described the same clinical condition as "cyclic albuminuria," by which is denoted the recurrence of traces of albumen in the urine at more or less regular intervals, which correspond chiefly to the periods of taking food. As the same author long ago proved (1863), the amount of albumen passed in cases of Bright's disease varies according to the amount and nature of the diet. Mr Lucas met with several cases among surgical out-patients, associated with flat-foot, in youths whom he believed to be habitually guilty of masturbation ('Brit. Med. Journ.,' May 3rd, 1884).

One of the most recent investigations on this subject was made by Dr A. W. Stirling on 369 boys between twelve and seventeen years of age, on a training ship at Grays, on the Thames ('Lancet,' 1887, vol. ii, p. 106). Of the whole number he found more or less albumen in 77, without any other sign of renal disease. It was far from constant, and most often present soon after rising. Besides the exposure to cold on leaving bed, two other causes were suggested—one that assigned by Mr Lucas, and the other, exertion in blowing wind instruments.

Dr Grainger Stewart has since published a series of careful observations which bear out to a certain extent the assertions of Capitan on French, and Leube on German soldiers, of Dr Munn on applicants for assurance in America, and of Dr Stirling on the boys at Grays. Among 205 soldiers in Edinburgh he found albumen in no less than 47;* among 100 presumably healthy civilians in only 7; among 150 workhouse children he found it only in 5; among 100 old people in 17. He found considerable increase in the frequency of albumen after severe muscular work, and

* Dr Stewart gives the numbers obtained by nitric and by picric acid: the figures quoted in the text are those obtained by the former method, for the reasons given above.

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less marked but decided increase after food. The results he obtained in the case of boys after playing wind-instruments agree with those of Dr Stirling, but in a much less degree.

Dr Parkes and Dr Pavy long ago showed that when albumen is present it is increased after food, but there is no evidence that eating eggs or anything else not poisonous will cause albuminuria in a previously healthy person.

The view which is taken by recent German writers of what they call "physiological albuminuria" is that it depends upon a congenital deficiency in the power of the glomerular epithelium to resist the passage of albumen through it. It may perhaps be urged in support of such a theory that there are two pairs of brothers among the seven cases of which Dr Moxon gives details; and Leube also mentions having met with the affection in two brothers. But these cases are quite exceptional.

The condition is not a constant but an intermittent or occasional one, and its causes are often not far to seek. Cold to the surface, causing congestion of the kidneys, is the most important. Thus, staying long in a cold bath is a frequent cause of this occasional albuminuria. It is also a cause of intermittent hæmoglobinuria; and in the case of the schoolboy cited above (p. 435), as in others, the same cause produced on most occasions hæmoglobinuria, on others albuminuria. The same explanation applies to the frequent passage of traces of albumen in the urine on first getting out of bed. There is no absolute line between physiology and pathology; and if we believe (as we shall see there is good reason to believe) that exposure to cold over a large surface of the skin leads to acute congestion of the kidneys with passage of albumen, and even to acute nephritis, it is surely reasonable to suppose a less degree of the same effect in the cases under consideration.

The effect of prolonged efforts of expiration, as in blowing trumpets, would produce passive renal congestion like that which leads to albuminuria in cardiac disease.

The passage of albumen associated with oxaluria and "nervous debility" has probably a somewhat different pathology.

Is it quite certain that in cases of "physiological albuminuria" the albumen was always renal in origin? Notwithstanding care to exclude such cases (which was certainly not taken by all observers), were not many of the cases in French and other armies due to *la goutte militaire?* Have not many old men in workhouses granular kidneys? Lastly, has not a precipitate of mucin by picric acid sometimes been taken for albumen?

But the most important question is whether in such cases, whether more or less rare, the occurrence of albuminuria indicates any tendency to the development of organic renal disease. Ought a young man in whom this affection is discovered to be regarded as eligible for life insurance at the ordinary rate? Leube and Fürbringer would doubtless have answered this question in the affirmative, and so would Moxon, if it were clearly ascertained that the urine contained albumen only occasionally, and that in the forenoon. Both Fürbringer and Moxon detected a few hyaline casts in more than one instance, but this is certainly a rare exception; as a rule, the amount of albumen is small, it is intermittent, not constant, and there are no other signs of disease in the urine.

It is clear that the cases above recorded by so many independent

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observers are far too numerous to be set down as examples of latent Bright's disease. Had they all been of that nature we may be sure that some of them would have revealed their real character while they were still under observation; moreover, latent Bright's disease is of the cirrhotic kind, which is almost unknown in early life. But is there equal reason to be sure that they are not examples of incipient Bright's disease, or of the want of power to resist external influences which is the preceding condition of all diseased action? Dr Johnson has expressed ('Brit. Med. Journ.,' 1879) a decided opinion that temporary albuminuria, even when traceable to food, or over-exertion, or exposure to cold, will, if neglected, sooner or later lead to persistent albuminuria and to fatal disease of the kidneys. Of the fact that it is possible for albuminuria to be the only indication of ill-health, and yet for the kidneys to be undergoing grave structural changes, there is no doubt whatever. Dr Johnson mentions the case of a medical man, actively engaged in a large practice until shortly before his death from uræmia at forty-five years of age, whose urine had been albuminous from the time when he had been a student, and probably earlier still, for he had scarlatinal dropsy when fifteen years old. Clearly, therefore, an insurance office which ignored the presence of albuminuria in applicants who appeared otherwise well would sometimes lose thereby.

The only insurance company that is known to have inquired into the subsequent state of health of persons whose lives had been declined on account of albuminuria is the United States Company in New York. Among those who made applications to that office in the three years, 1878–80, there were sixty-nine (or from 10 to 12 per cent. in each year) whose urine was found to be albuminous. Before the end of 1880 four of these persons died, and it is stated by Mr Munn that the "general appearance of the majority of the others who had been under observation for more than a year was gradually deteriorating." It is to be noted, however, that few of them were under the age of thirty, and that the albumen was often present in considerable quantity. Consequently, although the results of this investigation show that an insurance company runs a great risk if it neglects to have the urine of applicants tested, they can hardly be said to throw much light upon the question of the occurrence of a physiological albuminuria in young subjects.

It is a significant fact that Mr Eales, of Birmingham, found retinal changes in five out of fourteen cases of supposed temporary albuminuria in persons between the ages of eleven and twenty-eight ('Birmingham Medical Review,' 1880).

Albuminuria in non-renal diseases.—Albumen occurs in the urine without there being any marked or permanent lesions of the kidneys, under various morbid conditions, which may be briefly enumerated under the following heads:

i. *Mechanical causes.*—(1) Venous congestion; usually from obstruction of the general venous circulation as the result of valvular disease of the heart, or occasionally of dilatation of the right ventricle from emphysema or like affections of the lungs.

Obstruction of the renal veins, independently of any disease affecting the whole circulation, occurred in a case recorded by Bartels of obliteration of the inferior vena cava above the mouths of those veins. Albuminuria from pressure on the inférior cava and renal veins by ascites or tumour or a pregnant uterus is not uncommon.

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(2) Obstruction in the renal arteries, usually by an embolus detached from the heart, and producing congestion with escape of albumen or of blood into the urine, by the same mechanism as explains cerebral hæmorrhage from a similar cause.

(3) Arterial anæmia, leading to deficient blood supply to the kidneys through the renal arteries, as in cholera.

The same explanation probably belongs to albuminuria occurring in affections attended with abdominal pain and collapse. This cause of albuminuria has been especially pointed out by Fischl ('Deutsches Arch.,' 1881).

(4) Albuminuria appearing after obstruction of the ureters has been removed. This cause has been established by experiments on animals, and a case in point is given by Bartels in which the obstruction was produced by a calculus.

ii. *Fevers.*—Pyrexial albuminuria occurs most frequently in acute pneumonia and diphtheria, but also in typhus, enterica, cerebro-spinal meningitis, erysipelas, ague, pyæmia. In scarlet fever, also, albuminuria may occur during the pyrexial stage, and ought probably to be distinguished from that which appears later and is dependent upon nephritis. Cloudy swelling of the renal epithelium is constantly found in the bodies of those who have died of febrile maladies, but it is doubtful whether this causes the albuminuria, which is far less frequent.

iii. In certain affections of the nervous centres, especially cerebral hæmorrhages, concussion of the brain, epilepsy, tetanus, delirium tremens, albuminuria may occur; but it is doubtful whether it is caused by the nervous disorder. Cerebral hæmorrhage is notoriously frequent in cases of cirrhosis of the kidney, and drunkards are liable to Bright's disease. The albuminuria which follows a fit of epilepsy should always awake suspicion of uræmic eclampsia.

iv. *Poisoning* by different substances which act as direct renal irritants, *e. g.* cantharides and turpentine. Phosphorus probably acts in a different way.

If we ask what explanation can be given of albuminuria under these several conditions, the answer is more or less unsatisfactory, except in the case of cholera. Taking, for example, febrile albuminuria, we are quite unable to say whether the invisible physical change in the epithelium of the glomeruli, to which (as we believe) must be attributed the escape of albumen, is due to the heat of the blood itself, or to the action of the heat on the renal nerves, or to the chemical changes in the blood, or to the disturbance of the circulation through the kidneys as the result of diminished arterial pressure. When albuminuria follows an epileptic fit, or accompanies apoplexy or tetanus, it is probably a secondary result of venous obstruction from impeded respiration. But the relation of albuminuria to venous obstruction is itself susceptible of various interpretations. It may depend upon a deficient supply of arterial blood to the glomeruli, interfering with the due nutrition of their epithelium; or distension of veins at the line of junction of the renal cortex with the medulla may compress the straight tubes, and so interfere with the flow of urine, and exert pressure backwards in the Malpighian capsules upon the outer surface of the glomeruli. It has been thought that when albuminuria follows plugging of the ureter, the distended renal tubes press upon the veins; and thus that this cause of albumen in the urine may also be included under the head of "venous obstruction."

It may be stated generally of so-called "functional" and of "sym-

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ptomatic" albuminuria, that in none of its varieties does it approach in amount, either absolutely or in proportion to the urine, that is often found in Bright's disease. In some cases the urine may contain 5 per cent. of albumen, a quantity more than half as great as that in normal blood-serum, although the proportion is generally much smaller.*

2. *Tube-casts.*—The discovery of these bodies—"urine-cylinders," as the Germans call them—in the urine is generally associated with the name of Henle, who described them in 1844; but they had not escaped the notice of some previous observers. The merit of distinguishing and figuring their varieties, and of applying their discovery by the microscope to the diagnosis of renal disease, is undoubtedly due to Dr George Johnson, of King's College, London (1852). There are several different kinds of casts.

(1) *Hyaline or fibrinous casts.*—These are delicate, transparent, and colourless, with defined outlines, but so little refractile that they are not always recognised under the microscope in the fluid in which they float, unless stained by carmine or iodine or aniline dyes. They vary greatly in breadth, from 0·01 to 0·05 mm.; their length may be only a few times greater than their breadth, or may reach 1 mm.; they are the longest of any kind. They may be either straight or curved.

(2) *Blood-casts, i. e.* fibrinous cylinders filled with red blood-discs. Their presence shows that the exudation is hæmorrhagic and probably *acute*.

(3) *Corpuscular casts*, containing small round nucleated cells, which may be pretty certainly identified as leucocytes or "exudation corpuscles," *though* often confounded with epithelial cells. Like the hyaline casts they are signs of nephritis.

(4) *Epithelial casts*, containing glandular cells from the convoluted tubules, more or less altered and granular, but by their larger size and more angular shape distinct from white blood-corpuscles. They point to desquamation as the result of nephritis.

(5) *Oil-casts*, or fatty casts, containing highly refracting oil-drops. *These* prove that the nephritic process has become chronic.

(6) *Granular casts.*—These are the most common, and the least *distinctive*, for the granules may result from the disintegration of blood-discs (when they have a yellow tint), or of epithelium or leucocytes, and, in fact, are often mingled with these elements in the same cylinders. Or they may be fat-granules, shown as bright dark points. Or they may be only lithates accidentally deposited after the urine is passed.

(7) Lastly, one or two minute crystals of calcic oxalate or uric acid may be seen in a tube-cast.

In acute Bright's disease, for weeks together, the casts may contain red blood-discs, to the exclusion of all other elements. In other instances *tube*-casts look as if they consisted almost entirely of epithelial cells, packed so closely together that little or none of the hyaline sheath can be seen. The fat-granules or fat-drops are probably always derived from disintegrating epithelium; casts in which they are abundant are opaque and conspicuous, almost black by transmitted light. Wagner describes "granular casts" as

* We must remember that the urine as we obtain it is, after all, a mixture of the fluids poured out by an almost infinite number of glomeruli and renal tubes, which may yield secretions of very different quality. So that when renal emboli, or localised new growths, are surrounded by zones of hyperæmic and inflamed kidney-tissue, any albumen that may be contained in the secretion from these parts is necessarily distributed over the very much larger quantity of normal urine poured out from the rest of the cortex.

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sometimes "opaque like ground glass," "appearing as if eroded or breaking down at their edges," and "sometimes presenting numerous indentations, or looking as though they were made up of a number of square pieces fused together." Lastly, "waxy" or "lardaceous" casts, which show the reaction with iodine, are described as occurring, though very rarely; they are highly refractile, and show more resistance to reagents than the common hyaline casts. Bartels is disposed to admit the possibility of their acquiring the lardaceous character as the result of being long retained in the renal tubes; and some writers affirm that they are not peculiar to cases in which the kidneys are themselves lardaceous.*

After death, casts may be seen in the kidneys in every part of their substance, from the convoluted tubes near the glomeruli down to the wide collecting tubes in the pyramids. They are most abundant in the looped tubes; as Wagner thinks, because they are slow in passing through these narrow canals. Certain writers have doubted whether casts from the convoluted tubes are capable of traversing the looped tubes so as to be discharged with the urine; but they are so elastic and flexible that this does not seem impossible for at least the smaller casts, and probably some of the larger casts are formed in the convoluted segment just before the collecting tube. Possibly materials that originally solidified in the highest tubes close to the glomeruli may afterwards be, so to speak, recast, taking the form of tubes lower down. It is stated, both by Wagner and by Bartels, that very wide waxy or granular casts are found chiefly in cases in which the urine is very scanty, and especially in the chronic forms of Bright's disease, within a few days of the fatal termination; and Bartels observes that such casts, moulded in the collecting tubes of the pyramids, can only be retained there long enough to undergo secondary changes when the secreting activity of the kidney is at a very low point.

The chemical nature of the hyaline material which appears to be the basis of all recently formed tube-casts has been especially studied by Rovida, who arrived at the conclusion that it is not identical with either fibrin or albumen, so that it can only be described as an *albuminoid* substance.

There have been various opinions as to the mode of origin of casts. Some observers have supposed them to be produced by a process of secretion from the epithelial cells of the tubes. Bartels upholds this view on the ground that spheroidal masses of plasma can often be seen protruding from the cells into the lumen of a tube. Wagner, however, says that such appearances may be observed even in healthy kidneys. Another theory has been that they arise by the fusion together of altered epithelial cells. According to Weigert ('Volkmann's Sammlung,' 162–3) this is obviously the case in animals when nephritis is set up by the injection of chromate of potass under the skin; and in Bright's disease such an origin seems probable in the case of certain casts which have indented margins, or look as if they were made up of an agglomeration of angular pieces. Some writers

* Under the name of "cylindroids"—for which "false casts" would perhaps be the best English equivalent—some German observers have described certain flat, riband-like bodies, which are found in the urine of patients with scarlet fever, and also in cases of cholera and of recurrent fever. They are pale, homogeneous, colourless, and reach a much greater length than ordinary hyaline casts. Wagner says that their nature is still unknown. Some pathologists think that they are mucous, and that the urine does not contain them till after it has escaped from the pyramids; others believe that they have found them in the renal tubes. It does not appear clear that there are any transitional forms between "false" and "true" casts, though they may be found together in the same specimen of urine.

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think, too, that casts which turn reddish brown with iodine are formed out of epithelial cells that have first become lardaceous. But for the ordinary hyaline casts by far the most probable view is that they result from the coagulation of fibrinogen exuded from the glomeruli, just as in any other case of plastic or "croupous" inflammation. The fact that their reactions are not identical with those of fibrin may perhaps be explained by the supposition that they undergo some further chemical change under the influence of the acid urine which bathes their surface. The very short interval which sometimes passes between the commencement of a morbid change in the kidney and the appearance of casts in the urine affords strong argument in favour of the view that they arise by coagulation of exuded plasma. Bartels, for instance, states that in a patient who underwent the operation of transfusion with lamb's blood, and whose urine up to that time was normal, urine passed two hours afterwards contained not only albumen, but also hyaline casts. In another case, that of a man who fell from a height upon his sacrum, urine voided five hours later showed hyaline as well as blood-casts. There is, indeed, a very close relation between albuminuria and the presence of tube-casts. In some cases, however, they appear in the urine a few hours, or even a day or two, before albumen is discoverable. In the urine of jaundiced patients casts of a greenish-yellow colour are often found, and Dr Finlayson says that, as a rule, in such cases no albumen is present. Roberts alludes to cases of venous obstruction from heart disease or emphysema as being also accompanied with renal tube-casts, although there is no discoverable albuminuria. Of course nothing is proved by the fact that in cases of acute Bright's disease casts sometimes continue to be passed after albuminuria has ceased, because they may have been retained in the renal cortex for a considerable time after their formation. As a rule, the abundance of casts in a case of Bright's disease is proportionate to the amount of albumen in the urine; but to this there are exceptions, and in the same patient the number of casts may vary from day to day.

It is generally said that tube-casts possess great clinical importance from the fact that they prove the kidneys to be diseased. This is so far true that in cases in which the urine contains pus or blood, which may have been derived from the renal pelvis or the lower urinary passages, the discovery of casts is good evidence that the renal cortex is affected; although, if none can be found, it proves little, for their recognition is very difficult when leucocytes or red blood-discs are present in numbers. If albumen only is present, the presence of a few hyaline casts is not absolute proof of the existence of Bright's disease rather than of those slight or temporary changes in the glomeruli which occur in association with pyrexia, or in consequence of venous congestion, as the writer has verified after death; but when their number is considerable and their contents varied, the diagnosis of primary renal lesion is practically certain.

3. *Dropsy.*—In Bright's disease we meet with two kinds of dropsy. One is identical in its characters with that seen in heart disease, and depends upon obstruction of the systemic veins. When it appears in the course of morbus Brightii it is only an indirect effect of the primary malady, its immediate cause being failure of the heart to maintain the needful activity of the circulation. It is always more marked in the dependent parts of the body than elsewhere, especially in the lower limbs; and it is associated with dyspnœa, with orthopnœa, and often with lividity. It occurs only in the

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most chronic forms of Bright's disease, usually when the kidneys are contracted, red, and granular, *i. e.* cirrhotic.

Widely different from this is the kind of dropsy which, although perhaps not absolutely more frequent, has been always justly associated with Bright's disease as its characteristic symptom. This kind of dropsy often begins in the face, about the eyelids, even before it affects the ankles. Its distribution is not independent of the influence of gravitation; for one may often notice that whereas the face is œdematous when the patient rises in the morning, this subsides towards the latter part of the day, and the ankles are swollen when he goes to bed. But it is not limited to the dependent regions of the body like the other form of dropsy, and it is not accompanied by dyspnœa or lividity. Its favourite seats are the eyelids and conjunctiva, the penis and scrotum (or the labia in women) and the loins, when it forms what Bright called "the renal cushion." We may explain the two former seats as due to the fact that the skin of eyelids and genital organs has no subcutaneous fat. Often, however, the whole of the body and limbs swell at the same time, and acquire a peculiar white waxy appearance, which is very characteristic. The occurrence of such general dropsy is frequently the earliest symptom of Bright's disease, and first draws the patient's attention to the fact that he is unwell. Generally, however, the urine is found, if tested, to be already albuminous; and after scarlet fever, when the supervention of dropsy can be anticipated as likely to happen, albuminuria may be known to be present for several days before œdema can be detected. On the other hand, it sometimes happens that the dropsy precedes the albuminuria by a day or two.

Theory of renal dropsy.—The most obvious suggestion is that the characteristic anasarca of Bright's disease depends upon hydræmia, the result of diminished action of the kidneys. Bostock and Rees, who made analyses of the blood for Bright himself, Christison, and many later observers have found that the density of the serum is greatly reduced, being not more than 1020, or even 1013, instead of the normal density of 1030. It is natural to refer this physical change in the blood to the deficient excretion of water; but some writers have laid special stress upon the loss of albumen through the glomeruli of the kidneys, and the resulting sub-albuminous state of the blood, as the main cause of the low specific gravity of the blood-plasma. But Cohnheim points out that the amount of albumen which is secreted by the kidneys is after all inconsiderable. In most cases the percentage of albumen in the urine does not exceed 2 per cent.; in exceptional instances it may reach 4 or 5 per cent., but then the quantity of urine passed in the twenty-four hours is always much diminished, so that after all the total daily loss of albumen cannot be calculated at more than from eight to ten or twelve grammes (two or three drachms). It is obvious that, unless the assimilation of food is greatly interfered with, such an amount of albumen can be very easily replaced. Moreover, quite as large quantities of albumen are lost, without any dropsy resulting, by patients with large granulating wounds, and by those who have chyluria; and far larger quantities by women during lactation.

It has been urged by Bartels and others that the really important factor in the production of renal dropsy is the deficient excretion of water by the kidneys. Rehder is cited by Bartels as having made a very elaborate series of investigations, in several cases of Bright's disease, as to the relation between the amount of water drunk (that contained

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in the solid food being, however, left out of consideration) and that discharged in the urine from day to day; in one case particularly he found that during periods when the dropsy was on the increase the water excreted was not more than from 29 to 49 per cent. of that which was ingested, whereas during periods when the dropsy was decreasing the ratio was from 72·5 to 100·5 per cent. But, as Cohnheim remarks, such observations, after all, warrant no conclusion as to the nature of the connection between scantiness of the urine and dropsy. One has just as much right to suppose that the variations in the dropsy caused those in the activity of the kidneys as to take the converse view. The effect upon the blood of a deficient excretion of water by the kidneys (supposing it not to be corrected either by diminished ingestion of water, or by an increased loss of water through some other channel) must obviously be to increase the whole bulk of the circulating fluid, while diminishing the percentage of solids in it. Cohnheim expresses this by saying that the resulting state of the blood must be, not mere "hydræmia," but "hydræmic plethora." Now he and Lichtheim ('Virchow's Arch.,' lxix) made a series of experiments upon dogs, in which they found that the injection of enormous quantities of a half per cent. solution of salt into the blood produced not the slightest anasarca, even when the renal arteries were ligatured, so as to cut off the escape of the fluids through the kidneys. So far, therefore, as experiment can settle t he question, it appears that an "hydræmic plethora" is incapable of causing the dropsy of Bright's disease.

But, in fact, there is no evidence whatever that such a condition of the blood occurs in this disease, or *can* as the result of impairment of the renal functions. Unfortunately nothing is positively known as to the amount of water which escapes from the lungs or from the skin, though it must be admitted that the dry harsh state of the cutaneous surface in many cases of Bright's disease, and the difficulty with which visible sweating can be induced, render it unlikely that the skin takes up any part of the renal function. But in one way or other the inactivity of the kidneys is compensated for, and the volume of the blood remains unaltered or nearly so. Further, there is abundant clinical proof that even complete arrest of the secretion of urine causes no dropsy. Not to mention the anuria of hysterical women, there are the cases of "obstructive suppression" resulting from plugging of the ureter of a single kidney, the other kidney having been previously destroyed by disease (*infra*, p. 525). In animals, again, ligature of the ureters is equally incapable of producing dropsy. Lastly, in many cases of scarlet fever, dropsy sets in before there is evidence of impairment of the renal functions, and certainly long before there has been time for the development of any great change in the density or in the volume of the blood as the result of such impairment.

Such considerations render it clear that some further explanation is needed of the occurrence of general dropsy in Bright's disease; and this is sought for by Cohnheim in a change which he supposes to take place in the walls of the capillaries, rendering them more readily permeable by fluids than they are in normal circumstances. In most cases the deficiency of albumen in the blood might be imagined to produce such a change; but this view is inconsistent with the fact that in some instances the dropsy sets in before there can have been time for the blood to become "hypalbuminotic" or subalbuminous. Cohnheim, therefore, falls back upon the ingenious suggestion that the vessels of the skin and of the subcutaneous tissue

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become altered by the same cause which sets up the renal affection. He points out that whereas dropsy accompanies the nephritis that follows scarlet fever or exposure to cold, no such result is observed when a like nephritis arises in the course of diphtheria or of relapsing fever, in which diseases the skin remains intact. Obviously the explanation is valid only so far as anasarca is concerned; and Cohnheim accordingly insists that dropsy of the serous cavities and of mucous membranes does not occur in most cases of Bright's disease—at least in an early stage, when failure in the heart's action cannot be supposed to play any part in their production. But the author's experience certainly accords with that of Wagner, that in autopsies upon some most acute cases—as, for instance, after scarlet fever—one generally finds some fluid effused into deeper parts of the body, though not, perhaps, in very large quantity. In more chronic cases of tubal nephritis large effusions in one or both pleuræ or in the peritoneal cavity are very frequent, and certainly not of cardiac origin.

It is evident, as Cohnheim himself points out, that the hypothesis of a change in the capillary walls, as the immediate and fundamental cause of renal dropsy, brings the affection somewhat closer than before to the inflammatory forms of œdema. But in one respect there is an important difference, namely, as regards the composition of the effused liquid. This, in Bright's disease, has always an extremely low specific gravity, and contains but a very small quantity of albumen; in fact, it exactly resembles in these points the liquid that is poured out in the "mechanical dropsy" of heart disease, or of pulmonary emphysema. C. Schmidt found in one case that the dropsical fluid from the subcutaneous tissue contained 0·36 per cent. of albumen, that from the meninges 0·6—0·8 per cent., that from the peritoneum 1·13 per cent., that from the pleura 2·85 per cent. Bartels examined fluids taken directly after death from different parts of the body of a person who died of advanced dropsy, and found that the specific gravity of the blood-serum being 1015·60, that of the pericardial fluid was 1009·7, that of the peritoneal fluid 1009·6, and that of the anasarcous fluid 1007·65; in each of the dropsical fluids the main part of the solid constituents was made up of inorganic salts. Urea, in the proportion of about 0·3 per cent., was detected in anasarcous fluid, and also in ascitic fluid by Edelssen; in pericardial fluid he once found as much as 1 per cent. of urea.

Difficult as it is to answer all objections, it seems probable that the characteristic anasarca of acute Bright's disease, as well as the pleuritic and ascitic effusion, is really—as it was supposed to be before Bright began his researches—an inflammatory exudation. This may be diluted and increased by watery exudation proceeding not directly from hydræmia, but from diminished arterial and increased venous pressure everywhere.

Dropsy without albuminuria.—Cases are now and then met with in which there is dropsy of precisely the same character as that which is so constantly associated with Bright's disease, but in which no albumen can at any time be found in the urine. Such cases are sometimes dignified by the name of "*essential dropsy*," but one may fairly doubt whether the kidneys are healthy, although no clinical evidence to the contrary can be obtained. What, however, is more frequent is for a patient to come under observation with general dropsy that has already lasted several days, or even weeks, and for his urine to yield no coagulum, either then or at any subsequent period, while the dropsy more or less rapidly subsides. In these cases, which are not very rare, nothing is more likely than that albuminuria was really

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present at first, for it is well known that in the more transitory forms of Bright's disease the urine often becomes normal before the dropsy disappears. The writer has seen this acute anasarca without albuminuria once in a child five years old, and twice in adults—one a young man in hospital, the other a young married woman; two cases were watched throughout.*

4. *Secondary inflammations.*—Among the most serious effects of Bright's disease, as being frequently the direct cause of death, must be mentioned the occurrence of inflammation in one or more of the serous cavities, or in the lungs. Of the different serous membranes, the pleura is most apt to be attacked, the pericardium next, and the peritoneum only shortly before death or when paracentesis has been performed. Meningitis is very rare, and perhaps, when it seems to be of renal origin, some other well-accredited cause would be found if carefully looked for. A case has, however, lately occurred under the writer's care (November, 1890) in which a young man died from acute nephritis and purulent meningitis with entire absence of traumatic or other local cause for the complication.†

The pathology of these secondary inflammations is not very clear; they are commonly attributed to the undepurated state of the blood. Whatever their explanation, we may associate with them the occurrence of inflammation of the skin and subcutaneous tissues in Bright's disease. These are often produced by irritation; hence the caution necessary in using acupuncture, and still more a permanent trocar, to draw off the serum. *Erythema leve* frequently occurs after pricking the legs, or severe erysipelas and sloughs may ensue.

But such dermatitis, both superficial and deep, may arise spontaneously—erythema of the face, the legs, or the genitals, an eruption like "lichen," or papular eczema, or diffuse scaly dermatitis; and, occasionally, universal desquamative inflammation resembling pityriasis rubra. The writer has most often observed a red papular eruption affecting the trunk and limbs, but sparing the face.

5. *Albuminuric retinitis.*—One of the most characteristic indications of Bright's disease is, in some cases, the presence of changes in the retinæ. These are said to have been first noticed *post mortem* by Türck in 1850; but the discovery of their importance in relation to kidney disease is assigned to Heymann, in 1856. They occur only in cases which are already chronic; in advanced stages of tubal nephritis after scarlet fever or during pregnancy; when the kidneys are cirrhotic; very seldom in cases of lardaceous disease, and probably only when it has long been associated with atrophic changes. Sometimes, however, the recognition of changes in the retinæ by means of the ophthalmoscope is the first thing which suggests that the patient is out of health. Their frequency is believed by Dr. Gowers to agree with the statement of Mr Eales, who found them in 28 out of 100 cases of chronic Bright's disease, or in about 2 of every 7 cases ('Birm. Med. Rev.,' 1880).

* On this point see some cases in children recorded by Sir Dyce Duckworth in the 'St Barth. Hosp. Rep.,' and by Dr Johnson in his 'Lectures' (1887); also a paper by Dr Goodhart in the 'Guy's Hospital Reports' for 1884 (vol. xlii, p. 197).

† I do not remember to have ever seen a purulent exudation in the pericardium, even in the meshes of lymph.—C. H. F. On the other hand, Dr Sutton, a pathologist of no less experience, mentions purulent pericarditis as common, and almost characteristic of Bright's disease. Empyema is decidedly rare, but peritonitis in renal cases is undoubtedly very often purulent.

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They vary in character in different instances, but they are commonly included under the name of "albuminuric retinitis," although this is not quite appropriate to all the morbid conditions.

The most common form of this lesion is, in fact, one which seems to be merely degenerative. It consists in the formation of *whitish spots*, sometimes close to the optic disc, sometimes elsewhere; near the macula lutea they often appear as fan-like streaks. They may be round dots, so minute as to be only visible by the direct method of examination; or they may be large, irregular patches, which equal the disc in size, and which may coalesce into large areas round it. A less intense diffuse opacity often extends over more or less of the retina. Associated with the white spots, or occurring independently of them, *hæmorrhages* are very frequently observed. These lie, for the most part, in the nerve-fibre layer of the retina; and they therefore are often "flame-shaped" (to use Dr Gowers' expression), following the radiating course of the fibres. They may also run by the side of and parallel to the vessels. When they are large they may be irregular in shape, or may penetrate into the deeper layers of the retinæ. In some cases, again, *optic neuritis* may be the most conspicuous retinal change. If no white spots are discoverable, the appearance will then be identical with that which is so commonly produced by intracranial disease, and it may ultimately run on to atrophy in exactly the same manner. Lastly, there may be a general *œdema* of the retina, with complete obscuration of the disc. The arteries are narrow, and to a great extent concealed; the veins distended and tortuous. There are always many hæmorrhages, forming large streaks in the course of the nerve-fibres. White spots are commonly numerous, large, rounded, and soft-edged. Dr Gowers, from whom the above description is taken, says that this form of albuminuric retinitis is confined to cases of severe and rapidly fatal Bright's disease, so that there is rarely time for it to subside or to pass into an atrophic stage.

Anatomically the white spots depend upon a degenerative change in the nerve-fibres, which become greatly thickened, varicose, and filled with fat-globules; "compound granule-masses, too, appear in large numbers." The vertical fibres of Müller become swollen and fatty. The fan-like distribution of the spots near the macula lutea is said to depend upon the peculiar arrangement of these fibres in that position; they radiate from the *fovea centralis*, and their direction is somewhat oblique. The diffuse opacity is due to œdema, which gives rise, in hardened preparations, to the appearance of cavities, separating the retinal elements from one another. Dr Gowers' drawings of microscopical sections show how greatly some of the layers (including even the layer of rods and cones) may be thickened where there is a white spot.

With regard to the *pathology* of these retinal changes but little is yet understood. Traube insisted on the fact that they scarcely ever occur except in cases in which cardiac hypertrophy is already present, as showing that this was mainly concerned in producing them; and Cohnheim upheld the same opinion. But probably the material fact is rather that albuminuric retinitis requires a considerable time for its development, so that before it appears the heart is almost certain to become enlarged. Dr Gowers draws attention to the small size of the retinal arteries, which in some cases of chronic Bright's disease are seen with the ophthalmoscope to be not more than one half or even one third of the size of the veins; but he regards this as the result of a vital contraction of their coats, and not of

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any organic changes in them. Mr Brailey, however, has shown that these vessels become affected with an *endarteritis obliterans*, exactly like that which we shall presently find to occur in the arterioles of the kidneys and of other parts of the body. Dr Gowers describes and figures irregular dilatations of the capillaries, with increase of the nuclei in their walls; he thinks it probable that such dilatations often lead to hæmorrhages. As for the optic neuritis, which we have seen to be sometimes the main ophthalmoscopic change, he has observed this especially when there have been conspicuous symptoms of cerebral disturbance, such as intense headache, delirium, and convulsions. He is therefore disposed to regard them as the cause of its predominance over the other retinal lesions in the cases in question.

Albuminuric retinitis, in its less intense forms, may be altogether unaccompanied with subjective symptoms. Or there may be more or less marked amblyopia, which may cause the patient to seek professional advice. Voelcker speaks of cases in which transitory darkening of the field of vision occurs from time to time during excitement or on exertion. Even in the most severe forms of albuminuric retinitis it rarely happens that central vision is lost, or that complete blindness follows. It is to be observed, too, that the occurrence of defective sight in a case of Bright's disease may depend upon very different causes. Wagner remarks that, altogether apart from the presence of retinal changes, it is not uncommon for hypermetropic patients whose general health is much affected to complain of impairment of vision which is really due to failure of accommodation. Such amaurosis is often an effect of uræmia.

It must not be supposed that any retinal changes are in themselves conclusive of the existence of Bright's disease. Dr Gowers relates a case in which a chlorotic girl became affected with what seemed to be an idiopathic neuro-retinitis, which ultimately left appearances undistinguishable from those of albuminuric retinitis. And sometimes there are difficulties in the diagnosis of the retinal effects of renal from those of cerebral disease. When the white spots are very small, and limited to the region of the macula lutea, they require careful looking for, and dilatation of the pupil by atropine is often necessary to enable one to make sure of not missing them.

As a rule, when albuminuric retinitis has once developed itself, it persists until the patient's death. The exact appearances, however, vary from time to time: hæmorrhages may disappear and fresh ones may form; even the white spots may subside, though Dr Gowers says that is very rarely the case with those that surround the macula lutea. It is in the Bright's disease associated with pregnancy that there is most ground for hoping for a permanent recovery from the retinal affection. The free use of purgatives is believed to favour its subsidence, and to diminish the tendency to recurrence.

6. *Cardio-vascular changes: hypertrophy of the heart, and thickening of the arteries.*—In his commentary on the first hundred cases collected of autopsies on the disease afterwards called by his name, Dr Bright remarked ('Guy's Hosp. Rep.,' vol. i, p. 396), "The deviations from health in the heart are well worthy of observation; they have been so frequent as to show a most important and intimate connection with the disease of which we are treating." Excluding cases of valvular disease, in thirty-four there was

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marked hypertrophy, generally affecting the left ventricle. Since that date (1837) clinical teachers at Guy's Hospital—Barlow, Rees, and their successors—have never failed to insist upon the peculiar characters of the renal pulse as "hard," "wiry," "resisting," or "incompressible," or (to use a more modern expression) upon the *increase of arterial tension.**

The hypertrophy of the left ventricle can generally be detected during life by displacement of the apex-beat outwards, by its heaving character, and by alteration in the first sound—often a mere weakening of it without any change of quality, sometimes a prolongation or reduplication, and occasionally a murmur. That this is the result and not the cause of the high arterial pressure seems shown by the fact that in many cases the above signs are replaced by those of dilatation: a short snapping impulse, and in many cases a decided murmur—apical in seat, systolic in rhythm, and, except by its being inaudible in the axilla, not to be distinguished from that of primary mitral regurgitation. After death the left ventricle is found dilated, without valvular lesions.

There can be little doubt that this dilatation of the systemic ventricle is like the hypertrophy which usually precedes it, the immediate result of abnormal blood-pressure in the arteries, and comparable to the hypertrophy and dilatation which result on the right side of the heart from obstruction in the pulmonary circulation.

Bright himself proposed two causes of the ventricular hypertrophy: "That the altered character of the blood [conveyed to the heart in the coronary arteries] affords irregular and unwonted stimulus to the organ immediately; or that it so affects the minute [arterial?] and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system" ('Guy's Hosp. Rep.,' vol. i, p. 396). Each of these explanations has been since expanded into various subordinate theories.

The latter seems to be the more satisfactory. Apart from the results given above, we have no knowledge of alteration in the blood acting as a stimulus to overgrowth; and why should not the same stimulus produce hypertrophy of the diaphragm and other muscles?

But the question remains, what is the nature of the obstruction in the systemic vessels which raises the blood-pressure?

In the first place, hypertrophy of the heart occurs in acute as well as in chronic cases of morbus Brightii.† It may also develop when the kidneys have become atrophied as the result of hydronephrosis, or of some other affection of the renal pelvis, as in cases recorded by Cohnheim. In association with the lardaceous change, however, it is not seen unless the renal cortex has become also affected with advanced tubal nephritis. It is comparatively slight when chronic Bright's disease is complicated by phthisis, cancer, or some other wasting disease, and in those who are very old.

Evidently, therefore, no explanation of the occurrence of cardiac hypertrophy can be valid unless it is applicable to both the principal forms of chronic Bright's disease. The *extent* to which the heart becomes enlarged differs in different cases—partly according to their duration; and it is far

* This characteristic feature of most chronic and some acute cases of renal disease was first observed abroad by Traube.

† This fact was ascertained by Dr Galabin from cases in Guy's Hospital between 1868 and 1872; and Dr Goodhart's further experience of ten years (1873–82) confirms the conclusion ('Guy's Hosp. Rep.,' vol. xliii, p. 104). The left ventricle was hypertrophied in 109 cases, and normal in only 25.

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greater in renal cirrhosis than in any other form. Thus, whereas in the earlier stages of tubal nephritis its weight may attain fifteen or sixteen ounces, and in the latest granular and atrophic stage of that affection seventeen or possibly twenty-one ounces, there are some instances of (primary) renal cirrhosis in which it reaches twenty-three, twenty-four, twenty-five, or even twenty-eight ounces. Compare the tables of the weight of the heart found by Dr Goodhart in 188 cases of chronic parenchymatous nephritis, in 329 cases of cirrhosis ('Guy's Hosp. Rep.,' xliii, pp. 104, 109, 111), and in 146 cases of lardaceous disease.

The chamber chiefly affected is the left ventricle, the walls of which (and also the papillary columns of the mitral valve) become extraordinarily thick and fleshy, their substance being made up of muscular fibres of perfectly normal appearance. Sometimes the cavity is of normal size, sometimes more or less dilated. In a good many cases the right ventricle also is somewhat enlarged. This generally indicates that the left ventricle has not been able to maintain the circulation efficiently, or that pulmonary obstruction has arisen from bronchitis, œdema of lung, or some other cause. But to some extent it is inevitable that the right ventricle should share in the process of enlargement, especially when the left ventricle becomes very greatly increased in size.*

It is important to observe that the state of the left ventricle is one of real, not only apparent, hypertrophy. In some cases fibrous tissue is mixed with the muscular overgrowth (cf. p. 44), but in the most characteristic examples this is not the case.

With regard to the exact character of the changes that take place in the *arteries* there are extraordinary discrepancies in the statements of different observers. In fact, among the pathologists who have especially studied this question within the last few years hardly any two entirely agree in their descriptions. That in middle-aged or old persons affected with Bright's disease the lesions commonly termed atheromatous are often found in the aorta, in the cerebral arteries, and in other arteries of large or medium size, was well known to Bright himself, and also to Wilks, Dickinson, and others who at different times wrote on the subject. But no great importance seemed to attach to this circumstance on account of the frequency with which atheroma is seen in those who are advancing in years. It was Dr George Johnson who, in the 'Med.-Chir. Trans.' for 1868, pointed out that the arterioles, not only in the kidneys, but also in the subcutaneous and submucous tissues, in the muscles, and in the pia mater of the brain, become remarkably thickened; and he attributed this change mainly to an hypertrophy of the muscular fibres in their walls. Four years later Sir William Gull and Dr Sutton read before the Royal Medical and Chirurgical Society a paper in which they declared the thickening to be the result of a hyaline fibroid formation, partly outside the muscular layer, in the *tunica adventitia*, partly in the *intima*, the muscular layer itself being often rather atrophied than hypertrophied, and the nuclei of its fibres degenerated. In the course of the discussion which followed, the "hyaline" appearance described by these observers was shown to be due to the action of the acidulated glycerine in

* Traube asserted ('Ges. Abh.,' iii, p. 239) that he had been able clinically to determine the presence of hypertrophy of the heart within four weeks from the commencement of an acute renal affection. Similar cases were recorded by the late Dr Peacock, and since by Dr Stone in his Croonian Lectures (1879), and Dr Goodhart ('Path. Trans.,' xxx). A case of recession of an hypertrophied ventricle has been recorded by Sir William Roberts ('Glasgow Med. Journ.,' 1884).

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which the preparations were placed for examination. Leyden among foreign writers alone, lays stress upon this character; his figures ('Ztschrift. f. klin. Med.,' 1880) represent circumscribed glassy patches lying in the coats of the thickened vessels, and are very unlike those of Gull and Sutton. Few German pathologists, however, confirm Dr Johnson's statements as to the existence of muscular hypertrophy. Ewald, indeed, admits ('Virch. Arch.,' 1877) that there is an increase in the thickness of the muscular coat; but this, he says, is due to an enlargement, not to a multiplication of the fibres. But according to Sotnitschewsky ('Virch. Arch.,' 1880), when this coat is found thickened, it is as the result of increased fibrous tissue. Lastly, all recent writers agree in considering that an almost invariable condition affecting the intima is that which was originally named by Friedländer *arteritis obliterans*.

It seems impossible to reconcile these varying descriptions except by supposing that the affection of the arterioles in Bright's disease differs in different cases, perhaps even in the same case in different tissues, or at different periods in its course. Another explanation may be that this change is most constant in the chronic atrophic form of morbus Brightii, which is undoubtedly less common in Germany, and perhaps on the Continent generally, than in England. But after all, as we shall presently see, it is only with reference to the exclusive theory proposed by Dr Johnson that the exact character of the arterial change is of primary importance.

Apart from the anatomical question, let us now see what light is thrown on the general problem by physiological investigation.

We have seen that one of the most marked clinical features of Bright's disease is a state of high pressure or tension in the arteries, recognised in the hard or incompressible pulse (p. 13).

Since the invention of the sphygmograph this can be estimated much more accurately than before. The first point to be noted in all such tracings is that the pressure applied to the artery while they are being taken is far greater than that which brings out the characters of the pulse most distinctly in health: as registered by the late Dr Mahomed's spiral eccentric it was from four to six ounces, instead of being from one and a half to three ounces. The next points are the breadth or "bluntness" of the tidal wave and the distance of the dicrotic notch from the upstroke; these indicate prolongation of the ventricular systole. The last point is the distance of the bottom of the dicrotic notch above the base line (cf. p. 4).

If next we consider the pulse as it is felt by the finger, we find the following characters. First, it is *persistent*: even in the intervals between the cardiac beats the artery feels full. One might imagine that its coats were thickened, but on emptying it by pressure above one finds that it can no longer be rolled beneath the finger, as a thickened vessel would be. Next, it is *long*, not falling away as soon as it has reached the finger, but pushing or laboured in character. Lastly, it is *hard* or *incompressible*, requiring much force to overcome it. Both the last characters are really indications of the state of the left ventricle which is associated with high arterial tension—the slow prolonged systole and the hypertrophy of the ventricular walls.

Examination of the heart often yields valuable corroborative evidence. There may be an enlarged area of percussion-dulness, displacement of the apex outwards, and a heaving, laboured impulse. It is, however, important to be aware of the fact that these signs are not seldom absent, even when there is

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no obvious emphysema or other disease of the left lung to account for it. Thus in cases of renal cirrhosis, when perhaps the patient has been admitted with cerebral hæmorrhage, we have again and again failed to detect any indication of cardiac hypertrophy, although at the autopsy a day or two later the heart has been found enormously enlarged. It is particularly in those cases in which the hypertrophy is unattended with any dilatation that the difficulty arises.

On auscultation the first sound may be faint, or dull and prolonged, or reduplicated, or even replaced by a murmur.* A more characteristic auscultatory sign, and one which is the direct result of the increased arterial tension, is the loud, ringing, or metallic quality of the (aortic) second sound, as it is heard at the base of the heart, or over the carotid artery. Occasionally, also, a diastolic shock can be felt by the hand placed over the cardiac region.

The cause of the systemic obstruction which causes the high arterial tension in Bright's disease has been much debated, and is still unsettled.

Dr George Johnson (1868), relying upon his observations as to the existence of hypertrophy in the muscular walls of the arterioles, propounded the theory that these vessels exert a "stopcock" function, resisting the passage into the capillaries of blood which, as the result of defective elimination by the kidneys, is noxious to the tissues. In fact, he imagined an active antagonism between the heart and the arterioles, as the result of which they each become hypertrophied.

The late Dr Mahomed reverted to the view that the obstruction is in the capillaries, and this view is ably supported by Dr Saundby, of Birmingham; but, believing that the high tension in the arteries precedes the development of renal disease, their notion is that the supposed impurity of the blood is due, not to imperfect excretory activity on the part of the kidneys, but rather to over-eating and over-drinking, by which it becomes charged with injurious matters. The objection to all such theories, however, is that there is no proof whatever that any changes in the circulating fluid are capable of retarding its flow through the capillaries. Physiologists admit no causes of such retardation except alterations in the capillary walls themselves; and these only affect the circulation locally, as in the case of inflammation. And although the injection of urea into the blood is capable of increasing the arterial pressure in animals, yet this is only when the quantity injected is so large as to render the experiment inapplicable to human pathology.

Such considerations have led some German pathologists to look elsewhere for an explanation of the cardio-vascular changes in Bright's disease, and of the high arterial tension which is so closely associated with them. Traube, in 1856, suggested that destruction of the renal parenchyma would have two results, each of which might tend to augment the pressure in the arteries; one being the accumulation of water in the blood from impairment of the secretory activity of the kidneys, the other the diminution in the amount of blood flowing from the arterial into the venous system as a consequence of obliteration of vessels in those organs. But the first of these two conditions does not exist in the most marked cases of high pressure, which are accompanied by increased, not impaired, flow of urine; and the second certainly seems altogether inadequate to produce any marked effect. More-

* Mahomed also maintained ('Guy's Hosp. Rep.,' 1879) that it is sometimes preceded by a short sound resembling the presystolic murmur of mitral stenosis. Surely this is the same sign as what is called reduplication of the first sound?

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over, hypertrophy of the heart ought, on this view, to follow on amputation of the thigh or of two limbs more certainly than on the gradual diminution of the area of the renal circulation.

Dr Hamilton, of Aberdeen, has put forward the ingenious theory that the systemic obstruction is due to diminished specific gravity of the blood; this causes the red discs to mingle in the layer of leucocytes, which normally lie close to the vessel-wall, and thus friction is increased. Of the physical fact there is no doubt, but why should not the same effect occur in other forms of anæmia?

Cohnheim put this part of the question in a new light. He gave reasons for thinking that the activity of the circulation through the kidneys at any moment—in other words, the state of the smaller renal arteries as regards contraction or dilatation—depends not (as in the case of the tissues generally) upon the need of those organs for blood, but solely upon the amount of material for the urinary secretion that the circulatory fluid happens then to contain. This suggestion has bearings upon the development of hypertrophy in one kidney when the other has been entirely destroyed. But another consequence deducible from it is that when parts of both kidneys have undergone atrophy, the blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact. In order, however, for such a quantity of blood to pass through the restricted capillary area now open to it, an excessive pressure is obviously necessary. This can be brought to bear only by increased energy in the pulsations of the left ventricle, combined with the maintenance of a corresponding resistance in all other districts of the arterial system. And so one can account at once for the high arterial pressure, and for the consequent cardio-vascular changes.

There is not any novelty in the idea which forms the basis of this theory, namely, that the hypertrophy of the heart in Bright's disease is a *compensatory* change, enabling the organism to withstand the consequences of the disease. But what seems not to have been clearly perceived is that a hypertrophied heart cannot effect this result unless it is supported by a resistance in the systemic arterioles proportioned to that which exists in the kidneys. Surely the fact that the secretion of urine is maintained or increased shows that considerable compensatory changes must have taken place in the systemic arteries generally, as well as in the heart. One advantage of such an explanation of the high arterial tension of Bright's disease is that it enables us to see that the exact means by which it is kept up may vary in different cases and at dfferent periods of the same case. At an early stage of the parenchymatous affection it can only be by contraction of the muscular walls of the arterioles, such as Dr Gowers believes that he has seen in the retina; and even in the chronic form of Bright's disease this must still play an important part, at least in cases in which a state of low pressure and dicrotism can be induced by the inhalation of nitrite of amyl, as has been shown by Dr Broadbent: but the presence of *endarteritis obliterans* is no doubt also concerned in producing and maintaining the permanent peripheral resistance which leads to the high arterial tension.

Moreover, this view fits in with the fact that cardio-vascular changes, like those that occur in Bright's disease, may likewise accompany atrophy of the kidneys from hydronephrosis.

Lastly, it can be brought into accord with Mahomed's observation, that

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there are some young persons with presumably normal kidneys in whom the arterial tension is constantly high, notwithstanding that they seem in perfect health. Assuming that the urine in such cases is natural in quality and in quantity, one can but suppose that the kidneys are for some reason incapable of secreting such urine, except under excessive pressure. The condition would thus be comparable with the "renal inadequacy" described by Sir Andrew Clark. A very important question is whether it is to be regarded as a warning of the probable supervention at a later stage of Bright's disease, or of endarteritis and vascular changes. One of the points insisted on by Sir William Gull and Dr Sutton was, that the arterio-capillary fibrosis which they describe sometimes occurs independently of any affection of the kidneys.

Hæmorrhages.—It is doubtless as a more or less direct result of the high arterial tension of Bright's disease that the rupture of vessels in different situations is to be explained. Thus arise retinal hæmorrhages (p. 461), apoplexy, epistaxis, and hæmorrhage from the stomach and intestines. As regards epistaxis, Mahomed notes ('Guy's Hosp. Rep.,' 1881) the fact that even when the patient is much blanched by loss of blood the pressure in the arteries may still remain excessive.

7. *Uræmia.*—That in many cases the chief symptoms of chronic Bright's disease are cerebral, has been well known from an early period in its history; but there have been wide differences of opinion as to the mode of origin of such symptoms. These differences of opinion, however, have not prevented their being universally termed "uræmic," although the name uræmia (invented by Piorry) implies in strictness the acceptance of the theory that they depend upon an accumulation of urea in the blood, a theory which cannot now be accepted in that precise form.

a. The cerebral symptoms of acute uræmia vary in different cases. The most striking form consists in the occurrence of convulsive fits. Such "*epileptiform*" paroxysms are seen sometimes in patients who are already confined to bed with dropsy or suffering from other effects of Bright's disease; sometimes in those who are still engaged in their daily occupations, or who may even be apparently well. The onset may be either sudden, or preceded for a few hours or days by headache, drowsiness, vertigo, a strange fixed expression of the face, dragging pains in the extremities, or a transient rigidity of the face, or of the lower jaw, or of a limb. Nausea, again, and even vomiting, may be among the prodromal symptoms; or severe dyspnœa of some hours' duration. Wagner mentions that the pulse sometimes falls to 60 or even to 40 in the minute.

A description of uræmic paroxysms will be found in the chapter on epilepsy (vol. i, p. 752); for they are identical in every detail, even to the biting of the tongue, the foaming at the mouth, the involuntary discharge of urine and fæces, and the subsequent sleep or stupor, sometimes replaced by an attack of maniacal excitement. Wagner says that the pupils are generally dilated, seldom small; but at Guy's Hospital, from the time of Addison, it has been usual to describe them as being more often contracted or of the natural size; they usually retain their sensitiveness to light. The temperature may rise several degrees, reaching 102° or 104°, or even a higher point still—in one case 107°. During the coma which follows the convulsions it slowly falls to normal, or below; for some days afterwards it may remain as low as 94° or 95°. The pulse is commonly accelerated while the spasms continue; afterwards it returns to its natural rate, or may become slower

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still, remaining perhaps (as in a case of Wagner's) between 44 and 64 for the next fortnight. After the attack has ceased the patient is sometimes dull and depressed for some days. Hemiplegia has very rarely been observed; it might be expected to occur, as it sometimes does after true epilepsy.

In many cases, before the insensibility has passed off after one uræmic paroxysm, another sets in; and thus twenty or thirty may occur in succession, simulating the *status epilepticus* (vol. i, p. 753). The disease then is very likely to prove fatal. But even after a series of fits it is not uncommon for recovery to take place; the convulsions cease, and the patient regains consciousness, to the surprise of his friends. A single paroxysm seldom ends fatally; but in 1862 a woman, aged thirty, died in Guy's Hospital within seven minutes from the commencement of uræmic symptoms. She had just eaten her breakfast, when slight spasmodic movements of the arms suddenly set in. She became pale, and her lips and fingers livid; there was foaming at the mouth. The pupils were dilated. The heart at first continued to beat regularly, but its action very quickly ceased.

Sometimes, instead of a uræmic fit having the typical epileptiform character, its symptoms are of a different kind. In the 'Guy's Hospital Reports' for 1839, Addison described one variety as consisting in a "sudden attack of coma with stertor, or, in other words, *apoplexy*." Probably it is now a result of his teaching, that we at Guy's Hospital have for many years past been very cautious in diagnosing cerebral hæmorrhage in cases in which there was reason to suspect the existence of renal disease, notwithstanding the well-known frequency with which it occurs under such circumstances. Nevertheless, in the chapter on cerebral hæmorrhage, we found that our *post-mortem* room experience does not support the view that it is liable to be simulated by uræmia, at least when there have been no convulsions (cf. vol. i, p. 588).* Addison was of opinion that the two affections might be distinguished by the characters of the stertor that accompanied them; in uræmia, he said, the sound was more hissing, "as if produced by the air striking against the hard palate, or even the lips, rather than against the velum and the throat, as in ordinary apoplectic stertor." He also believed that the respiration was from the first much more hurried than in true apoplexy.

In certain instances uræmia may manifest itself by *delirium*, lasting for days together, or by *rigidity* of one or more of the limbs, or, according to Charcot, by *tremors* like those of paralysis agitans. In a case of Roberts's in which the paroxysms coincided with the catamenial periods, consciousness was not lost; "during the convulsions the patient knew the persons about her, and called loudly to be held fast." Bright, in the 'Guy's Hospital Reports' for 1840, related a case in which for two days before death there occurred a very distressing and almost incessant twitching of the muscles, which increased until the arms and the legs were forcibly drawn up and the face was distorted by the spasms, yet the faculties of the mind were perfect to the last.

In some cases uræmia shows itself by much slighter symptoms; by

* Roberts cites three uræmic apoplectiform cases, but each of them is open to criticism. One patient had had "a few drops" of laudanum given to him for diarrhœa just before the cerebral symptoms set in, so that it may be doubtful whether they were not due to the excessive action which even small doses of that drug are known to exert when the kidneys are diseased; in the second case epileptiform convulsions were present; in the third case there seems to have been no autopsy, so that the possibility of cerebral hæmorrhage is not excluded.

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transitory *trismus* perhaps, or frequently, as a premonitory symptom, by short attacks of *clonic spasm* in some of the facial muscles, or in those of the eyeballs, or of a limb; the patient retaining consciousness, or being at most a little confused or dull of intelligence.

Perhaps the most remarkable of all the effects of uræmia is *amaurosis*. This not infrequently occurs along with eclampsia; the patient, when he recovers consciousness, finds himself blind. Sometimes, according to Wagner, it precedes the convulsions. But more often it is the only symptom, except headache. It sets in suddenly, is bilateral, and is almost always complete, the patient having not the slightest perception of light. Dr Gowers says that the pupils generally still react to light; but Wagner says that they are sluggish, and that they may in some cases be altogether insensible and widely dilated. The ophthalmoscope does not show any change in the optic discs or retinæ.* This alarming affection rapidly subsides, so that the patient regains sight within twelve or twenty-four hours, or at the longest in the course of a few days. Wagner says that such a favourable termination may occur even when the pupils have lost their sensitiveness to light. The fact that, as a rule, the pupils react, seems to show that the seat of uræmic amaurosis must be above the corpora quadrigemina.

It is said that in some instances a transitory defect of hearing, or even complete deafness, has been observed as a sequela of a uræmic seizure.

b. In striking contrast with these varied forms of what may be termed acute uræmia are some which are described as chronic. The latter are not, like the former, always obviously cerebral in their character. The lungs or the digestive organs may appear to be the parts affected. But there is good reason to believe that even in such cases the starting-point of the symptoms is generally, if not always, in the brain.

The *cerebral symptoms* usually consist of headache, giddiness, or drowsiness, any of which may go on for weeks or months, either continuously or with intermissions. The patient's aspect is often remarkably dull and expressionless; he lies in bed, taking no notice of what goes on around him, and altogether indifferent to his own condition. Ultimately he may fall into complete stupor. A patient of the writer's recently died of tubal nephritis of about three months' standing, which was passing from the acute to the chronic stage, when he became increasingly dull and apathetic until at last he lay insensible, but without stertorous breathing, for several days before his death. Sometimes the general symptoms are like those of the typhoid state, the tongue being dry and brown, and sordes collecting upon the teeth and lips. Such cases might be mistaken for enteric fever.

In other cases the principal indication of chronic uræmia is *dyspnœa*. This is generally paroxysmal, and is apt to come on at night, like asthma. It may be expiratory, like asthma, or inspiratory, as though there were laryngeal stenosis; or, again, both inspiration and expiration may be free, but unnaturally hurried. The type of breathing named after Cheyne and Stokes is not infrequently observed in cases of chronic uræmia (cf. vol. i, p. 945).

Another sign of uræmia may be an intense *itching* of the skin. Sometimes patients go on scratching or rubbing themselves, even when they are so far unconscious that it is impossible to rouse them.

One of the most characteristic symptoms is *vomiting*. At first it may occur only in the morning when the stomach is empty. Afterwards it may

* Two instances, however, are cited by Dr Gowers, in which slight œdema of the discs is said to have been detected, which passed off with the amaurosis.

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take place whenever any food is taken, and become exceedingly intractable, continuing for weeks, or even for months. Urea may often be discovered in the matters rejected, and some have supposed the vomiting to be the result of its presence in the stomach. But in Voit's experiments upon animals it was found that urea ingested with the food did not set up vomiting at once, but only when there had been time for it to be absorbed into the blood, and to act upon the nervous centres. The vomited matters are sometimes alkaline, perhaps from some of the urea having been decomposed into carbonate of ammonia; but as a rule they are acid. Bartels has suggested that œdema of the walls of the stomach may be a cause of vomiting in Bright's disease; but such an affection is rarely observed in the deadhouse, and to suppose it capable of producing vomiting is a mere assumption.

Hiccough is not uncommon in association with other effects of uræmia, and Wagner mentions one instance of chronic Bright's disease in which hiccough and slight œdema of the lower limbs were the sole symptoms.

Diarrhœa is of rather frequent occurrence, and often accompanies vomiting. It sometimes seems to depend upon an inflammatory affection of the intestinal mucous membrane, which may be œdematous, or ecchymosed, or even in a state of severe "diphtheritic" inflammation, like that which is met with in dysentery. Occasionally its surface is covered with large leathery patches, or is extensively ulcerated. In cases of this kind the evacuations often contain blood, and mucus and pus in large quantity. There is some doubt as to whether the diarrhœa and enteritis of Bright's disease are strictly of uræmic origin. Cohnheim and other recent writers think that they are rather effects of local irritation from carbonate of ammonia produced by decomposition of urea in the intestine.

Prognosis.—Epileptiform convulsions and the other symptoms that have been grouped together under the name of acute uræmia may accompany any form of Bright's disease, and are very frequent in the nephritis of scarlet fever, cholera, and of pregnancy. They sometimes pass off; and it may be added that their occurrence is no certain proof that the renal disease which causes them is so severe or so advanced as to be incapable of recovery. On the other hand, the prolonged stupor, the typhoid symptoms, and the other phenomena of chronic uræmia almost always end fatally. They are never seen in the more acute forms of Bright's disease—as, for example, after scarlet fever, or in association with pregnancy—unless the so-called "cholera-typhoid" (vol. i, p. 232) is an instance to the contrary.

Theory of uræmia.—In discussing the cause of the symptoms that we have grouped together under the name of uræmia, we must, in the first place, consider whether those which are obviously cerebral may possibly depend on actual lesions of the nervous centres. Some years ago Traube supported the theory, originally propounded by Rees, that they were due to œdema, combined with anæmia of the brain. The œdema he supposed to be brought about by the action of a hypertrophied heart upon the smaller intracranial blood-vessels, assisted by a watery condition of the blood itself; but every part of his theory has since been shown to be untenable. In many instances the brain is found after death to be perfectly dry; and when it is œdematous, Bartels is probably right in thinking that this is an effect, rather than the cause, of any convulsive seizures that may have occurred, unless, indeed, it is a mere accidental result of wasting of the brain, as is doubtless very often the case. Sometimes minute spots of hæmorrhage are found in greater or less numbers in the substance of the brain. In two instances of Bright's

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disease, both with large white kidneys, the pons and the bulb were found full of such capillary hæmorrhages. It seems most likely that they also are produced by the disturbance of the intracranial circulation, which cannot but accompany the uræmic paroxysm; their occurrence is far too exceptional to admit of their being regarded as its cause.

We are therefore driven to what may be termed the chemical theories of uræmia. There are, however, considerable difficulties in accepting the most natural supposition, namely, that it depends upon the retention in the blood of urea which the kidneys have failed to excrete. Frerichs consequently suggested in 1851 that the poisonous agent was really carbonate of ammonia formed in the blood by decomposition of urea. Subsequently Treitz amended the hypothesis by supposing that the carbonate of ammonia was produced, not in the blood, but in the stomach and intestine, a vicarious excretion of urea into the alimentary canal first taking place, and the carbonate of ammonia being afterwards reabsorbed into the blood. But this theory of "ammoniæmia," though at one time it was widely adopted in Germany, is now universally abandoned. On the one hand, it has been shown that though carbonate of ammonia, injected into the blood of animals, causes symptoms somewhat like those of uræmia, the resemblance is, after all, incomplete, and that many other salts produce like effects. On the other hand, many observers fail to detect carbonate of ammonia in the blood of uræmic patients, and there appears to be no doubt whatever that, if present at all, it is not in sufficient quantity to account for the effects attributed to it. One clinical point which Frerichs adduced in support of his view was that by holding a glass rod moistened with hydrochloric acid near the mouth of a uræmic patient the presence of carbonate of ammonia could be recognised in the expired air by the white fumes of chloride of ammonium that were formed. Schottin, however, showed that in many uræmic patients this test completely failed, whereas it often succeeded in other patients who lay in a typhoid state from whatever cause, the carbonate of ammonia being set free from dried secretions within the mouth, and not exhaled from the lungs.

Some recent observers have been fain to fall back upon the older and simpler theory, which supposes that the symptoms of uræmia are directly due to the presence in the blood of excess of urea.

The objections which had caused this view to be discarded were drawn partly from experiments upon animals, partly from clinical observations of Bright's disease. Experiments had seemed to show that urea, and even urine itself, could be introduced in large quantity into the blood of animals without giving rise to any ill effects. Voit and Oertel, however, found ('Ztschft. f. Biol.,' 1868) that although urea when added to the food of a dog produces no symptoms so long as it can be freely excreted by the kidneys, yet, if the animal is not allowed to drink any water, symptoms like those of uræmia appear.

The clinical objections to what may be termed the "urea" theory of uræmia were taken by Frerichs from a work which had shortly before been published by Owen Rees; they were briefly that the occurrence and the severity of the paroxysms bore no necessary relation to the quantity of urine secreted, and that the blood was sometimes loaded with urea without any such symptoms appearing.

In fact, the characters of the urine in cases of Bright's disease at the time when uræmia develops itself differ in different cases. As a rule, the renal

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secretion is very much diminished in quantity for several days before the symptoms set in; it may even be completely suppressed. But sometimes there is a normal flow of urine, although it contains much less than the due amount of urea; and sometimes the quantity of urine is above normal, as in a case of Wagner's, in which the patient for three successive days passed seventy ounces daily. In that instance, however, its specific gravity ranged only from 1006 to 1010, so that after all the excretion of urea and of the other solid constituents of the urine was probably defective.*

Certain observations made by Fleischer ('Deutsches Arch.,' xxix, 1881) have thrown some fresh light upon this difficult question of the relation of uræmic symptoms to the excreting action of the kidneys. He instituted careful analyses of the urine passed by persons affected with Bright's disease, comparing them in each instance with analyses of the urine of healthy persons placed under exactly the same conditions as regards diet. He found, as a rule, that the amount of urea excreted by those who had Bright's disease was much diminished; but when uræmia set in, the amount of urea became increased far beyond the normal, either on the day of the seizure or else a day or two later. The explanation which he suggests is that when the accumulation of urea (and of other urinary constituents) reaches a point at which the system ceases to be indifferent to their presence, so that uræmia results, they at the same time stimulate the heart and the kidneys to expel them. Henceforth, therefore, it must be borne in mind that the fact of an abundant elimination of urine taking place during or after a uræmic fit, is no proof that it may not previously have been deficient, and this deficiency the cause of the attack.

The difficulty remains, that although systematic analyses of the urine may show the amount of urea excreted to be only 200 or 150 grains daily, yet that in many other cases it is quite as low, without any uræmic symptoms arising. Cases also occur in which the urine is more or less completely suppressed for a week or longer before uræmia develops itself. We can only say that such facts are in accordance with clinical experience in general. The effective operation of all "causes" of disease is liable to be interfered with by the patient's power of resistance and other unknown conditions.†

All doubts as to the occurrence of imperfect elimination by the kidneys in uræmia might be set aside if it were known that the blood in this state invariably contains more urea than in health. The earliest analyses of the blood in chronic Bright's disease seem to have been made by Dr Guy Babington, who states that in a case under the care of Dr Bright himself there was as much urea in the circulating blood as in the urine, a thousand grains of blood yielding fifteen grains of urea! Later observers have found much smaller quantities than this. Wagner says that, instead of the

* The man had not been œdematous, and there was therefore no reason for supposing that a reabsorption of dropsical fluid had anything to do with the large amount of urine poured out by his kidneys. Wagner, however, remarks that the tissues of the dead body may be found to be distinctly œdematous, when there had been no clinical evidence of it It seems, therefore, not impossible that the absorption into the blood of such a latent accumulation of fluid may sometimes be the real cause of an excessive flow of urine previous to the development of uræmia, especially as the subsidence of dropsy is known to be frequently followed by uræmic symptoms.

† It is remarkable that uræmia is seldom met with in persons advanced in years; perhaps this suggests that a "predisposition" on the part of young subjects is one factor in its ætiology.—C. H. F.

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normal proportion of 0·16 or 0·2 part per 1000, there may be 0·4 or 0·6 part, or more. He further cites an observation of Hoppe-Seyler's, who, in the blood-serum of a cholera patient with uræmia, discovered 1·27 parts of urea per 1000. But elsewhere he says that the quantity of urea in the blood has several times been found to be small, "so that an overloading of the blood with this substance certainly cannot be in all cases the cause of uræmia." But before we accept this important conclusion, we ought to know exactly at what period of the disease the analyses have been made in which no excess of urea has been detected. If Fleischer's observations are correct, it seems quite possible that in the course of a uræmic seizure, or afterwards, the blood might contain no excess of urea, and yet that a great excess might previously have been present, and have given rise to the attack.

Urea may also be discovered in considerable quantity in the various secretions. Its presence in the gastric and intestinal contents has been already incidentally mentioned. In one case in which there were bronchitis and extensive pneumonia, Fleischer found it in the sputum to the amount of about thirty grains in the thirty-seven ounces expectorated during twenty-four hours.

It is a curious fact that in some uræmic patients urea is excreted by the skin. This seems only to occur shortly before death, and scarcely ever without the urine being completely suppressed. Schottin first observed it in 1862 in cholera patients. The 'Guy's Hospital Reports' for 1874 contain a report, by Dr Frederick Taylor, of a patient with Bright's disease, in whom, two days before death, there appeared on the face, neck, and hands white masses which adhered pretty firmly, and which, when removed, were found to be irregularly shaped, with crystalline spiculæ and prisms projecting from them. They yielded the several reactions of urea. The patient's face is described as having looked as though flour had been sprinkled over it. In some other cases the appearance is said to have been just as though a lather of soap had been allowed to dry on the surface, or as though the beard were frosted.

If neither urea as such nor ammonia-carbonate be the poison which causes the symptoms of "uræmia," may we look to lithic acid, hippurates, kreatinin, or any other normal constituents of the urine, as accumulating in the blood when the kidneys are diseased and producing the symptoms in question?*

Voit put forward the hypothesis ('Zeitschft. f. Biol.,' 1868) that uræmia is not due to the poisonous action of any one ingredient of the urine—whether urea, uric acid, kreatinin, or other extractives, but was inclined to attribute a considerable share to the salts of potass. He believed that it may be produced by "any substance which is not a normal constituent of the body if it accumulates in large quantities and is not eliminated."

This vague statement, however, is opposed to one clinical fact, for a clear recognition of which we are indebted to Roberts, and which is of the highest importance, both to the physiologist and to the physician. It is that symptoms altogether unlike those of uræmia, and holding a completely different course towards a fatal issue, are presented by cases in which the failure to eliminate urea and the other ingredients of the urine is absolute, but in which the cause of the suppression of the renal secretion is not an

* Professor Sée believes that uræmia from urea and from carbonate of ammonia are both pathological conditions, and that they can be diagnosed from one another during life, but the evidence for each of these beliefs is defective.

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affection of the cortex of the kidneys, but obstruction of the ureters. These clinical symptoms are met with in what is called "obstructive suppression of urine (*v. infra*, p. 524). The absence of uræmia in these cases seems clearly to show that where there is healthy kidney substance, with an active circulation through it, the waste products which should be excreted in the urine undergo some chemical changes that render them incapable of producing uræmia, notwithstanding that they are retained in the body. Nor do the results of experiments upon the lower animals appear to be inconsistent with this view. Obstructive suppression is, of course, easily produced by ligature of the ureters; the effects of this operation are spoken of as identical with uræmia, but it is perhaps not to be expected that distinctions between different groups of symptoms should be so obvious in animals as in man. On the other hand, it is by no means easy to bring about a non-obstructive suppression in such a way as to afford a satisfactory comparison. There are two ways in which one ought to be able to annul the activity of the secreting substance of the kidneys; one is by ligaturing the renal arteries, the other by excising the kidneys. Now, ligature of the renal arteries has been shown by Hermann ('Sitzungsbericht d. Wien. Acad.,' 1861) not to be effectual in arresting the blood-supply to the kidneys, which may continue to pour forth urine. And excision of the kidneys is an exceedingly severe operation, very apt to produce vomiting and other ill-effects that make it unfair to contrast it with so simple a procedure as ligature of the ureters.*

Some maintain that the cause of uræmia is the accumulation in the blood of products intermediate between urea (or uric acid) and the albuminous substances from which it has its origin: such products as kreatin and kreatinin, hypoxanthin and xanthin, or leucin (amido-caproic acid), or aspartic (amido-succinic) acid and tyrosin.

In conclusion, there seems to be no doubt that uræmia is produced by the poisonous action upon the nervous centres of materials accumulated in the blood as the result of defective excretion by the kidneys; but it is still uncertain whether this action is excited by one substance or by more.

In many cases the actual outbreak of convulsions is immediately due to some obvious disturbance of the balance of excretory functions, which may be supposed to have been previously unstable. Thus Bartels relates a striking instance in which the production of profuse sweating in a dropsical patient by a hot bath, followed by hot packing, at once brought about a series of uræmic attacks; next day the dropsy was gone. Sometimes, perhaps, the immediate cause of uræmic symptoms is the sudden failure of the heart to keep up an active circulation through the renal vessels, so that the excretory function of the kidneys, which may for a long time have been more or less impaired, now becomes altogether ineffectual.

The remarkable symptoms, the explanation of which we have been discussing—albuminuria, dropsy, lesions of the optic nerve and retina, cardiac hypertrophy and arterial tension, and the most remarkable and obscure of all, uræmia—are common to all the varieties of Bright's disease, although in very different degrees.

We now proceed to describe the distinguishing and particular clinical

* After excision of the kidneys the quantity of urea which is found in the blood is not so great as after ligature of the ureters, but, as Salkowski points out, the cause of this may very well be the vomiting that follows the former operation.

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features which belong to the two great types of Bright's disease, and to their several stages and modifications, as enumerated on p. 443.

I. TUBAL NEPHRITIS.*—As remarked before, this name is adopted as the most suitable, but not as implying that the pathological changes in this form of Bright's disease are strictly limited to the secreting cells of the renal cortex. On the contrary, we shall presently find that the glomeruli, and even the connective tissue, are in many cases markedly affected.

Ætiology.—Parenchymatous nephritis is often due to obvious causes. It may be definitely traceable to *cold*. Bartels cites three well-marked examples of this: one is that of a patient who was taken ill as the direct result of going to sleep half undressed by an open window on a winter's night, after having spent the evening in dancing; another is that of a man who, while perspiring freely, left his smithy and went out into the open air in his shirt, getting wet through with a sleety rain; the third is that of a person who was skating, when he broke through the ice, and had much difficulty in extricating himself.

Very many cases arise from *scarlatina;* and some in which no definite cause can be found may be really due to a latent attack of scarlet fever during childhood. Cholera, erysipelas, enteric fever, smallpox, measles—all more or less frequently give rise to albuminuria, and to some degree of nephritis, but it seems doubtful whether the renal affection in any one of these diseases leads to permanent albuminuria, to dropsy, uræmia, or any of the symptoms above described, or, lastly, to the anatomical lesions characteristic of any form.

In women, *pregnancy* is often the cause, as the late Dr Lever showed many years ago, especially in primiparæ, and, above all, when there are twins. Sometimes the disease appears to recur in successive pregnancies. It generally manifests itself during the months of gestation. How it is brought about is not at all clear. It certainly is not due to pressure by the gravid womb upon the renal veins; and the most plausible view seems to be that it results from the kidneys having extra work thrown upon them in the elimination of effete matters. Its onset is often insidious, but those writers who separate acute from chronic Bright's disease include it under the former head.

In countries in which *ague* is endemic, it is said to be a frequent cause of Bright's disease.

Among the causes that are more or less slow in their action the writer would be inclined to place indulgence in *alcohol* as a very important one, and this was the original opinion of Bright, although it is disputed by later observers. For example, a solicitor, usually moderate in his habits, acquired temporary albuminuria as the apparent result of drinking sherry in large quantities to induce sleep, at a time when he had a great trouble weighing upon him. Another patient's urine was for many years albuminous during habitual excess in stimulants; he changed his ways, and two years later no evidence of any renal affection could be discovered, and he seemed to have regained his usual health.

Hereditary predisposition does not seem to be at all frequently observed, though in so common a disease; but Dr Dickinson has put on record one

* *Synonyms.*—Parenchymatous nephritis—Tubular nephritis—Desquamative nephritis Néphrite albumineuse—Croupöse nephritis—Acute and chronic epithelial catarrh of the kidney.

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remarkable case, in which it appeared in several generations of the same family ('Path. Trans.,' 1889, p. 144).

Dr Johnson has suggested inhalation of sewer gas as an occasional cause of acute nephritis; and the list of causes would be incomplete if we could not add that the urine in some cases contains streptococci, which disappear when the chronic stage is reached.

Anatomy.—The appearances presented differ considerably, even at the same period.

(1) *The large red and speckled kidney.*—Should the disease prove fatal during the first two or three months, the kidneys are sometimes found of nearly natural size, but more often enlarged, and occasionally of twice their natural weight. It may be noticed that they are rounded in shape, so as to stretch their capsule, which is not thickened and can be as easily stripped off as from a normal kidney. The colour of the surface is usually dull red, but when incised the medulla presents a much deeper reddish-purple tint than the cortex. If there has been complete suppression of urine, or if death has been due to convulsions attended with great pulmonary congestion, the kidneys are often found gorged with blood, and of a dark chocolate colour (see Dr Dickinson's 5th plate). In most cases red points are scattered over the cortex; some of them are blood-filled glomeruli, others are punctiform hæmorrhages.

In some instances, particularly among those attributable to scarlet fever, the kidneys show scarcely any deviation from their natural appearance; but often the scarlatinal kidney is swollen, bright red, and dripping with blood when cut.

The principal morbid change affects the convoluted tubes of the cortex, the epithelium of which first becomes cloudy and granular, and afterwards proliferates, so as to fill them with masses of irregular or rounded cells. These are seen in sections blocking up the tubules and greatly increasing the thickness of the cortex. In the acute stage the epithelium and granules are mingled with blood-discs and leucocytes; as the affection becomes chronic, minute oil-drops appear and render the tubes black by transmitted light (see Dr Stewart's 3rd plate, fig. 1). Osmic acid shows this change early.

The histology of scarlatinal cases has been recently worked out by Klebs, and in this country by Klein and by Greenfield; and these observers are agreed that the most constant lesions concern the glomeruli and their capsules. Not only do the nuclei of the capillary tufts of the glomerulus proliferate, but there is also an abundant growth of nuclei within the capsule, leading to adhesion between it and the glomerulus, and ultimately to compression and atrophy of the latter. The connective tissue around the glomerulus also becomes crowded with nuclei, which ultimately develop into fibroid tissue; and the afferent vessel of the glomerulus, as well as its capillaries, undergoes a peculiar hyaline change. Sometimes these lesions are limited to a few of the glomeruli only; sometimes they are wide-spread. It is obvious that the obliteration of the space naturally existing between the tuft and the capsule that encloses it must completely abolish the functions not only of the glomerulus itself, but also of the whole length of the convoluted tube that corresponds with it; and, further, the changes in the tufts themselves may be supposed to obstruct the blood-supply to the convoluted tubes, and to affect the nutrition of their epithelium. Consequently some pathologists are now disposed to see in "glomerulo-nephritis," as they term it, the fundamental morbid process that follows scarlet fever, and

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to regard the lesions of the tubal epithelium as secondary and relatively unimportant.

Glomerulo-nephritis is certainly not peculiar to scarlatinal cases, for Cohnheim met with a typical example of it in the case of a man who died some weeks after having his skin rubbed all over with petroleum for four days consecutively.

It is not only present in the early stages of Bright's disease consecutive to scarlet fever, but also to that which arises from cold or pregnancy, or begins insidiously without any assignable cause.

That the convoluted tubules are also affected in these cases, and that leucocytes are found between them as well, are well-ascertained facts.

(2) *The large white kidney.**—In cases that have been of longer duration (three or four months or more) the kidneys present appearances deviating still more markedly from the normal. The cortex has now an opaque white or whitish-yellow colour, both on its surface and on section; and this contrasts very strikingly with the red colour of the medullary portion of the pyramids. The cortex is very much thickened, and the kidneys are sometimes very large, much more so than during the reddish-grey or chocolate-coloured stage. In three cases the weight of a pair of kidneys was twenty-eight and a half or twenty-nine ounces, and in a fourth Dr Moxon found them weighing within half an ounce of three pounds. Nevertheless it sometimes happens that histologically similar kidneys, still retaining their smooth surface, are of natural size, or even slightly smaller; but such cases are exceptions.

The shortest time within which we have seen the kidneys assume the "large white" character is five or six weeks; more often the disease has lasted several months. In each of the two cases just mentioned of great enlargement the patient had been ill for fourteen months. On the other hand, in one instance the kidneys were still of a brick-red colour at the end of six months. It is to be remembered that a considerable proportion of cases of tubal nephritis fatal at this stage are of insidious origin and slow development, so that their duration cannot be fixed. And probably many cases that have been classed as examples of the "large white" variety of Bright's disease really belong to the lardaceous form. Kidneys from the bodies of syphilitic patients, or of those who had phthisis or other suppurative affections, should never be set down to primary parenchymatous nephritis until the absence of the lardaceous change has been determined by microscopical examination.

Histologically, the most striking appearance in most specimens of the large white kidney is the accumulation of immense quantities of fatty epithelial cells in the renal tubes. It is this that gives the opaque yellow colour with reflected light; in thin sections, viewed by transmitted light, the tubes often appear quite black. The stroma of the cortex also is closely studded with fat-granules. Hence such kidneys are sometimes spoken of as "fatty," but very improperly, since that designation ought to be reserved for cases in which a fatty degeneration of the kidneys is primary, as in obese persons who also have fatty liver, or in cases of poisoning by phosphorus. Nor must it be assumed that all large white kidneys are fatty. In those which are translucent and greyish white the amount of fat is often very small.

* Including the pale, marbled, or mottled kidney, and the large, granular, smooth kidney of Bright—granular describing the appearance to the eye, not the feel to the hand. (See his 1st, 5th, and 7th plates in the 'Guy's Hosp. Reports' for 1838.)

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Dr Greenfield, in his summary of renal pathology in the Sydenham Society's 'Atlas,' speaks of cases, especially in pregnant women, in which the microscope shows that the lesions are almost entirely "interstitial." One distinguishing character of such kidneys is their toughness, almost like that of caoutchouc, contrasting strongly with the soft pulpy texture of those in which the principal changes affect the tubal epithelium. But the fact is that in almost all, if not in all, cases of advanced "parenchymatous" nephritis, interstitial lesions are really present to a greater or less extent. This has been insisted on by Mahomed and Saundby in this country, and in Germany by Weigert, who says that he has for years vainly sought for a specimen altogether free from interstitial changes. They are not uniformly diffused through the renal substance, but consist of patches of nuclear growth, which afterwards develop into tracts of connective tissue, in which the nuclei are less numerous. The glomeruli also have their capsules thickened, and pass through every stage in the process of conversion into structureless globular cysts.

(3) *The contracted white kidney.*—If tubal nephritis runs on long enough, without ending either in recovery or in death, the kidneys become shrunken, small, and rough on the surface, though they still retain more or less of the opaque whitish-yellow colour. This continues to distinguish them from kidneys affected with the cirrhotic form of Bright's disease; but the distinction is lost in those cases in which organs primarily cirrhotic become the seat of secondary parenchymatous changes. The occurrence of a granular stage as the ultimate issue of nephritis arising from scarlet fever, or of any other cause that commonly produces a large kidney, was at one time denied; but it has now been clearly established, and a typical example is figured in plate iii of the Sydenham Society's 'Atlas of Pathology.' The case was that of a girl, aged ten, who, rather more than two years before her death, became dropsical as the result of scarlet fever. After four months she recovered, but a year later the face began to swell from time to time, and she died at last with cerebral symptoms. At the autopsy the kidneys were found to be very small indeed, with thick, opaque capsules, hard, tough, and puckered on the surface, presenting on section yellowish-grey masses.*

The records of *post-mortem* examinations at Guy's Hospital contain a good many cases, in young subjects, of a more or less similar kind, the weight of the pair of kidneys being from four to eight ounces. In most of them the history affords no clue as to the date at which the disease had begun. But in some instances it is recorded that there had been an illness attended with dropsy several years before death.

Symptoms.—The parenchymatous or tubular form of Bright's disease varies in its mode of onset in different cases. When it is due to scarlet fever, or to a definite exposure to cold, it may begin with a rigor and pyrexia.

More commonly the earliest indication of the patient's illness is the occurrence of *dropsy*. This may first appear in the loose tissue round the

* A case occurring at the London Hospital, in a young woman of twenty-four, is figured in the same 3rd plate of the Sydenham Soc. 'Atlas.' She was said to have been ill for only three months; but her kidneys were found by Dr Sutton to be reduced to about half their normal size, to be "very granular, and of a reddish colour, everywhere mottled with a yellowish or purplish or greyish substance."

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eyes, and in slight cases it is especially noticeable before the patient gets up in the morning; the lower eyelid is œdematous, and the conjunctiva also. This conjunctival œdema produces "the bright eye" of renal disease, and "the tear that does not run over." Generally dropsy also affects the limbs; and a favourite seat for it is the lower part of the back, as far down as the sacrum—"the lumbar cushion." The external genitals, too, are very apt to become swollen, and the prepuce is sometimes so stretched and twisted as to interfere with micturition. In extreme cases almost the whole body becomes bloated, while the extreme anæmia, which rapidly develops itself, gives the skin a white, or sometimes a yellowish, wax-like colour.

The appetite is often bad, the tongue furred, and the bowels constipated. Vomiting is frequently a marked symptom, and there may be much headache. Pain in the loins is often altogether absent, but sometimes it is severe, radiating to the groins and down the thighs, and accompanied with tenderness to pressure.

The *urine* in the acute stage is always scanty, and sometimes entirely suppressed. Though the patient is constantly striving to micturate, a few drops of blood-stained liquid may be all there is to pass. Complete suppression is a very grave symptom, and generally points to a fatal issue, which is seldom long delayed, even if the kidneys should afterwards, to some slight extent, resume their functions. When some ounces of urine are secreted daily it is commonly of high specific gravity (1025—1030) and of dark colour, more or less red or brown from the presence of blood. Its appearance may often be compared with that of strong tea or of porter. It is turbid, and throws down a chocolate-coloured deposit, containing altered blood-discs, swollen epithelial cells, and casts, some hyaline, others full of blood-discs or epithelial cells.

Albumen is of course always present in the urine when there is blood; but the quantity of albumen is often not so large at this period as it is a little later, when the hæmaturia has passed off. The late Dr Mahomed described, in the 'Med.-Chir. Trans.' for 1874, what he termed a *pre-albuminuric* stage, in which, while no albumen can be detected, the guaiacum test reveals the presence of colouring matter of blood, and the sphygmograph shows a marked increase of arterial tension. Bartels also mentions cases in which, when scarlatinal dropsy first set in, the urine, though exceedingly scanty, was non-albuminous; and he cites a case of Henoch's, in which no albumen could at any time be detected, except on the day before death, when the patient was cyanotic and almost pulseless, as the result of an attack of convulsions. Commonly the amount of albumen ranges from 2 to 5 per cent. The total quantity passed in the twenty-four hours is said to be from 80 to about 400 grains. The excretion of urea is greatly diminished, falling to half the normal amount, or even less.

When tubal nephritis ends in recovery in the course of a few weeks—as occurs in the large majority of scarlatinal cases, and in many of those due to other causes—the dropsy and the other symptoms subside, and the urine gradually recovers its normal characters. It is more abundant, of lower specific gravity, and paler; it no longer contains blood, which, indeed, is generally present only at the very commencement of the attack; the quantity of albumen in it becomes less and less, until at length there is none.

When the disease runs on for months, the dropsy continues, or increases. The patient remains bloated and anæmic, for "large white kidneys make a large white body." He lies helplessly in bed, his

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back propped up with pillows, his legs stretched stiffly out before him, or supported by a pillow under the knees. The swelling of the external genitals is often extreme. The scrotum looks like a bladder full of water, and is so large that there is no room for it between the thighs; while the prepuce is distorted, swollen, and almost translucent. Sometimes the cuticle over some of the distended parts cracks, and the dropsical fluid oozes out in such quantities as to soak through the bedding. This may cause considerable tracts to become excoriated, and ultimately to be covered with pale granulations, which, when they skin over, give the surface a warty appearance. Or inflammation may set in, attended at first with a crimson redness (*erythema leve*), like that of erysipelas, and leading to more or less extensive gangrene of the skin, and even of the subcutaneous tissues. Erythematous or exfoliative forms of dermatitis are not uncommon, independently of the irritation of urine or exuded serum, or of tension of the integuments (cf. p. 460).

In this more advanced stage the state of the urine is very variable. It may still be scanty and of rather high specific gravity; but more frequently it gradually becomes abundant and pale, and then its specific gravity falls considerably below the normal, to 1010 or 1005, or even lower. It is still albuminous, sometimes highly so; indeed, the quantity of albumen in it may reach 5 per cent., or even more. But often as the disease goes on the amount of albumen lessens. Blood in small quantity may be present from time to time, but this is quite exceptional. Casts are commonly found in abundance, some of them hyaline, others containing leucocytes, epithelium, and fat-granules, others completely opaque and granular.*

The excretion of urea is at all periods of the disease much below the normal amount. Even when the flow of urine becomes more abundant, the total quantity of urea contained in it in the twenty-four hours, instead of increasing, is less than before. Albuminuric retinitis, in all its forms, is of frequent occurrence (p. 458). Pleuritic effusion is frequent, and œdema of the lungs.

Event.—In some cases of tubal nephritis, at whatever stage of the disease, death occurs as the result of the dropsy or of inflammation; especially from accumulation of fluid in the great serous cavities of the chest, or (though very rarely) from œdema of the larynx. In many instances it is the result of pulmonary œdema, or of pneumonia which is often more or less œdematous. In others it is brought about by the supervention of acute pleurisy or pericarditis. Certain cases, again, end by uræmia; while in a few, failure of the heart, from dilatation of the left ventricle, is the direct cause of the fatal issue.†

Complete recovery seldom takes place when the disease has lasted many months, still more rarely when it has been prolonged for years. But it is surprising how symptoms will sometimes subside and disappear, and how, even after the patient has been waterlogged and has had alarming uræmic attacks, he may regain what appears to be a fair state of health.

* The fatty casts appear not to be the only vehicles for excretion of the products of nephritis, for Dr Hamilton gives reasons for believing that the greater part is absorbed into the intertubular spaces and removed by the lymphatics ('Journ. of Anat. and Phys.,' vol. xxv, p. 198).

† See Dr Goodhart's paper in the 'Guy's Hospital Reports' for 1879 (3rd series, vol. xxiv); and, on this and other clinical points in the origin and natural history of scarlatinal nephritis, Dr E. W. Goodall's report based on a very large number of cases at the London Fever Hospital (*ibid.*, vol. xlvi, p. 91).

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A striking instance of the removal of an albuminuria of long standing by treatment is the case of a medical man, aged twenty-six, whose urine after an attack of scarlet fever was continuously albuminous after meals for more than six years. By the advice of Dr Johnson he was strictly dieted, and at the end of nine months the urine became normal, and remained so eighteen months later ('Brit. Med. Journ.,' 1879, ii).

A young man of twenty was under the writer's care with acute albuminuria, excessive anasarca, ascites, and anæmia, who was tapped and continued dropsical for twenty months. Yet he finally recovered and was able to be about again.

II. Lardaceous Disease of the Kidney.*—From the time of Rokitansky a lardaceous affection of the kidneys has been described as one of the forms of Bright's disease, and Wilks has shown good reason for supposing that one of the specimens figured by Bright himself as a "large white kidney" was lardaceous, inasmuch as the liver of the same patient, which is still kept in the museum of Guy's Hospital, presents that morbid change. It is remarkable that in the liver and in the spleen the lardaceous change is never associated with diffused inflammation; but in the case of the kidney the tubular or diffused nephritis is unquestionably set up by the same degeneration. This gives to lardaceous disease of the kidneys a clinical importance which does not belong to the like affection of any other organ; and therefore the general bearings of that morbid change may be better discussed here than elsewhere.

The lardaceous degeneration.†—The first step in the study of this disease was the discovery by Meckel of the fact that the tissues affected give a peculiar reaction with iodine, turning a walnut or mahogany-brown colour, whereas healthy tissues remain pale yellow; he thought this due to the presence of cholesterine. Virchow afterwards stated that on the addition of dilute sulphuric acid a more or less distinct blue or purple tint can be detected, to which he attached great significance, regarding it as an indication that the lardaceous material was chemically related to principles of vegetable origin, such as starch or cellulose. But many observers have failed altogether to obtain any blue tint, and most will agree with Ziegler that this reaction is at best imperfect. Unfortunately, it led Virchow to give to lardaceous disease in general the name of "amyloid." In Germany this name is still used, though in France and England it never replaced the older term, which embodies no erroneous theory. It applies not to the fat (*lard*, *lardacé*, a comparison made by Portal), but to the firm, elastic, translucent rind of bacon. It was shown by Kekulé that the "lardacein" (as it is now called) contains nitrogen, and afterwards by Kühne and Rudnett that it is really allied to albumin.‡

* *Synonyms.*—Rokitansky's Speckniere—The waxy kidney of the Edinburgh pathologists—Virchow's amyloide Nierenentartung—Depurative nephritis of Dickinson.

† The earliest term applied to this condition was "lardaceous" (*speckartig*, like bacon-rind) by Rokitansky and the Vienna school. In Edinburgh it was named "waxy" by the late Professor Sanders. Budd and other English writers called it "albuminous," or "scrofulous enlargement." Schrant, and afterwards Oppolzer, used the vague epithet "colloid." Any of these is better than the misleading term "amyloid," starch-like; but "lardaceous" is used in France and America, and is adopted in the Nomenclature of the Royal College of Physicians. Moreover, it has no misleading meaning, and is unambiguous.

‡ Kühne's plan was very ingenious. Lardacein is insoluble and unaffected by reagents, alkalies, acids, or digestion. He therefore submitted a lardaceous liver to artificial gastric digestion, and when everything else was dissolved away, analysed the residue.

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Tests.—An aqueous solution of iodine and iodide of potassium is the best test for the lardaceous material. The cut surface of the organ to be tested must be first washed free of blood, which, if present in large quantity, makes it impossible to speak with confidence as to the presence or absence of the reaction. The surface is then lightly brushed over with the solution. A considerable degree of lardaceous change becomes apparent in a few seconds, by the formation of brown or black spots or streaks. If, however, the change is very slight in amount, it may be perceptible only with the microscope, and after the application of the iodine to a thin section. In 1875 Jürgensen made known in 'Virchow's Archiv,' and also Cornil in the 'Archives de Physiologie,' the fact that methyl violet, prepared by the action of iodide of methyl upon aniline, gives a beautiful colour-reaction with lardaceous tissues. Those parts which present the lardaceous change slowly become red, whereas the unaltered parts are stained blue. The main advantage of this over the iodine test is the definiteness with which the reaction remains limited to certain elements in a complex structure. Thus it is invaluable in determining whether the secreting cells of an organ, or the adjacent capillaries, are the seat of the morbid change. Another important point is that it enables permanent preparations to be made and preserved in glycerine. On the other hand, it cannot be used as a rough test in the *post-mortem* room; and even as applied to microscopical sections of the tissues it seems to be in no way superior to the iodine test when the question is merely the presence or amount of the lardaceous change.

Without the addition of any colouring matter, lardaceous degeneration is plainly recognisable with the microscope in thin sections of an organ. The affected parts of the tissue have a swollen, homogeneous, glistening appearance, which cannot be mistaken by a practised eye.

Theory.—As to the nature of the process by which lardacein is substituted for the natural proteids of the tissues, nothing is as yet known. Whether this material is formed where it is found as a "degeneration" of the normal structures, or whether it is a "deposit" or an "infiltration" derived from the blood, is still uncertain. It cannot be detected in the blood; and in every organ the parts most apt to become lardaceous are not the proper elements of its structure, but the capillaries and the small arteries. On the "infiltration" theory, it is extremely hard to understand why the change should be limited to the vessels in certain organs, and not equally distributed over those of the entire body. Moreover, even in a single organ—such as the kidney—it is apt to be very partial, occurring only in certain glomeruli, or only in certain loops of a glomerulus. Surely this looks more like a local "degeneration." There is little analogy between the lardaceous change and the process of calcification, for that is due to the deposition of lime-salts from solution in the blood.

Locality.—The lardaceous change has from time to time been met with in strange situations, where its occurrence certainly would not have been anticipated; and what is still more remarkable is that in these cases it has often been absent from its usual seats. As examples may be cited Burow's case of lardaceous degeneration of laryngeal tumours, Ziegler's case of lardaceous nodules at the base of the tongue, Birch-Hirschfeld's statements as to the presence of lardaceous material in mesenteric glands after enteric fever, and, above all, the curious examples of a lardaceous change in the vessels of the conjunctiva recorded by Sämisch, by Leber, and by Kyber.

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References to all these observations may be found in a paper by Kyber in vol. lxxxi of 'Virchow's Archiv.' It has also been shown that by carefully searching the various structures of the body, when the lardaceous change has its usual distribution, one can often detect its presence in many other parts besides those in which it is commonly recognised.

As an important pathological process, lardaceous degeneration may be regarded as limited to a small number of organs, these being the kidneys, the liver, the spleen, the intestine, the thyroid, and the adrenals; and in the last two, so far as is at present known, it gives rise to no symptoms and has no clinical significance.

Dr Goodhart found that of 150 cases at Guy's Hospital the kidney was affected in 110, the spleen 99, the liver 73, and the intestine 63. At the London Hospital Dr F. C. Turner found, among 58 cases, the spleen affected 48 times, the liver 30, the kidneys only 15, and intestine 10 ('Path. Trans.,' vol. xxx, p. 517).

Ætiology.—Many writers, Bartels for example, speak of scrofula, chronic tuberculosis, and syphilis, as being all alike constitutional diseases which predispose to the development of lardaceous degeneration, probably in consequence of their liability to induce chronic ulcerations of the bones, skin, or the mucous membranes. He also admits, however, that it may show itself as the result of chronic and protracted suppuration apart from constitutional disposition. Others admit as causes, malaria, cancer, and other forms of cachexia. Now, such a method of statement appears to be incorrect in two important respects. In the first place, it is certain that syphilis in some mysterious way induces the lardaceous change in cases in which formation of pus has occurred only to the most insignificant extent, if at all. In the second place, it is more than doubtful whether "scrofula" (in other words the presence of tubercle in the lymph-glands or joints) has any influence in the same direction, apart from its tendency to cause suppurative lesions. We may therefore reduce the known causes of the lardaceous change to two—chronic *suppuration* and *syphilis*; which are frequently combined, but are capable of acting separately. Some pathologists add the malarial cachexia, but with no adequate evidence. It is, however, possible that there may be other causes besides the two above enumerated; for instance, there is some reason for thinking that the lardaceous change occurs in association with Hodgkin's disease without there having been sufficient suppuration to account for it.

It must not be supposed that in all the cases which after an autopsy are positively set down as syphilitic, the presence of this disease was, or could have been, recognised during life. On the contrary, there are a great many instances in which it is only by the discovery in the dead body of such lesions as fibroid degeneration of the testes, or small gummata or cicatrices in the liver, that the syphilitic character of the case is made out. In this respect there is a wide difference between the two great causes of the lardaceous change. For the occurrence of suppuration to an extent adequate to produce lardaceous affections can hardly ever be overlooked during the patient's life, except perhaps when it follows intestinal ulceration (as in dysentery), or when there is no external discharge of pus at all, but merely a large deep-seated abscess, the contents of which undergo slow inspissation and caseation.

In 1876 the author brought before the Pathological Society a tabulated statement as to what appeared to have been the ætiology of 244 cases of

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lardaceous disease of the viscera, collected for him by Mr H. F. Lancaster from the records of autopsies in Guy's Hospital over a period of twenty-one years.

In 154 there had been *prolonged suppuration;* 67 of these were cases of *phthisis;* in 51 there was disease of some joint (generally the hip- or knee-joint), or caries of the spine, or of some other bone; in the remaining 36 there were a variety of affections, amongst them empyema, dysentery, calculous and scrofulous pyelitis, ulcerating, cancerous, or sarcomatous tumours, cystitis from stricture, and bedsores of long standing as the result of disease of the spine.

There were also 5 other cases in which there had, indeed, been suppuration, but in which it seemed doubtful whether this had been enough to afford a reasonable explanation of the lardaceous change. Thus in one instance there had been chronic discharge from one ear as well as from the nose; in another, one testicle had been inflamed and suppurating as the result of a blow two months and a half before death, but with open discharge only for a fortnight; and in a third there was merely tubercular peritonitis with caseous disease of the mesenteric glands.

In five or six of the 154 cases that were clearly due to prolonged suppuration, this had had a definite starting-point, so that some idea could be formed of the length of time required for the development of lardaceous lesions. One patient had had a carbuncle eight months; another had had pelvic cellulitis for exactly the same period; a third had had a bedsore seven months; a fourth was affected with a sarcomatous growth, which had been discharging for four months. In a fifth case there had been fracture of the spine three months before death, bedsores two months and a half, and also a double empyema; but syphilis was probably also present, inasmuch as the testes presented fibroid changes and there was a scar in the groin. A sixth patient had suffered amputation of the leg three months and a half before death, on account of a compound fracture with abscess; in that instance it is particularly noticed that both in the liver and in the spleen the lardaceous change was just beginning.

Next, among the 244 cases of lardaceous disease there were 76 in which there was satisfactory proof (either from the history, or from appearances after death, or from both together) of *syphilis;* and in 3 others there was at least a suspicion of its presence. In about 34 of these 76 cases there was evidence of there having been bone disease or suppuration, leaving 42 to be ascribed to syphilis alone. In no instance is it stated that the syphilis had been inherited; but Bartels speaks positively of having seen lardaceous affections in cases of inherited syphilis attended with ulceration of skin and bone.

Of the 244 cases, there are thus left only six examples of lardaceous disease that were not accounted for by the presence either of syphilis or of suppuration; and in some of them the notes of the autopsy are incomplete, the state of the testes, in particular, being unrecorded. Now, it seems far from unlikely that in each of these six cases the cause of the lardaceous change was really syphilis. When we consider how slight and accidental are often the lesions on which we rely as proofs of the presence of syphilis in the dead body, it appears almost certain that there must be other cases in which no syphilitic lesions were observed, but in which syphilis was really present. Six cases among 244 are not perhaps too numerous to be covered by this plea; even if we ought, in fairness, to add to them some

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of the five other cases in which, although suppuration had occurred, there was a doubt whether it was adequate to the production of the lardaceous change.

Since the first edition of the present work, the writer has obtained a series of statistics in continuation of those just given. They were collected by Dr H. J. Campbell from the *post-mortem* records of Guy's Hospital, and extend from 1876 to 1889 inclusive. The total number of cases of lardaceous disease recorded in these fourteen years was 302. Prolonged *suppuration* had been present in 124; there were 35 cases of (probably tuberculous) disease of the joints, usually the knee, hip, or sacro-iliac synchondrosis; 31 cases of vertebral disease, often associated with psoas abscess; 5 cases of caries or necrosis of other parts of the skeleton; 5 of empyema; 11 of pelvic abscess; 2 of chronic caseous disease of the kidney; and the 35 remaining cases were of suppuration from chronic abscesses, bedsores, ulcers, *&c.* There were 121 cases of pulmonary *phthisis*, together with one of acute general tuberculosis, and another of tuberculous disease of the uterus and of the peritoneum. There were 43 cases of *syphilis*, shown by anatomical changes after death, whether or not by symptoms observed during life.

These three chief causes, therefore, account together for 289 cases out of the total of 302. The remaining 13 cases were very miscellaneous, as is shown by only two coming under the same denomination. One was a case of acute Bright's disease, one of chronic tubal nephritis with cirrhosis of the liver, and one of valvular disease of the heart with a large white kidney. In another case there was found a renal calculus and shrivelled kidney, which was probably once the seat of suppuration. Two were cases of chronic non-tuberculous disease of the lung without any history of prolonged or excessive purulent expectoration. In two an adherent pericardium was found, one cut short by diphtheria, the other with a history of chyluria seven years before. One was a case of hobnail liver, and one of glio-sarcoma of the brain and lung. There remain only three cases, two of multiple lymphomata (Hodgkin's disease), and one in which lardaceous degeneration appeared in cancerous glands.

In many of the thirteen cases just enumerated the lardaceous change was slight, and limited to the diseased liver or kidney or lymph-glands. Probably some of them might have been brought under the head of suppuration or of syphilis; but it is best to leave them as the conditions stand recorded by independent observers in the records of autopsies.

The result is strongly to corroborate the conclusions derived from Dr Fagge's series. Putting both together, we find that in 546 cases of lardaceous disease occurring consecutively in the same hospital during the thirty-six years from 1854 to 1889 inclusive, there were 216 which could be ascribed to suppuration, 190 cases to phthisis, and 122 to syphilis, leaving only 19 unaccounted for by the anatomical evidence of previous disease.*

Age and sex.—The period of life at which lardaceous lesions due to

* Dr F. C. Turner referred 42 of his 58 cases ('Path. Trans.,' xxx) to prolonged *suppuration* (including 20 cases of *phthisis*), 8 or 9 to *syphilis*, and 6 to *malignant disease*, while the remaining 2 were unaccounted for. Of the six cancerous cases, in one there had probably been lues and certainly considerable suppuration, in another there had been six months' ulceration, in a third there was history of syphilis and ague, in the fourth a history of ague; and one was a case of Hodgkin's disease, with lardaceous change in the enlarged lymph-glands. Only one of the six cases, epithelial cancer of the tongue, seems to exclude any of the generally recognised causes.

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syphilis are most apt to occur is from thirty-one to forty. At Guy's Hospital there have been fourteen fatal cases in persons between those years, against six in persons between twenty-one and thirty. As the result of protracted suppuration, on the other hand, the affection has been slightly more frequent during the earlier period; it occurs, too, in adolescents, and even in children; there has been one case at Guy's Hospital in a boy only four years old. Above the age of fifty lardaceous disease is decidedly uncommon, but we have had one instance of it in a man of sixty-five.

Among our cases male patients were more numerous than females in the proportion of two to one.

Anatomy of the lardaceous kidney.—The appearances presented by a kidney affected with this disease vary widely in different cases. Sometimes the lardaceous degeneration of its vessels is the only change in the organ discoverable, whether by the naked eye or with the microscope. This, however, is only the case when the immediate cause of death has been some other disease; generally one of the maladies to which, as we have seen, the lardaceous change is commonly secondary. No instance can be recalled in which, at this stage of the renal affection, the patient has succumbed to cachexia from the simultaneous development of the lardaceous change in several organs. How rare it is in the *post-mortem* room for a kidney to be seen which presents no other lesions than the lardaceous change, may appear from a collection of more than sixty cases of lardaceous kidney taken from our records at Guy's Hospital; among them there seem to be only three or four uncomplicated examples.

A kidney thus affected is of the natural size, and looks smooth and healthy, except that a keen eye may perhaps perceive the glomeruli to be somewhat more distinct and more translucent in appearance than is natural. On the addition of iodine the change is generally most marked in the glomeruli; and sometimes it is entirely limited to them. Nor does it affect all of them at once, being, on the contrary, often confined to a few of them only, and even in the same glomerulus to one or two of its coils. Not unfrequently the afferent arteries are involved as well, their middle coat being the first part to suffer. But sometimes the reaction is obtained, not in the cortex, but in the straight vessels of the pyramids. And sometimes it seems to begin in both the cortex and the pyramids simultaneously. When the change is very far advanced, the capillaries round the renal tubes are also affected, as well as the basement membrane of the tubes, and perhaps the epithelium lining them.*

In the great majority of instances lardaceous kidneys are the seat of other lesions likewise, by which they are greatly increased in size. In about a quarter of the sixty cases the weight of the two organs together was from twenty to twenty-seven ounces. The appearance of such kidneys is sometimes very peculiar, and fully justifies their being termed "waxy" or *butternieren*. Their surface is smooth and pale yellow, with conspicuous stellate veins; their section is shining and polished, of a semi-translucent grey or yellowish colour, sometimes showing obviously the grey swollen glomeruli, sometimes mottled more or less thickly with creamy opaque spots and streaks, where fatty granules are present in abundance. What causes the

* Dr Dickinson's 8th plate gives an excellent picture of the appearance of the kidney to the naked eye, and his 9th and 10th of its histology; the latter is also well illustrated by Dr Stewart's 5th and 6th plates.

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enlargement of the organs in these cases is undoubtedly the accumulation of inflammatory products, not only in the tubes, but also in the interstitial tissue. But why such diffused nephritis should arise is difficult to say.

Cohnheim is inclined to believe that it is an independent result of the same cause which produces the lardaceous change, and which we shall presently find to be generally either syphilis or prolonged suppuration. But the association of the two morbid processes occurs far too frequently to admit of such an explanation, especially as no pathologist will maintain that diffuse nephritis, apart from the lardaceous change, is a common result of syphilis or of suppuration.

The known ætiology of the lardaceous change clearly excludes the view of Cornil and Ranvier that it is preceded by the nephritis.

We have therefore no alternative but to suppose that in some way lardaceous degeneration must cause the nephritis. And the objection that no similar inflammatory process is found to arise in a lardaceous liver or spleen loses its chief force when we consider that neither of these organs is subject, under any circumstances, to an affection comparable to Bright's disease.

Weigert observes that the peculiar position of the glomeruli, between the arterioles of the renal cortex and the capillary blood-supply to the tubes, may well render any obstruction to the flow of blood through the glomeruli the cause of damage to the tissue beyond. He believes that the first result of the lardaceous change is to produce fatty degeneration of the epithelium, and that this degeneration, whatever its origin, is apt to set up diffused interstitial nephritis. But certainly fatty degeneration of the kidneys is not uncommon, without the least trace of consecutive nephritis; and we have no reason to assume the presence of obstruction.

Sometimes lardaceous kidneys are found after death to be smaller than natural. This occurred in about one out of five or six of the sixty cases collected from our *post-mortem* records. As a rule the loss of size was not very obvious, the two organs weighing together not less than seven and a half or eight and a half ounces; but in one instance the weight (of what happened to be a "horseshoe" kidney) was only three and a half ounces. There was always, however, much irregularity and shrinking of the surface —a more or less markedly "granular" condition; and the loss of substance was no doubt far more considerable than was indicated by the weighing machine, inasmuch as the kidneys still retained large quantities of inflammatory exudation, as well as the lardaceous material itself. Such wasted organs clearly represent the most advanced stage of the lardaceous affection, and it is fair to assume that its course has been more than usually slow and protracted. But there is no evidence that kidneys that ultimately become thus reduced in size have passed through an earlier stage in which they were enlarged. Some observers have tried to account for such cases by supposing that the kidneys were already contracted and granular (from gout or some other cause) before they began to be affected with the lardaceous change, as the result of syphilis or protracted suppuration. And there seems to be no doubt that in 1866 we had at Guy's Hospital an instance of the accidental association of the two morbid processes in the case of a woman who died at the age of forty-five of cerebral hæmorrhage, and whose kidneys, besides being lardaceous, were granular and full of minute cysts. Weigert remarks that there ought not, with the microscope, to be any difficulty in distinguishing mixed cases of this kind, since the glomeruli would doubtless be shrunken and atrophied (in the manner described at p. 496,

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infra), whereas in the most advanced stages of a lardaceous kidney they continue to present the appearances characteristic of that affection. The hypothesis of an independent granular change could, however, be hardly applied to two of our cases, each of which occurred in a young man of twenty-four, for at that age such a change in the kidneys is almost unknown; in one of these cases the organs weighed seven and a half, in the other six and a half, ounces.

Symptoms.—The characters of the urine secreted by lardaceous kidneys were first studied by Traube in 1858; and his account of them has been confirmed by the labours of Grainger Stewart and others who have since taken up the subject. But although cases doubtless sometimes occur, in which the state of the urine may alone suggest to an experienced observer the idea that the renal affection from which the patient is suffering is lardaceous, and not one of the other forms of Bright's disease, such cases are rare. Indeed, considering how constantly the lardaceous change becomes associated with other lesions in the kidney, and how diverse these lesions may be, it is very unlikely that the urine will always present the same characters. The cases in which the influence of the lardaceous affection should be most clearly traceable are, of course, those in which it is uncomplicated with inflammatory changes in the renal cortex. But there is much force in Cohnheim's remark, that the existence of such "simple" lardaceous kidneys can scarcely ever be determined, except in patients who have fatal disease of other organs which causes them to be wasted and anæmic, and to suffer from pyrexia, diarrhœa, and other symptoms that must in themselves disturb the balance of the nutritive changes in the body. Thus Bartels appears to be right in declaring that there is no evidence that the lardaceous change in itself interferes with the excretion of urea. If this is diminished it is probably due to concomitant, tubal or interstitial, nephritis. The effect of the lardaceous change in the glomeruli appears to render their walls more permeable than before, to water, salts, and urea.

With regard to the *quantity* of urine secreted by lardaceous kidneys, different writers make very different statements. According to Grainger Stewart, unless an extreme degree of nephritis is present, it is excessive, ranging from fifty to two hundred ounces daily. He also maintains that an increased flow of urine is in many instances of great clinical importance as an early symptom of the lardaceous affection, preceding albuminuria. Dr Johnson supports him in both these statements, and so does Dr Dickinson, who puts the daily amount at from fifty to ninety ounces. Bartels, however, gives much smaller average amounts—from fifty to sixty or seventy-six ounces; and he expressly remarks that there is never such a degree of polyuria as occurs (for example) in some cases of granular disease of the kidneys. He mentions a case in which the average daily secretion amounted to less than seventeen ounces. Wagner also declares that in the majority of cases—almost constantly in the last few weeks, but sometimes in the whole course of the disease—the urine is more or less scanty, sometimes with intervals of a few days in which it is normal or increased in quantity. No doubt these discrepancies of opinion depend mainly upon the degree of care taken by different observers to recognise an underlying lardaceous change in kidneys that would formerly have been set down as examples of parenchymatous nephritis. Wagner, for instance, says that in the *post-mortem* room the lardaceous form of Bright's disease is seen more frequently than any other—"much more frequently than chronic parenchymatous

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nephritis or cirrhosis of the kidney;" and the author must confess that his experience at Guy's Hospital accords very much with this.

The specific gravity of the urine varies with the quantity; it may be as low as 1003 or 1005, or it may occasionally reach 1025. For several days before death the urine is often very scanty, and of high density.

In all but exceptional cases *albumen* is present. Lecorche has maintained that so long as the lardaceous change is uncomplicated with nephritis the urine is non-albuminous. But Bartels denies that this is correct; and one at least of our cases at Guy's Hospital affords proof to the contrary. On the other hand, there can be no doubt that Bartels himself goes too far when he declares that albuminuria is always present, except perhaps at the very commencement of the affection, when the change in the vessels is just beginning. Many observers have noted that even in cases in which albumen is sometimes to be detected, it may at other times be absent from the urine. And Cohnheim speaks of having made autopsies in several cases in which albuminuria was said to have been altogether wanting. The quantity of albumen varies greatly. There may at first be only a little; but the rule is that the quantity is large, and it may reach from 1 to 3 per cent. Probably the presence of such considerable amounts of albumen may be taken as evidence that there is nephritis as well as the lardaceous change.

Blood is very rarely present, even in small amount. The statement made by Senator some years ago that the presence of paraglobulin in large quantities is characteristic of this rather than of other forms of Bright's disease, has not been confirmed since the introduction of Hammarsten's method of separating paraglobulin from serum-albumen (p. 445).

In some exceptional cases there may be found in the urine glistening epithelial cells, which turn reddish brown with iodine from having undergone the lardaceous change before being shed. It has been supposed that their presence is conclusive as to the nature of the renal affection; but the writer has frequently observed this reaction with iodine, not only without any symptoms of lardaceous disease of the kidney, but in spheroidal and transitional epithelium of cystic origin.

The urine is generally pale and transparent, throwing down no deposit, or at most a few *hyaline casts* and epithelial cells.

When, however, there is much nephritis, the urine may be high-coloured, and may give an abundant precipitate containing lithates, as well as numerous hyaline and granular casts, to which epithelial and fatty cylinders may be added (see Dickinson's plate vii, fig. 2).

The other symptoms which accompany lardaceous disease of the kidneys are very variable and uncertain. Pallor is the most constant, but anæmia, cachexia, and wasting sometimes may be absent. Bartels speaks of having had patients who were able to follow laborious occupations at a time when there was unequivocal evidence of this form of Bright's disease. And one of our cases at Guy's Hospital was that of a man, aged thirty-six, who was admitted with a fractured spine, the result of a fall while he was carrying a sack of barley on his shoulders; lardaceous changes were found in the liver, the spleen, and the adrenals, as well as in the kidneys, which were enlarged, and weighed sixteen ounces.

Dr Stewart lays stress upon a pasty or waxy complexion, with a little pigment about the eyelids, and distension of small blood-vessels upon the cheeks, as being suggestive of this form of renal disease.

Dropsy is sometimes wanting as a symptom of lardaceous disease of the

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kidney, especially when there is diarrhœa from a coincident affection of the intestinal mucous membrane. But this symptom is frequently present in an extreme degree, affecting the face and the arms and the whole body, exactly as in cases of the "large white kidney," for which (as we have seen) cases really lardaceous have so often been mistaken, both at the bedside and after death. Bartels declares that when dropsy does occur it is ordinarily confined to the lower limbs and to the abdomen. But though reports of some few cases, taken by themselves, might seem to support this statement, our experience, on the whole, at Guy's Hospital does not bear it out. When such a limitation of the dropsy is observed, it is an evidence that the immediate cause of the œdema was cardiac failure (cf. *supra*, p. 456).

But it is agreed by all recent writers that the heart is seldom found enlarged in cases of lardaceous disease of the kidneys. Of more than sixty cases observed at Guy's Hospital there were only four in which the heart weighed from eleven to thirteen ounces; and in two of these the kidneys also were enlarged, weighing fourteen and eighteen ounces respectively. The frequent absence of cardiac hypertrophy seems to be sufficiently explained by the cachectic and anæmic condition of the great majority of those suffering from lardaceous degeneration. Cohnheim asserts that even when there is wasting and shrinking of the kidneys, the heart does not hypertrophy unless the lardaceous change was preceded by the cirrhotic. However this may be, primary cirrhosis of the kidneys is not infrequently complicated by lardaceous degeneration, and in such cases cardiac hypertrophy and dilatation with œdema of the legs would follow, and might explain Bartels' statement.

Retinitis and *uræmia* are said to be rarely observed in cases of lardaceous affection of the kidney; but in three cases in Guy's Hospital death was ushered in by convulsive seizures. One patient, who had been in the hospital nine months previously with dropsy, was readmitted four days before death in a state of collapse, with cold and blue extremities, the result of severe diarrhœa and vomiting. In several instances acute peritonitis, or acute pericarditis, was what immediately brought the disease to a fatal termination.

Diagnosis.—From what has been stated with regard to the clinical effects of the lardaceous affection of the kidneys, it is evident that, apart from other circumstances, neither the characters of the urine, nor any other symptoms, can be relied on to suggest its real nature, as distinguished from the other forms of Bright's disease. What in practice generally enables us to make a diagnosis is either that indications of lardaceous change are presented by the liver or by the spleen, or by both; or that one or other of the known causes of this change is present.

The liver is always enlarged when the seat of lardaceous disease; but diffused lardaceous infiltration of the spleen and lardaceous disease of its Malpighian follicles forming the "sago-spleen" may either of them exist without increasing its bulk. Further, even when both spleen and liver are considerably enlarged, it may be impossible to detect the fact if the patient is very fat or dropsical. Knowledge of the causes of the affection is therefore all-important.

Prevention and treatment.—In order to *prevent* lardaceous affections, it is clearly most important that all inflammatory diseases likely to lead to protracted suppuration should be so treated as to shorten their course as much as may be; and in cases of syphilis it is desirable, on this account, as well as with the direct object of eradicating the disease, to continue the administration of mercury or iodide of potassium longer than suffices to remove symptoms.

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It might be thought that the clinical diagnosis of lardaceous degeneration from other forms of Bright's disease is, after all, a matter of no great moment, since it is to the presence of diffuse nephritis that most of the symptoms are really due. But, as regards both prognosis and treatment, it is important not to overlook the presence of lardaceous complication. When general dropsy sets in, the downward progress of cases with lardaceous kidneys is often more rapid than in other forms of Bright's disease. Again, it not infrequently happens that albumen is detected in the urine of syphilitic patients several years before other symptoms of renal disease appear; and there is evidence that such cases, even when dropsy occurs, may end in recovery, and the urine gradually resume its normal characters.

Two instances of this are recorded by Dr Dickinson in vol. xxx of the Pathological Society's 'Transactions.' One was that of a patient who contracted syphilis in 1861, and who in 1874 began to suffer severely from periosteal nodes; soon afterwards his legs became œdematous, and his urine loaded with albumen, so that the coagulum filled one fourth or one half of the test-tube. Under specific treatment, assisted by a residence on the Riviera during four winters, the disease slowly subsided, and by June, 1879, the urine was normal and the patient apparently in perfect health.

The other case was that of a young man with secondary syphilis, caries, albuminuria, and dropsy. After two years' treatment, chiefly with *iodides* of potassium and of iron, the urine became normal, and all his symptoms disappeared.

As to what changes take place in the kidneys when recovery occurs, nothing is yet known. It seems not impossible that the glomeruli and other vessels that were lardaceous may return to a normal condition, if the albuminuria has only lasted a short time. But in protracted cases, such as Dr Dickinson's, it is far more likely that the affected parts of the kidney shrink and undergo atrophy, and that parts which had escaped the disease perform the whole renal functions, with possibly some compensatory hypertrophy.

In practice the only way in which one can escape the danger of overlooking among one's cases of Bright's disease some early stages of the lardaceous form, is to give a trial to iodide of potassium in any case of obscure origin, without waiting for certain signs of syphilis or for a history which is often difficult to obtain or to trust.

CIRRHOSIS OF THE KIDNEY.*—The morbid process which produces the "small red kidney" is the more frequent, the most insidious and in some respects the most important of all those described by Bright. The *general* characters of this form of morbus Brightii have been already defined (p. 442).

It is held by most English pathologists that those cases of renal atrophy which are secondary to tubal nephritis or to lardaceous degeneration should be distinguished from those which are the results of a primary interstitial nephritis. One proof of the reality of this distinction is afforded by the difference in the appearance of granular kidneys at different periods of life. In autopsies on children, and even in adults under the age of twenty, twenty-five, or perhaps thirty years, we do not meet with the red or brown contracted and granular kidneys which are so frequent in middle-aged and

* *Synonyms.*—Chronic interstitial nephritis—Red granular atrophy—Granular degeneration, producing the small red kidney of Bright—Chronic gouty nephritis.—*Fr.* Maladie de Bright, forme chronique aux reins ratatinés.—*Germ.* Schrumpfniere (the contracted kidney).

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in old people. When granular kidneys are seen in young subjects, they are full of opaque whitish-yellow spots or patches. It is true, doubtless, that these tend to become fewer and less conspicuous as the affection becomes more and more advanced; so that it is quite possible that in some exceptional instances they may disappear, and leave the organ in a condition like that which is usually met with at a later age. But even if the means of distinguishing them should thus sometimes fail, it would not at all follow that we should forget the obvious differences between the two diseases which are presented by the majority of cases.

On the other hand, in older patients the criterion afforded by the appearance of the kidneys not infrequently ceases to be applicable. The reason is that when a part of the renal substance has been destroyed by cirrhosis the remainder is apt to become affected with the parenchymatous change. Thus the records of *post-mortem* examinations at Guy's Hospital furnish no fewer than thirteen cases in which the kidneys of patients who had been the subject of gout, and whose joints contained urate of soda, were wasted and granular, but at the same time whitish yellow in colour. Of most of these cases the clinical history given in the *post-mortem* notes is too imperfect to enable one to determine satisfactorily the period at which the second affection supervened. But in 1873, a woman aged forty-four, a gin-drinker, died after an illness of seven weeks' duration, which she said began one day with pains in the loins while she was working in a cold wash-house. On the following day the face was swollen, vomiting then set in, and afterwards the legs and the abdomen became swollen. The urine was albuminous, of sp. gr. 1002. After death the kidneys were found mottled with yellow; but they weighed only eight ounces, they were granular on the surface, and their cortex was much narrowed. The arteries were rigid. The heart weighed twelve ounces, the left ventricle being hypertrophied.

No doubt the pathologist, if he has no clinical history to guide him, is unable to distinguish such "mixed" cases as these from cases of primary parenchymatous nephritis in its most advanced or granular stage; though even without any history he may be guided to the right conclusion if he discovers the gouty origin of the disease from the presence of urate of soda in the joints. But in this country the "mixed" cases after all form an insignificant minority in comparison with the very numerous cases in which the kidneys are purely cirrhotic.

Ætiology.—What proportion of cases of renal cirrhosis are associated with gouty deposits in the joints it is impossible to say, for unfortunately the joints are still too often forgotten in *post-mortem* examinations. But we have very often found the joints affected in the bodies of those who were not known to have had any gouty attack. At the meeting of the International Congress in 1881, Dr Ord stated the results of autopsies made at St Thomas's Hospital, on twenty-four or twenty-five cases of granular disease of the kidneys; in sixteen gouty deposits were present in the joints, in eight or nine they were absent.

It is not unlikely that the rarity of gout in Germany brings with it a corresponding rarity of renal cirrhosis, and this may be the chief reason why some German writers fail to recognise the latter affection as distinct from other forms of Bright's disease. But they speak of a "senile atrophy," which they regard as devoid of clinical importance; and probably under that name they put aside many of the less marked instances, especially when the cause of death is not obviously dependent upon the state of the kidneys.

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Few pathologists would admit that in this country the kidneys are naturally liable to any purely senile change, except slight shrinking corresponding with the loss of weight in the tissues generally that comes with advancing years. At the same time we may admit that granular degeneration of the kidneys is associated with vascular and other changes which belong to the latter periods of life, and in the exceptional cases in which it occurs under thirty-five years of age there may often be found other indications of premature old age.

Next to gout, the best known cause of cirrhosis of the kidneys is chronic poisoning by *lead*. The disease is accordingly seen in painters, in printers, and in type-founders, and sometimes in those whose tissues have accidentally become impregnated with the metal. But lead-poisoning also produces gout, and therefore it is difficult to prove the extent of its direct effect in leading to renal cirrhosis.

Most observers think that excessive indulgence in *alcohol*, altogether apart from its tendency to set up gout, is a common cause of granular atrophy of the kidneys. Although Dr Dickinson gives reasons for doubting this conclusion, the general impression is strongly in its favour.* The reports of *post-mortem* examinations at Guy's Hospital do not bear out the statement sometimes made that the renal affection is frequently found in association with cirrhosis of the liver, in spite of the apparent resemblance between the two diseases from an histological point of view.† Dr Pitt found granular kidneys in one fourth of the cases of cirrhosis of the liver which he examined at Guy's Hospital, and Dr Hadden in one sixth of those at St Thomas's Hospital. Moreover, if lead produces gout, so does drink, and therefore gouty nephritis may be mistaken for directly alcoholic nephritis. Perhaps one of the best arguments in favour of intemperance leading to the more chronic forms of Bright's disease is that it has been so generally assumed without argument by men of wide clinical experience, including Bright himself. There is no belief, as there is no evidence, that syphilis is a cause of nephritis.

Chronic interstitial nephritis, secondary to affections of the excretory apparatus of the kidney, will be treated separately as *consecutive* cirrhosis (p. 502).

Age and sex.—This form of Bright's disease is almost unknown in youth and early adult life. Out of 121 cases at Guy's Hospital death occurred in eighteen between 31 and 40 years, in thirty-nine between 41 and 50, in thirty-six between 51 and 60, in twenty-four between 61 and 70, in four between 71 and 80. Below the age of 30 typical instances are rarely, if ever, met with, but several of the patients between 35 and 40 had suffered from gout, and presented the disease in a perfectly characteristic form. These figures correspond pretty closely with those given by Dr Dickinson. They are, of course, very unlike those of Wagner, who mixes up together all forms of "contracted kidney." Eichhorst, however, gives 40 to 60 as the usual age in Germany.

The proportion of males to females in the Guy's Hospital cases was almost exactly as two to one; and in Dr Dickinson's cases it was 165 to 85.

Anatomy.‡—In its earlier stages renal cirrhosis is by no means a con-

* See Sir Wm. Roberts's criticism on Dr Dickinson's statistics ('Brit. Med. Journ., November 4, 1871).

† 'Guy's Hosp. Rep.,' 3rd series, vol. xx, p. 196.

‡ The following account is based upon notes of considerably more than 100 examples of primary and uncomplicated renal cirrhosis, inspected by the author, and entered by him in the volumes of *post-mortem* records at Guy's Hospital.

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spicuous morbid change. The kidneys may be of natural size and colour; and beyond the fact that the normal arrangement of the pyramids of Ferrein is no longer visible upon the cut surface of the cortex, and that the capsule is thick and too adherent, there may be nothing to suggest disease, so that one is sometimes surprised to find with the microscope to what an extent the cortex has been destroyed.

In advanced stages the case is very different. The capsule is greatly thickened, and cannot be stripped off without the cortex tearing and coming away with it. The surface of the kidney is covered with little projections or granulations, which, in uncomplicated cases, are of a red or reddish-grey colour. Hence the phrase "raspberry kidney" applied to this granular degeneration. They no doubt consist (like the granules of a cirrhotic liver) of portions of the cortex which are less altered and wasted than the rest. Kelsch and Charcot maintain that they correspond in position with the summits of the "medullary rays" or columns of straight tubes that traverse the cortex, the depressions between them answering to the intervening tracts of convoluted tubes; but Weigert and Wagner dispute this statement. The reduction in the thickness of the superficial parts of the cortex is often extreme. The bases of the pyramids may be separated from the surface of the kidney by a layer of tissue only a line or so thick. The interpyramidal portions of the cortex are not generally wasted to the same degree; indeed, in the author's belief, they undergo a compensatory hypertrophy during the early stages of the process.

The weight of the two kidneys is often reduced from eight or nine ounces to five, four, three, and sometimes even to two and a half ounces. The smallest kidneys we have seen at Guy's Hospital were found by Dr Wilks to weigh together thirty grains short of an ounce and a half. Yet neither the diminution in size, nor the loss of weight, gives an adequate idea of the destruction of the renal cortex; for the pelvis is proportionately wider than in the healthy organ, and is filled with what appears an excessive amount of adipose tissue, over which the substance of the kidney is spread out as a thin shell.

Histologically, the tubular structure of the renal cortex is found to be replaced by connective tissue in various stages of development. At first there is merely a "small-cell infiltration," which forms foci scattered here and there through the organ, especially round the capsules of the glomeruli, but with prolongations between the tubes immediately adjacent to them. Gradually the beautiful pattern of the renal cortex is disturbed. The tubules are squeezed here and dilated there; they lose their epithelium and become obliterated, or converted into minute cysts. The glomeruli are at first less affected than the rest of the vessels, and the Malpighian capsules less than the tubules, so that they may sometimes seem little altered, though crowded together by the atrophy of the tissue between. The intertubular blood-vessels and lymphatics, with remains of shrunken tubes, form the elements of the hard and bloodless new tissue. Ultimately the exuded leucocytes undergo conversion into new tissue, in which there are generally very few blood-vessels, though these are sometimes remarkably wide. Relics of tubes are usually to be seen embedded in this tissue, and are filled with cells unlike normal renal epithelium or with hyaline casts. There are also areas in which the tubes are comparatively unaltered, except that they are too wide and patent, with flattened epithelium, and here the glomeruli are often considerably larger than natural.

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Sometimes hardly a trace of renal structure can be detected over extensive tracts. The glomeruli and Malpighian capsules are more or less completely destroyed. The capsules often become thickened by the formation of concentric layers, giving them a fibrillated appearance. The tufts degenerate into a structureless material, containing only a few scattered nuclei. By the shrinking of the intervening tissues these "glassy globes" are often drawn close together, so that a large number of them are seen in the same microscopic field. Ultimately nothing may be left but round translucent masses, of which the diameter is not more than one half or one third of that of the normal glomerulus.*

It is held by some German pathologists that degeneration of the glomeruli is the primary change in the tissue of the kidney in this disease, and that it again is dependent upon an *arteritis obliterans* affecting the smaller branches of the renal artery in the cortex, and the afferent vessels of the tufts. Thoma has shown ('Virchow's Archiv,' 1877) that, when a glomerulus becomes obsolete, its afferent artery may remain pervious, and may open straight into the efferent vessel or into the interlobular capillary network. The thickening of the larger arterial branches is undoubtedly one of the most obvious morbid appearances in a red granular kidney; they stand out upon the cut surface of the organ with patent mouths, like so many little quills. But it is very doubtful whether the arterial lesion is the starting-point of the whole process. The theory just stated approaches very near to Gull's and Sutton's of primary arterial capillary fibrosis, of which the granular kidneys are only one result. But it is far more probable that the two processes are concomitant.

Dr Johnson held that the disease begins in the convoluted tubules which shed their epithelium instead of retaining it as in the large white kidney, and accordingly proposed the name of "desquamative nephritis" for granular degeneration or cirrhosis of the kidneys ('Med.-Chir. Trans.,' vol. xxx).

A study of the early stages of the disease make it, however, more likely that it begins as subacute inflammation. In response to repeated but slight irritation, leucocytes are exuded between the tubules, and then gradually form a granulation-tissue which closely contracts, squeezing the tubules, destroying their epithelium, and thus reproducing Virchow's type of chronic interstitial inflammation with cicatrisation, fibrosis, and contraction, which we have seen exemplified in sclerosis of the cord, fibroid pneumonia, and cirrhosis of the liver.

In many cases the histological characters of a cirrhotic kidney are further complicated by the presence of innumerable minute *cysts*; cases of this kind have been separately described as "micro-cystic" kidneys. The great majority of the cysts are invisible to the naked eye, but some may be of all sizes up to that of a pea or even larger. They may make up by far the larger part of a microscopical section. Their contents are often a yellowish-brown jelly-like substance, which can be turned out of the larger cysts as a solid mass. With regard to their nature there was at one time some controversy. It was thought that they might be overgrown epithelial cells, or dilated Malpighian capsules. But the former mode of origin seems very improbable, and the latter would not account for more than a fraction

* For instructive figures illustrating renal cirrhosis see Dr Dickinson's 5th plate, Dr Coats's fig. 274, and figs. 65 and 66 in Dr Woodhead's 'Practical Pathology.' Also Dr Saundby's and Dr Greenfield's plates in the 'Path. Trans.' for 1880, with their comments.

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of the numberless cysts that are often present. Probably they consist of portions of tubes that have become cut off, and have assumed a spherical shape as "retention-cysts." Not only are they sometimes arranged in rows, like beads in a necklace, but intermediate forms are often met with—cylindrical cavities with constrictions here and there in their course. This explanation is now generally accepted, and if correct it confirms the view above advocated of the general pathology of the disease.

Another appearance that is observed in cirrhotic kidneys is due to the deposition of *lithic acid* or of its salts in the renal tissues. It consists in the presence of minute whitish-yellow grains scattered through the cortex, or arranged in lines in the pyramids, the summits of which occasionally appear to be encrusted by them. Some of these deposits are amorphous, some are made up of bundles of acicular crystals. They appear to lie partly between the tubes, partly in their interior. It has been supposed that such deposits are the results of gout, and that, acting as foreign bodies, they produce albuminuria and set up the renal disease in association with which they are found after death. But they are found in cases in which there is no other evidence of gout, and they are frequently met with in Germany, where gout is rare.

The urine.—In the slighter degrees, or in the earlier stages, of cirrhosis of the kidneys, the urine may be normal in every respect. Dr Grainger Stewart, for instance, relates a case of a man of sixty-five, who died of phthisis, and who passed but forty ounces daily, the specific gravity being 1020; yet the kidneys were found to be granular after his death. But when the disease is advanced, the quantity of urine is increased and its specific gravity is low.

The *quantity* amounts to a total of 70 to 200 ounces or more daily; in a case recorded by Bartels it was measured on a single occasion from 8 p.m. to 8 a.m., and was found during that period to reach 210 ounces. It is commonly secreted more abundantly at night than in the day. Bartels had the urine of one of his patients measured for twenty-six days, that passed from 10 p.m. to 7 a.m. being separated from that passed from 7 a.m. to 10 p.m.; during the nine hours of night the average quantity was found to be seventy-seven ounces; during the fifteen hours of day only forty-eight ounces.

The specific gravity varies from 1004 to 1010. The urine is faintly acid, pale and clear, depositing no sediment, or only a very slight cloud.

It usually contains only a small quantity of *albumen*, less than ·5 per cent.; the whole amount of albumen in the twenty-four hours is said by Wagner to be not more than from twenty to ninety grains. Indeed, unless care be used in applying the tests, the presence of albumen may be altogether overlooked; after pouring the urine upon nitric acid one must allow two or three minutes for the opalescent zone to appear.

It may happen that albumen is entirely absent for days together; or the urine passed during the night may be constantly free from it, while it is as regularly present in that passed during the day. That the urine is ever ex-albuminous throughout the whole course of the disease is extremely improbable. There have been cases in which no albumen has ever been detected, but the doubt is whether the urine has been tested sufficiently often and carefully. When albumen is more or less constantly present, it is often more abundant in the day than in the night; and in the urine passed after meals than in the *urina sanguinis* of the early morning, which the patient is often told to bring.

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Casts are often entirely absent; when any are found they are commonly hyaline and narrow, but sometimes opaque and granular.

From time to time the urine may become scanty and the albumen in it abundant or even tinged with blood. This can usually be explained by the supervention of intercurrent nephritis, as bronchitis may occur in the course of chronic phthisis.

Another cause of the hæmaturia which not infrequently appears during the course of the most chronic cases of renal cirrhosis, is probably hæmorrhage from the degenerated renal arterioles, in fact hæmorrhage of the same kind as the epistaxis and apoplexy of Bright's disease (p. 468).

The amount of *urea* secreted daily seems, according to recent observations, to be not so deficient as used to be supposed. It is true that the proportion in a given quantity of urine is generally not more than 1 or 2 per cent., but so abundant is the urine that Bartels found in four of his cases that the daily average of urea ranged from 296 to 522 grains.

Even when the quantity of urine passed is not noticed to be excessive, the patient may be obliged to get up out of bed three or four times every night to micturate. He sometimes will complain of this, but one has often to ask whether it is so. If nocturnal irritability of the bladder has existed for a considerable time, it may be fairly inferred that the renal affection is of at least as long standing.

Gouty persons who have been accustomed to pass high-coloured urine, becoming thick with urates, are often deluded by the idea that the kidneys are performing their functions much better than before, when the secretion becomes pale and clear as the result of the development of cirrhotic changes.

Again, when the flow is much increased, with consequently great thirst, patients sometimes take alarm and seek advice for what they imagine to be diabetes. When the urine is tested and no sugar is found in it, this supposition is of course negatived. But, if albumen happens for a time to be absent, the diagnosis of *diabetes insipidus* may be wrongly given.

Other symptoms.—The character of the *pulse*, and the state of the *heart*, form very important elements in the diagnosis of cirrhosis of the kidney, especially when no albumen is found in the urine (cf. p. 462). The hypertrophy of the left ventricle, the thickening of the smaller arteries and the high tension of the pulse, common in some degree to most forms of Bright's disease, reach their fullest development in the typical cases of chronic granular degeneration. Frequently, however, towards the end of a case, when cardiac dilatation takes the place of hypertrophy, the "renal" character of the pulse disappears, and it becomes soft and irregular like that of mitral disease.

Clinical forms.—The disease comes under observation in several different ways:

1. Some patients only complain of *weakness and exhaustion*; and the most conspicuous physical change discoverable in them is that they are anæmic and wasted, with flabby muscles. A man, aged fifty-nine, with what proved to be chronic Bright's disease, was so pallid that his disease was set down during life as idiopathic ("pernicious") anæmia.

2. In certain patients the chief symptoms are *gastric or intestinal*; uncontrollable vomiting or diarrhœa, or both together. It is especially in such cases that the breath is sometimes horribly fœtid, as mentioned by Bartels. Vomiting is sometimes of a uræmic character, and of serious import. Diarrhœa towards the end of a chronic case is often uncontrollable, and depends upon intestinal catarrh with œdema of the mucous

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membrane. Occasionally ulcerative colitis is present, and is the immediate cause of death.

3. In many instances the patient comes under medical observation owing to the occurrence of *acute pneumonia*, *pleurisy*, or *pericarditis*. Peritonitis, which is not uncommon in parenchymatous nephritis, is scarcely ever seen as a complication of renal cirrhosis. Acute pleurisy is often rapidly fatal, and pericarditis, when it occurs in a case of Bright's disease, is particularly dangerous: but sometimes it seems to cause little disturbance of the general health, and subsides after a while more or less completely. Even when it is still active at the time of the patient's death the amount of lymph exuded is often but small, so that it is not always clear whether it has brought about the fatal isssue. Purulent pericarditis is, in our experience, very rare.

Inflammatory œdema of the lungs is another cause of death in cases of renal cirrhosis, though less frequent than in those of tubal nephritis.

4. *Cerebral hæmorrhage* is often the cause of death in renal cirrhosis, and in many cases it is not preceded by any symptoms that are recognised as indicating that the patient is seriously out of health, or affected with any organic disease. Sometimes the occurrence of hemiplegia is due, not to extravasation of blood, but to partial softening of the brain-substance, as the result of arterial changes.

5. *Uræmia* is of less frequent occurrence than in tubal nephritis. Among 120 cases at Guy's Hospital only fifteen or sixteen ended thus. Three of them occurred in patients between thirty-one and forty, six between forty-one and fifty, two between fifty-one and sixty, two between sixty-one and seventy, two between seventy-one and eighty. The statement commonly made that this form of kidney disease is the one above all others in which uræmia is apt to occur, appears to be traceable to the fact that the advanced stages of parenchymatous nephritis have been often mistaken for it. Headache, giddiness, and other cerebral symptoms are often complained of; but such symptoms appear in many cases to be dependent upon the diseased state of the intracranial arteries rather than upon the condition of the blood. In 1880 a man, aged forty, was admitted into Guy's Hospital for pain and heat of the head. He was found to have albuminuric retinitis, and he afterwards became melancholic with suicidal tendencies.

It is important to remember that in patients affected with Bright's disease (particularly when there is extensive destruction of the renal cortex) the administration of even a small dose of opium or morphia may be followed by fatal cerebral symptoms. Thus in one case a grain of opium, prescribed for pain in the head, appeared to be the cause of convulsions and stupor that ended in death; and in another case like results seemed to be due to the administration of a third of a grain of morphia for lead colic. A patient under the writer's care, who was suffering from cancer of the throat, died comatose from subcutaneous injection of a fourth of a grain of morphia. The kidneys were found to be the seat of chronic cirrhosis with wasting of the cortex. This caution was given by Dr Bright himself, and has generally determined practice since; but lately Dr Stephen Mackenzie has published cases in which uræmic symptoms were apparently relieved by morphia. It is possible that there may be exceptions to the general rule, but at present we do not know under what peculiar circumstances the danger may be absent.

6. Sometimes the patient comes under observation with "*renal dropsy*,"

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having the characters described above (p. 455). Such cases are, as a rule, examples of the supervention of parenchymatous nephritis upon antecedent cirrhosis, and at the autopsy the renal affection is found to be of the "mixed" kind. But, unless the patient has been under observation previously, it may be impossible to diagnose the presence of any but recent changes in the kidneys; for the state of the urine is indistinguishable from that which might be produced by them alone. In all persons beyond middle age affected with renal dropsy it is very important, with a view to prognosis, that the probability of there being chronic as well as acute lesions should be borne in mind.

7. The last and perhaps the most important clinical group of cases of cirrhosis of the kidneys is that in which the main symptoms are *cardiac*, the patient coming under observation with dyspnœa, palpitation, and dropsy of the dependent parts of the body. Dyspnœa, especially, is often an early and important symptom. Dr Mahomed ('Guy's Hosp. Rep.,' 1879) stated that such "cardiac" cases make up 17 per cent. of all those in which granular atrophy of the kidneys is found after death, and the author's analysis of a somewhat larger number of cases yields an even higher figure. After death the left ventricle is usually found dilated as well as hypertrophied, and its walls may be considered to have yielded to the excessive strain upon them. But occasionally the anatomical evidence of dilatation is by no means complete. Degeneration of the muscular tissue of the heart is probably in many cases the cause of its failure; its substance may be soft and flabby, and under the microscope the fibres may be seen to be granular, and to have lost their consistency, breaking into short fragments when an attempt is made to separate them with needles. A fibroid change in the papillary muscles of the mitral valve is not uncommonly seen; they taper gradually into the tendinous cords, instead of appearing as stout fleshy columns.

A systolic apex-murmur, having some of the characters of the mitral regurgitant bruit, but not usually audible in the axilla, may be heard in many cases; but dilatation of the left ventricle, without any lesion of the bicuspid curtains, is probably an adequate cause for such a murmur. It is not often that positive proof of regurgitation is afforded at the autopsy by the presence of "ripple lines" on the posterior wall of the left auricle, as in a case recorded by Dr Mahomed ('Guy's Hosp. Rep.,' 1879). Sometimes the edge of the anterior flap of the mitral valve is thickened, and can be readily bent inwards. The occurrence of marked endocarditis secondary to Bright's disease is certainly not common; but undoubtedly cases are every now and then seen which appear to admit of no other explanation.

Until recently cases of cardiac failure secondary to cirrhosis of the kidneys were almost always regarded during life as examples of *morbus* cordis. Over and over again, when the autopsy showed that the renal changes had reached an extreme point, the diagnosis sent down from the wards has been "mitral regurgitation." Sometimes, however, one may be fairly in doubt as to the correct interpretation of the appearances after death. The kidneys, perhaps, are of nearly average size, although hard, red, and glistening on section. Even the microscope, while revealing a certain amount of fibroid change, with degeneration of some glomeruli, may leave one in doubt whether this is more than an accidental feature of the case, especially if the patient was advanced in years. Dr Mahomed pointed out, too, that mitral stenosis and other common effects of rheumatic inflam-

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mation of the heart are not very rarely found in association with cirrhosis of the kidneys, although the relation between the two affections is probably only one of coincidence.

In the clinical diagnosis of the cardiac dropsy secondary to renal disease from that which depends on primary disease of the heart, the state of the urine does not always help us. In either case it may be scanty, high-coloured, and loaded with lithates, and may contain more or less albumen as well as casts. Wagner says, however, that in cases of renal cirrhosis, even when the urine is scanty, its specific gravity is seldom above 1012; and he cites an observation of Traube's that in extreme instances it may remain pale and of low specific gravity even when there is great obstruction to the venous circulation or (on the other hand) when some febrile disease, such as acute pneumonia, develops itself. In two of our cases, however, at Guy's Hospital, the specific gravity of the urine ranged from 1020 to 1025, although at the autopsy the kidneys weighed only seven and a half or eight ounces, and were very granular; and in a foot-note to Dr Southey's translation of Bartels' work on 'Bright's Disease' a case is mentioned in which in spite of very great wasting of the kidneys the urine had a specific gravity of 1028, and deposited lithates on cooling. Indeed, whatever may be the rule as to the more advanced stages of the affection, so long as the kidneys are not much shrunken, although the microscope may afterwards show that they have undergone extensive fibroid changes, they commonly yield a high-coloured secretion, of great density, during the time that cardiac dropsy is present.

In all such cases it is to the pulse that we must look for guidance in our diagnosis. But it is doubtful whether Dr Mahomed was right in laying stress upon a visible and tortuous condition of the temporal arteries, for this is often due to local senile changes in the coats of these vessels. Again, as this observer himself admits, in cases in which there is much cyanosis from emphysema it is not safe to rely even upon a persistent pulse at the wrist as proof of renal disease. Probably the occurrence of high arterial tension in such circumstances is comparable with the rise of blood-pressure in the arteries that is observed at a certain period of asphyxia in experiments on animals.

Course and events.—Renal cirrhosis is always an insidious disease, obscure in its origin and extremely slow in its course. The only apparent exceptions are cases which develop as the result of acute scarlatinal nephritis, but probably most if not all of these are examples of secondary atrophy—"the small white kidney" described above (p. 479).

We suspect the presence of granular degeneration when a patient complains of "rheumatic" pains, of muscular weakness and lassitude, of having to rise in the night to pass water, of slight nausea while dressing in the morning, or of troublesome rather than severe headache. The suspicion is much increased if he has had gout, or is exposed to lead-poisoning, or is intemperate in liquor. A pale, sallow face, a hard pulse, a weak or muffled first and a ringing or reduplicated second cardiac sound, with a little œdema of the eyes in the morning and of the ankles at night, make the diagnosis almost certain before we examine the urine; and if this is abundant, pale, and of low density we may be sure that we are right, even if at first neither albumen nor casts are to be found.

Such patients, judiciously treated, often go on for many years with little or no aggravation of their symptoms, and it may be very long before severe cardiac symptoms come on, or attacks of uræmia or of apoplexy.

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But they are always in danger. Exposure to cold is likely to bring on bronchitis or pleurisy, or perhaps a subacute attack of tubal nephritis with general dropsy. If they are attacked by pneumonia the prognosis is serious; and if an injury befalls them or a surgical operation is proposed, the case assumes unusual gravity. If they escape these accidents, and are not cut short by intercurrent diseases, the hypertrophied heart will probably sooner or later dilate, and they will slowly die with cardiac symptoms.

Nevertheless, with care and good fortune, the renal degeneration often seems to cease in its gradual advance, and we may see patients live on with chronic Bright's disease for ten or twenty years, and even to the full term of life.

Consecutive renal cirrhosis.—Under this name may be appropriately described an affection of the kidneys which is proved by its histological characters to be a form of Bright's disease, but which is secondary to morbid changes in the renal pelvis, or in some part of the lower urinary passages. Cases of this kind have until recently attracted but little notice. The effects of pressure upon the kidneys have, indeed, long been recognised; but although it has been well known that, when hydronephrosis occurs, the pyramids are flattened and the cortex is thinned out, it seems to have been generally assumed that such appearances are only mechanical in their origin, and independent of any inflammatory process. Both physicians and surgeons are, of course, familiar with suppurative nephritis as the result of affections of the bladder or of the urethra, but this also is a different pathological process.

Consecutive interstitial nephritis is often associated with hydronephrosis (*infra*, p. 526); but it may occur when the pelvis of the kidney is perfectly healthy, as the result of urethral stricture, prolapse of the uterus, or other cause of urinary obstruction. It is often cut short in its course by the supervention of rapidly fatal suppuration of the kidneys ("ascending nephritis"); but it is quite capable of destroying life without any such aid. An excellent account of this form of renal cirrhosis was given by Mr Marcus Beck in the fifth volume of 'Reynolds' System of Medicine' (1879).

Anatomy.—The kidneys in these cases vary. There is an acute or subacute form in which they are swollen, mottled, with red and white patches, and of a soft consistence; but far more often they are very tough and hard, of a dull white or opaque waxy yellow colour. The surface is generally smooth; it is often very adherent to the capsule, which is thickened, and connected more firmly than is natural with indurated adipose tissue around it. But sometimes there is a marked granulation of the surface, so that the appearance is exactly like that of ordinary cirrhosis of the kidneys. In 1879, in an autopsy on a boy aged twelve, who had an hypertrophied and contracted bladder and great dilatation of the ureter and of the renal pelvis on each side, the two kidneys together, with much adventitious fat about them, weighed less than two ounces. The cortex was in most places shrunken to a thin red line; but, contrasting with the rest, there were some raised, soft, pale yellow, rounded nodules.

Even when the cause of the disease is situated in the bladder, or in the urethra, the two kidneys are by no means always affected to an equal extent; and when it is in the renal pelvis on one side only, that kidney may suffer alone. Thus, in 1876, in the case of a woman aged forty-seven, who for twelve years had suffered from calculous pyelitis of the left kidney, we

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found the cortex of that kidney reduced to a thin shell of white fibrous material; the other one weighed nineteen ounces, and had undergone hypertrophy, though it was also affected with recent lardaceous and other changes which had evidently been the cause of the woman's death by uræmic coma. In another case a calculus lay in one calyx of a kidney, and the corresponding part of the cortex was narrow and granular, all the rest of the organ being healthy. Moreover, when consecutive cirrhosis affects the whole of both kidneys, it often happens that the change is far more advanced in some parts than in others, so as to produce deep puckered cicatrices. This is one of the points of difference between the consecutive and the primary or medical form of renal cirrhosis, where the difference between the two kidneys is seldom marked. The former process affects the kidneys anatomically, the latter physiologically.

The morbid process, as described by Mr Beck, is identical in its histology with that which characterises other forms of Bright's disease in which the interstitial tissue is mainly affected. There is first an accumulation of immense numbers of leucocytes in different parts of the cortex, chiefly round the Malpighian capsules, but also between the tubes. The tubal epithelium is but little altered, being at most somewhat swollen and granular; but sometimes small extravasations of blood occur within the tubes, as well as into the intertubular tissue. The glomeruli become crowded with nuclei, and gradually shrivel into transparent bodies surrounded by thickened capsules. Ultimately the small-cell growth develops into fibrous tissue.

Consecutive Bright's disease occurs in various surgical affections of the urinary organs, such as *stricture*, disease of the prostate, villous disease of the bladder, stone in the bladder. But it is also seen by physicians as a result of any of those diseases that will be enumerated in the next chapter as causes of *hydronephrosis*. It occasionally affects one kidney as a consequence of pyelitis; and in cases of tuberculous disease it has sometimes an important part in completing the work of destruction. Lastly, *procidentia uteri* and other affections of the female genitalia, which drag upon the ureters or obstruct the urethra, are important causes of consecutive cirrhosis affecting both kidneys.

The clinical *diagnosis* of this form of renal disease is beset with peculiar difficulties. The urine is often so altered as the consequence of cystitis or pyelitis that its characters lend scarcely any assistance. Even if it is free from blood and pus, it may probably contain neither albumen nor casts, though sometimes it is albuminous, and occasionally a few hyaline casts may be discovered in it. It is generally rather excessive in quantity, and of low specific gravity. But no conclusion must be drawn from the density of a single specimen, especially soon after surgical interference with the urinary passages. Mr Beck mentions a case of lithotrity in which the specific gravity of the first sample of urine passed was only 1003, whereas that of the whole twenty-four hours' urine was 1018. The quantity of urea excreted may be little, if at all below normal; certainly it is often quite as great as can be expected if one takes into account the small amount of food which the patient can eat and digest.

Nor is the presence of consecutive Bright's disease clearly indicated by any marked general symptoms. In subacute cases there is often an evening rise of temperature to 100° or 101°, whereas the morning temperature may be constantly normal or even subnormal. The patient feels weak and

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languid, and steadily loses flesh. The tongue may be very foul, with a thick white or dirty fur, and may even become dry and brown. There may be much thirst, with little appetite for food, and more or less nausea. The skin is usually moist and clammy. There is neither tenderness nor pain in the loins. The patient's mental state is often placid; and he may be drowsy, like a person slightly under the influence of opium. Death sometimes occurs by exhaustion, sometimes by the supervention of some acute disease, sometimes by deepening stupor, or by uræmia. But if the primary disease can be relieved by surgical treatment, it is surprising how all the symptoms may subside that had appeared to indicate grave renal mischief, so that after all one may be left uncertain whether such consecutive results really existed, and whether they were not merely dependent upon the primary disease itself. But even restoration to a fair state of health is no proof that the kidneys have not been damaged; and in the most chronic cases of all, in which the organs become yellowish white and tough, there are often for a long time no symptoms at all: the patient remains fairly nourished, he is not anæmic, and he eats, drinks, and sleeps as usual.

It is an important question whether this form of Bright's disease is liable, like the others, to produce cardio-vascular changes. Mr Beck says that the heart does not become enlarged, although he admits that the renal arterioles show hypertrophy of their muscular coats. Wagner, however, states that the heart was hypertrophied in each of five cases that came under his observation; in one instance there was a well-marked albuminuric retinitis, which indeed was the first symptom noticed by the patient. In two cases secondary to stricture of the urethra we found the heart weigh sixteen and nineteen ounces respectively.

Cystic disease of the kidneys.—We stated that in many cases of renal cirrhosis there are found in the kidneys immense numbers of cysts, both microscopic and visible to the naked eye. Such kidneys have been termed "micro-cystic."

There are, however, other cases in which the cysts are much larger, sometimes as big as oranges, and these have been described separately under the name of "cystic disease of the kidneys." The organs then look as if they were each made up of a mass of rounded cavities, embedded in and separated by an abundant fibrous matrix. But in these there still exist remnants of secreting tissue, by which the renal functions have, however imperfectly, been kept up. The calyces and the pelvis are little, if at all, altered; so that the affection must not be confounded with hydronephrosis. The cysts have walls of varying thickness. In the character of their contents they differ widely among themselves, even in the same kidney. Some are filled with yellow fluid, others with red, some with a gelatinous substance. The fluid always contains albumen, and sometimes blood-discs, leucocytes, or plates of cholesterine; urea and uric acid are said to be generally absent.

There seems to be no doubt that the cysts are formed out of the tubes of the cortex, exactly like those which occur in cases of renal cirrhosis, and this is a strong support of the opinion expressed by Wilks and Moxon in their work on pathological anatomy, that the affection is a form of Bright's disease. One important difference between the megalo-cystic kidneys and those in which minute cysts complicate ordinary cirrhosis is the fact that in the former they are often so enlarged as to be felt during life through the

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abdominal walls. Bright many years ago recorded such a case, in which a distinct tumour was detected in the left loin some months before death, and afterwards another was discovered in the right loin. Sir William Roberts relates a case in which he successfully diagnosed not only the renal nature of the two tumours that he discovered, but also the exact character of the disease by which the kidneys were affected; the tumours appeared to be soft, but not fluctuating, about as large as cocoa-nuts, but disproportionately long. After death one kidney weighed twenty-eight ounces, the other twenty-six. In many instances they have been much larger still. There occurred at Guy's Hospital in 1867 a case in which the right kidney weighed eighty-four ounces, the left fifty-three. In two other published cases the weight of the two kidneys together was six and a half pounds, and eight and three quarter pounds respectively. But the most remarkable case of all is one which was brought before the Pathological Society by Dr Hare (vol. iii), in which the left kidney alone weighed sixteen pounds, the right being in a comparatively early stage of the disease, so that it was only of about twice the natural size. During life a tumour filled the whole left side of the abdomen.

Such monstrous cystic kidneys are sometimes congenital, and may form a serious or insuperable obstacle to delivery of the fœtus (Virchow, 'Ges. Abh.,' p. 864). The origin of this fœtal condition is very obscure; it is supposed to depend on inflammation of the straight collecting tubes of the kidneys *in utero.*

In some cases in the adult also there is reason to suspect that the affection is of intra-uterine origin, and apparently it has ended its course, for there may be no symptoms of chronic renal disease, and none of its effects on the heart; moreover, microscopical examination shows plenty of healthy secreting tissue. In such cases compensatory hypertrophy has probably taken place after birth. In other cases, as in one observed by the editor in a man of fifty-three, there were symptoms which led to the diagnosis of renal cirrhosis during life (compare Mr Eve's case, 'Path. Trans.,' vol. xxxi, p. 164).

What is most remarkable is that these hypertrophic cystic kidneys are sometimes found in association with cysts of the liver. Dr Bristowe has twice recorded this coincidence ('Path. Trans.,' vols. vii and x), and in the same seventh volume Dr Wilks relates a similar case. Rindfleisch has met with one, and Frerichs with another. A marked example was brought before the Pathological Society in 1881 by the present writer ('Path. Trans.,' p. 117, vol. xxxii) with histological figures. As there stated, microscopical examination "proves that this extremely remarkable form of cystic degeneration is histologically the same as the ordinary microcystic form of kidney associated with the later stages of chronic Bright's disease." This is far more probable than Rindfleisch's suggestion that hypertrophic cystic kidneys may be examples of cysto-sarcoma.

The cysts in the liver seem to have a different origin—not to be retention-cysts, but to result from vacuolation of the hepatic cells: so Dr Beale concluded in his report on Dr. Bristowe's case in 1856, and the same result was independently reached from a study of the case in 1881.*

* Drs Savage and Hale White have since described two cases of general paralysis in which cystic degeneration affected not only the liver and kidneys, but also the brain, the lungs, and the heart ('Path. Trans.,' 1883, p. 1, with plate). In the same volume Dr Mahomed recorded another case of cystic liver and kidneys (xxxiv, p. 182). See also Dr Paterson's paper ('Brit. Med. Journ.,' Sept. 27, 1890, p. 735).

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The *symptoms* of cystic disease of the kidneys are like those of other forms of chronic Bright's disease. The urine is often excessive in quantity, pale and of very low specific gravity. It generally contains albumen, and occasional hæmaturia has been observed. There has sometimes been marked emaciation with great prostration of strength. But many patients have not been known to be ill until they were attacked with uncontrollable vomiting, or with uræmic convulsions and coma, or (as has happened in two instances at Guy's Hospital) with cerebral hæmorrhage.

It is obvious that, apart from the presence of abdominal tumours, the clinical recognition of this affection must depend entirely upon the extent to which the renal cortex is destroyed. Sometimes even the portions left between the cysts are more or less changed into fibrous tissue. On the other hand, a few scattered cysts, of various sizes, are frequently found after death in the bodies of those whose kidneys are in all other respects healthy; their presence is then unimportant.

Mutual relation of the several forms of Bright's disease.—At present there is general concurrence as to the broad divisions of the group of affections first recognised by Bright. The supposition of Frerichs that all the various anatomical forms present different stages of the same pathological process is now universally abandoned. The eight species of morbus Brightii once described by Rokitansky, and the thirteen described by Rayer, are scarcely remembered. The three anatomical forms recognised by Bright's successors at Guy's Hospital, Barlow, Rees, and Wilks, viz. the large red kidney of acute nephritis, the large, smooth, white kidney of subacute and chronic nephritis, and the contracted granular kidney of cirrhosis, are now generally accepted. To these Virchow added the lardaceous form of disease as a fourth variety of morbus Brightii.

The first and second forms are closely related, and may be conveniently treated together as acute and chronic stages of the same process. The fourth may either be considered, as we have considered it in this volume, independently, or may be regarded (with Bartels and Lecorché) as part of a general degeneration affecting other organs besides the kidney, and when attacking the kidney producing or complicating either the tubal or the interstitial form of Bright's disease.

Subject to these differences (which only affect nomenclature and classification) the division into acute and chronic tubal (or parenchymatous) nephritis, cirrhosis or chronic interstitial nephritis, and lardaceous disease is recognised by Johnson, Stewart, Roberts, and Dickinson in this country, by Wagner, Niemeyer, and Eichhorst in Germany, and by Charcot in France.

The origin, sequence, and exact relation of the several pathological processes remain a difficult question. Some of the chief points still disputed are the following:—

a. Does the "coarse," hard, congested kidney (the *Stauungsniere* of German authors), so often seen in cases of chronic valvular disease of the heart, go on to become either a large, rough, white, or a contracted granular kidney? Probably the true answer is that it may end in interstitial cirrhosis, but that this result is very rarely reached because the primary cardiac disease goes on much more quickly: cf. p. 444 (γ).

b. Is acute glomerular nephritis distinct from the first stage of tubal nephritis, and may it go on to produce the large white kidney? It does not

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appear to be more than a frequent complication of acute nephritis, and such cases may end in a chronic form.

c. Does the renal affection of diphtheria and other specific febrile diseases ever lead to a chronic structural lesion with the clinical symptoms of any form of Bright's disease? There is no reason to suppose that it does: the nephritis which follows scarlatina is probably a distinct process throughout.

d. Is the large white kidney always preceded by an acute stage, or may it be produced by a process of tubal nephritis which begins insidiously? While the possibility of such a process cannot be denied, the more carefully cases of renal dropsy are investigated the less is its likelihood.

e. Does the large white kidney ever become contracted and granular? This question, of the existence of a third atrophic stage of tubal nephritis, may now be considered as definitely decided in the affirmative. It is less frequent than recovery and less frequent than death in the second stage, but it undoubtedly occurs, and can be distinguished from primary cirrhosis.

f. Is the contracted granular kidney always the result of a slow and insidious process from the beginning, or does it ever originate in an acute form of nephritis, such, for instance, as that which follows scarlatina? That the latter pathological sequence is extremely rare is certain, but that exceptional cases occur there is reason to believe.

g. May lardaceous disease of the kidneys produce symptoms of Bright's disease without tubal catarrh or interstitial inflammation? Such cases must be very rare, and slight lardaceous degeneration without secondary changes in the kidneys is often found where no symptoms of Bright's disease have been present during life. The rule is for a lardaceous kidney to become a large white one; when the lardaceous change coincides with cirrhosis it is probable that the former is subsequent in date to the latter. Just as a contracted granular kidney is often complicated by tubal nephritis, so it may be complicated by lardaceous changes.

h. Is consecutive cirrhosis of one kidney pathologically identical with chronic Bright's disease? We have seen reason to affirm that it is.

i. Are hypertrophic cystic kidneys to be ranked with microcystic specimens as exceptional cases of chronic Bright's disease? This also may be answered in the affirmative if we exclude congenital cases.

Geographical distribution.—The acuter forms of Bright's disease are most common in temperate Europe, in Australia, and in the United States,* rare in colder climates, as Iceland (according to Dr Hjaltelin, quoted by Dickinson) and Canada, and rare in the south of Europe and at the Cape, in India, and in the tropics generally.

Lardaceous nephritis, being almost always secondary to suppuration or syphilis, is found wherever these conditions occur. It is relatively and perhaps absolutely more frequent in India than in Europe.

Cirrhosis of the kidneys is decidedly more common in England, Scotland, and America than on the Continent of Europe. Its prevalence is probably connected with that of gout, and both directly and indirectly with intemperance.

Occurrence in animals.—Acute tubal nephritis with hæmaturia is well known as "red water" among lambs and horned cattle.

* Here, again, Bright's disease is more common in the colder districts of New England and the middle States, and less so in the Gulf States and the dry, though cold, districts of the western plateaux.

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Chronic interstitial nephritis is, according to Mr Bland Sutton ('Path. Trans.,' vol. xxxvii, p. 579), a well-known disease in veterinary medicine, and its association with arterio-capillary fibrosis and cardiac lesions is recognised. He reports, from his own observation, changes in the medium-sized arteries and hypertropby of the left ventricle concurring with interstitial nephritis in horses.

Treatment of Bright's disease.—This must depend upon the stage of the malady, and upon the symptoms present at the time.

Acuter cases.—In the early period of parenchymatous nephritis the patient should be kept strictly in bed. It is often wise to place him between blankets, and he should always wear a flannel gown with sleeves down to the wrists, so that the arms may not get cold when they are put outside the bedclothes. The diet should consist mainly of milk, but farinaceous food may be allowed. Beef-tea and meat extracts are probably injurious from the kreatine and other stimulating nitrogenous compounds they contain.

Where there is severe lumbar pain, it may often be relieved by leeches or cupping-glasses; and this local bloodletting sometimes relieves threatening suppression of urine. Bleeding from the arm is indicated in the most acute and formidable stage.

It is well to keep up a flow of water through the glomeruli, so as to wash out the tubes and empty them of the cell-masses and casts by which they are blocked up. Thus Dr Dickinson recommends his patient to drink water freely; and Dr Grainger Stewart prescribes the diuretic salts of potass, or the spiritus ætheris nitrosi, and inhalations of oil of juniper.

More commonly we order such medicines as are supposed to be diaphoretic in their action, especially the liquor ammoniæ acetatis (the use of which has been traditional at Guy's Hospital since the days of Addison), with or without small doses of antimonial wine.

Sir William Roberts believes that the administration of the acetate or citrate of a fixed alkali, such as potass, is of benefit, because the salt is converted into a carbonate which tends to diminish the acidity of the urine, and so to prevent its irritating the kidneys as it passes through them. There seems to be no objection, at any stage of the disease, to the use of such vegetable diuretics as broom, horseradish, juniper, and uva ursi, but not much good is to be looked for from them.

Digitalis might be supposed to have an injurious effect by augmenting arterial pressure, but this, according to Mahomed, is not found to be really the case to any appreciable extent. Its chief use is to be found in the latter stages of renal cirrhosis, when the heart has yielded to the blood-pressure and become dilated, while the pulse has lost its previous hardness.

The importance of setting the skin freely in action is generally recognised.* It is best effected by *baths*. One plan, advocated by Liebermeister, is that of placing the patient in a bath at about 100°, and then gradually adding more and more hot water until it reaches 104° or 106°; he is left in the bath for half an hour, or even an hour, and is afterwards closely packed in a sheet and warm blankets for two or three hours longer, during which time profuse sweating occurs. It is said that after such a bath the weight is often reduced by from two to four pounds, and even in children by half a pound or a pound. Johnson, Ziemssen, and other

* It was strongly advocated by Dr Osborne, of Dublin, as early as 1835.

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writers advise packing the patient in a sheet wrung out of hot-water, and in one or two well-warmed blankets. We often employ "hot air baths," a lamp being placed in the bed in which the patient lies, while the bed-clothes are raised by means of a cradle so as to keep a confined space of air around his body. Any one of these methods may be repeated every day or every other day. Sometimes, however, they cause faintness or headache, and a feeling of oppression, or a rise of temperature, and they have been known to bring on uræmic convulsions. It is especially in cases attended with severe dropsy that they require to be used frequently and energetically. Their effect upon this symptom is often very striking, and they may be followed by an increased secretion of urine as well as of urea; but if there is prostration or great dyspnœa the hot bath may be dangerous.

Of late years the pharmacopœia has been enriched with a powerful diaphoretic in *pilocarpine*, the active principle of jaborandi, of which from one sixth to one third of a grain may be injected subcutaneously, or about twice that quantity taken by the mouth. It gives rise to a great flow of saliva and also to abundant sweating, but the latter effect is not always so well marked in persons who have Bright's disease as in those who are healthy. In some cases, however, it produces unpleasant symptoms, such as nausea, vomiting, or even collapse. Wagner says that these may be obviated by the administration of a little brandy, or wine, or coffee, before the pilocarpine is injected.

The regular administration of *purgatives* is also of importance. The most usual practice is to give a dose of compound jalap powder twice or three times in the week. But if this causes sickness some other preparation may be substituted for it. It is essential that mercurials should not be employed systematically, for comparatively small quantities are apt to set up salivation in persons who have diseased kidneys. This caution was given by Osborne and by Bright himself ('Guy's Hosp. Rep.' for 1836, p. 375).

When dropsy cannot be otherwise got rid of, it becomes necessary to resort to *acupuncture*. This is generally done with a needle, which is pushed into the subcutaneous tissue of the thigh, or leg, or foot, and moved about before being withdrawn, so as to make an open channel, through which the fluid may find its way and escape. The surface should be first well greased, in order that inflammation of the skin may be as much as possible prevented. It is surprising how freely the fluid will often trickle away. It should be soaked up by blankets wrapped round the limb; and these must be frequently changed and well washed before being used a second time. If four or six punctures are made, there is often a great diminution of the anasarca within a day or two; and the abdomen too may appear to be much less swollen than before. Some physicians prefer to make small incisions, or to employ Southey's tubes. The great objection to the latter methods, and, in a less degree, to puncturing the skin at all, is the danger that an erysipeloid inflammation may be set up in consequence, which may cause sloughing and prove fatal by fever or exhaustion.

For the more acute forms of *uræmia* venesection is often the best remedy; the abstraction of ten or even twenty ounces of blood may be followed by a strikingly rapid subsidence of the symptoms. A couple of leeches on each temple is one of the most efficient modes of relieving uræmic headache, and probably of warding off more dangerous symptoms. In other cases the inhalation of chloroform proves effectual.

It is generally advisable to give a drastic purge; and for this purpose we may prescribe gamboge or elaterium, a drug which, on account of its irritating

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properties, is better avoided in other circumstances. Sometimes cold affusion to the head may be employed with advantage. If further experience should confirm the idea that the occurrence of uræmia often leads to a greater activity on the part of the kidneys, the conclusion would seem to be that it should not be treated by diaphoretics, such as pilocarpine or hot-air baths.

During *convalescence* from an acute attack of Bright's disease the greatest care must be taken to prevent the occurrence of a relapse. The patient should be kept warmly clad, the use of flannel next the skin being especially insisted on. He must avoid exposure to cold, as well as bodily fatigue. The diet should be restricted in quantity, and the amount of animal food and stimulants must be very small.

At this stage, and also when the disease becomes chronic and is attended with marked anæmia, *ferruginous compounds* are very useful. The tinctura ferri perchloridi is most usually prescribed, but sometimes it is not well borne, so that some milder preparation must be substituted, such as the ferri et ammoniæ citras, or the tinctura ferri acetatis. Digitalis may be given as well; and if there is headache the bromide of potassium is very useful. It is often of great advantage to the patient to spend the winter in a hot, dry climate, such as that of the Riviera, or of Egypt.

The diet must still be carefully regulated, as regards both solid food and alcoholic drinks. Dr Johnson has published some remarkable cases of recovery in grave cases under a purely milk diet continued for weeks or months.

Chronic cases.—In renal cirrhosis the first indication is to control the arterial tension. At first sight the physiological explanation given at p. 467, according to which this is part of a system of compensation for the renal disease, might seem to involve the conclusion that it should not be interfered with by treatment. But even from that point of view it must be desirable to relieve the circulating fluid, as far as possible, of any substances which the kidneys may find difficulty in removing, and the accumulation of which renders the excessive pressure necessary. As a matter of experience, the regular administration of *purgatives* is believed by Dr Broadbent, as by Dr Mahomed, to be of great value, and even to be capable of warding off for a time the occurrence of cerebral hæmorrhage. *Nitro-glycerine*, again, is sometimes serviceable, especially in relieving uræmic dyspnœa; or recourse may be had to inhalations of nitrite of amyl, the effect of which is, however, too temporary to be of much assistance. When sedatives are needed chloral hydrate is safe, and usually efficient.

In cases of cardiac dropsy, secondary to renal cirrhosis, *digitalis* is often invaluable. By it the disease can often be kept at bay for a considerable time, and the patient may even be restored to a state of apparent health.

By such treatment with drugs, combined with strict diet, and, if possible, removal to a warm climate during winter and spring, cases of Bright's disease, both parenchymatous and interstitial, may be greatly alleviated and not infrequently cured. The statement made thirty years ago, that parenchymatous nephritis, when once established, is as hopeless as tuberculous phthisis, is certainly untrue now, even with the better prognosis of phthisis which pathology and clinical experience alike have taught us.

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RENAL CALCULI AND THEIR EFFECTS

"I had a while talked with him, first of his diseases, both in his brest of olde and in his reynes nowe by reason of grauel and stone, and of the crampe that diuers times grypeth him in his legges."—SIR THOS. MORE: *Letter to Lady Alington*, fol. 1484.

Structure and formation of calculi—Lithic or Uric acid and Lithates—Oxalate of lime—Cystine, Xanthine, Indigo, and other rare calculi—Phosphate of lime—Carbonate of lime—Fusible calculus—Ætiology of calculi.
Symptoms—hæmaturia: its other causes—nephralgia—vomiting—treatment.
Effects—obstruction and suppression of urine—atrophy of one kidney——hydronephrosis—pyelitis and renal abscess, with other causes of pyuria.
Solvent and operative treatment of calculi—Nephrotomy, nephrectomy, and nephro lithotomy.

THE great emunctory organs of the body are adapted for removal of its excreta in different physical conditions. Carbonic dioxide passes off in a gaseous state from the lungs; water from the lungs, skin, and kidneys; and insoluble solids from the bowels; while the soluble excreta are removed by the kidneys alone. Under certain physical and chemical conditions these last products are no longer held in solution, but form urinary precipitates, chiefly red sand or gravel consisting of lithic acid and the lithates, and white gravel consisting of the earthy phosphates. These have been fully discussed in a former chapter. But sometimes instead of forming crystalline or amorphous deposits, these products form larger aggregations which are known as calculi or stones, and produce characteristic and very serious symptoms.

The concretion of insoluble mineral products into calculi in the urinary passages is exactly comparable to that of gall-stones, of salivary or of prostatic calculi, and of rhinoliths; and we shall find that there is a remarkable likeness in the effects of biliary and renal calculi.

Formation of calculi.—It has long been known that a calculus is generally made up of concentric layers which often differ in composition, and that the nucleus or central part may be quite distinct in character from the rest of the stone. Most calculi are formed chiefly of lithic acid, according to Sir William Roberts five out of six; and until lately it was thought to be the most frequent constituent of their nuclei. This, however, has now been shown to be a mistake. Even when the apparent nucleus really consists of uric acid, the microscope generally shows that the very centre of all has a different composition. It consists either of an aggregation of spheroids or dumb-bells of oxalate of lime, or else of hedgehog-crystals of urate of soda. In countries where *Bilharzia hæmatobia* is endemic, its ova frequently form a nucleus. In other cases it consists of inspissated mucus, or of a small blood-clot, or of casts of the renal tubes. Ebstein, in 'Ziemssen's Cyclopædia,' mentions that he once found in the urine a deposit of epithelial cells from the renal pelvis beautifully encrusted with uric acid; the patient, a woman, afterwards had symptoms of renal colic and passed concretions, but

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he does not state whether their nuclei also consisted of epithelium. As regards the spheroids of oxalate of lime which forms the nuclei of most calculi, Dr Beale maintains that they often have their origin within the tubes of the kidney. Not only has he found dumb-bells in the substance of casts, but he describes and figures microscopic calculi, already laminated, which he says he has many times seen in the renal tubes after death. Lastly, vesical calculi are sometimes moulded on foreign bodies introduced into the bladder from without.

In the laminæ which generally make up the body of a calculus the materials are often laid down in a definite manner. Thus lithic acid appears in the form of rods or columns, piled one upon another or arranged side by side; the lithates are seen to form globules, with concentric rings; oxalate of lime may form spheroids, and dumb-bells of this substance are sometimes embedded in laminæ consisting mainly of urates. Clear crystalline layers, however, make up a large part of some concretions. These facts, which have been observed by Dr Vandyke Carter, do not seem to indicate that calculi are anything else than "precipitates or aggregations of ordinary crystalline and amorphous deposits held together by mucus," though he thinks such a theory inadequate. It is true that when the principal ingredients of a stone are removed by solvents, a small quantity of organic matter is left; but to speak of this as an "organic basis" or "matrix" is to suggest misleading analogies with the very different processes of true growth seen in bones and in shells.

Dr Beale describes a remarkable instance in which a smooth, oval urethral calculus, two inches and a quarter in length, was composed entirely of minute concretions of calcic and triple phosphates, united by a whitish material; this, however, was altogether an exceptional specimen, for it is said to have lain in the urethra for fifty years before it was extracted.*

Few calculi consist entirely of one substance. Still conspicuous chemical distinctions exist between calculi; and there are corresponding differences in their physical characters.

1. *Lithic acid calculi* ($C_5H_4N_4O_3$).—These are the commonest of all stones. They occur as extremely hard bodies, of round or oval shape; smooth on the surface or tuberculated; of an ochrey, fawn, or reddish colour. They are formed in the pelvis of the kidney. Sometimes they are passed in enormous numbers while still very small, from the size of poppy seeds up to that of mustard seeds or split peas. Not infrequently several lithic acid stones, perhaps as large as marbles, are found in the pelvis of the kidney, or in the bladder. They then often have flat surfaces or smaller facets, produced by contact with one another; the presence of such surfaces is important as an indication that the concretion is not solitary. Calculi of uric acid sometimes weigh three or four ounces.

2. *Lithate of soda* ($C_5H_3NaN_4O_3$).—These are soft concretions, of rare occurrence, which appear never to reach a large size, except by the addition of lithic acid, or some other substance. Like crystalline deposits of lithates, they occur chiefly in children.

3. *Oxalate of lime* (CaC_2O_4).—These are the only common renal calculi beside those of lithic acid. They are characterised by their extreme hardness and their rough irregular surface, whence the name of "mulberry

* In vol. xiii of the 'Pathological Transactions' is a full account of this case by Mr Haynes Walton.

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calculi." They are, however, often passed safely while still small; they then appear as smooth, rounded, greyish or brown bodies, which are compared to hemp seeds; or they may be covered with glistening crystals. As a cause of hæmaturia, pyelitis, or renal colic in middle-aged people, such little calculi are more frequent, in the author's experience, than those of any other kind. The larger "mulberry" stones are generally of a blackish-brown colour, and irregularly rounded form; Roberts, however, says that stones of the same composition are sometimes oval, smooth, and of a bluish-grey colour. When crushed, they break into sharp, angular pieces. Mulberry calculi are usually solitary, and never present in large numbers. It seldom happens that more than two of the small hemp-seed concretions are passed by the same patient, and then they are passed at long intervals.

4. *Cystine* ($C_3H_5NSO_2$).—Calculi of this substance are very rare. They are usually egg-shaped; their surface is granular, and glistens with minute crystals; they are of a honey-yellow colour, and on section look semi-translucent, like beeswax, and show indications of a radiating structure. It is curious that when exposed to daylight for a long time they slowly acquire a delicate green hue. They are of rather soft consistence, so as to be marked by the nail—a distinction from the uric acid calculi, which they somewhat resemble. They are soluble in ammonia. They may reach a considerable size, weighing as much as three or four ounces, notwithstanding the low specific gravity of cystine. Roberts describes a specimen which was passed *per urethram*, which was cylindrical, an inch and a quarter long, and weighed twenty-seven grains. They usually consist of pure cystine; but one mentioned by the same author had a nucleus of uric acid, with an outer layer made up of a mixture of uric acid and cystine. In the 'Path. Trans.' for 1880 (p. 182) Dr Shattock described a cystine calculus which in all parts contained a minute proportion of oxalate of lime, and had in its interior a defined thin layer consisting entirely of that salt: the nucleus was of cystine. In the Guy's Hosp. Museum there are ten cystine calculi successively passed by the same patient (Prep. 2144, *seq.*). Mr Jacobson has lately removed from a patient's kidney a calculus of cystine weighing 387 grains.

The following five kinds of calculus are, like cystine, so rare as to be pathological curiosities.

5. *Xanthine* ($C_5H_4N_4O_2$).—This substance was first discovered by Dr Marcet, about the year 1817, in a calculus given him by Dr Babington, and only five instances of its occurrence are on record altogether. Its chemical composition is that of uric acid, less one atom of oxygen; hence it was at one time termed "uric oxide." The characteristic test for it is analogous to the well-known murexide test for uric acid: when moistened with nitric acid it dissolves without effervescence, and on evaporation there is left a bright yellow residue; this, when cool, becomes violet-red (not purple) if treated with a solution of caustic potass. It is insoluble in cold and sparingly soluble in hot water (1 in 1000 parts at 100° C.), but is readily dissolved by liquor ammoniæ or liquor potassæ.

Hitherto xanthine calculi seem never to have been found in the renal pelvis, only in the bladder. In their physical characters they resemble uric acid calculi. One removed by Langenbeck from a child weighed 339 grains ('Guy's Hosp. Reports,' vii, 202; Museum, 2145[90]).

6. *Indigo* C_8H_5NO).—This substance, like xanthine, scarcely ever occurs

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as a urinary deposit, though it sometimes colours lithates. As a concretion it has been found only once in the renal pelvis of a middle-aged woman, by Dr Ord ('Path. Trans.,' 1878). It formed a flat broad cake, like a lozenge in shape and size, weighing forty grains; its surface was partly dark brown, partly bluish black; its section grey and polished. On paper it left a blue-black mark.

7. *Urostealith.*—This name was given by Heller in 1845 ('Heller's Arch.,' Band ii) to certain soft, elastic concretions, like india-rubber, which were passed by a young man. Dr Moore, of Dublin, has since ('Dubl. Quart. Journ.,' xvii) met with similar specimens, and in the museum of the College of Surgeons there are two which belonged to Hunter's collection. They were taken from the bladder, and perhaps the fatty salts of lime, of which they are entirely made up, were formed by the decomposition of a solution of soap, which might have been injected for therapeutical purposes (cf. p. 540, *note*). This theory, however, seems not to apply to the other cases.

In the 'Med.-Chir. Trans.' for 1872 Mr McCarthy described certain calculi of peculiar form, eleven in number, which were taken from the left kidney of a woman after death. When first removed they felt soft and greasy, and they each consisted of a central globular body, with long tapering spines projecting from it. None of these appear to have been analysed, but a somewhat similar concretion from the right kidney was found by Dr Tidy to contain 36·6 per cent. of fat and cholesterine, the other chief ingredients being lithates (35 per cent.) and oxalates (9 per cent.). Similar specimens in the museum of the College of Surgeons are said to consist of oxalate of lime. Mr Benjamin Duke, of Clapham, once showed the author a number of similar calculi removed after death.

8. *Phosphate of lime, or bone earth* ($Ca_3 2PO_4$).—All the varieties of calculi hitherto described appear to occur in acid urine; certainly this is the case with the two of clinical importance—uric acid and calcic oxalate. There is, therefore, a broad distinction between them and the three varieties which remain, for the latter can only be formed when the urine is alkaline. One, which is very rare, consists of a phosphate of lime. Concretions formed of this substance are described by Roberts as being white and chalky in appearance, and rather smooth on the surface, with an earthy fracture. Their texture is sometimes loose, sometimes very compact. They vary in size from that of a pea to that of a hen's egg. In the museum of the Manchester Infirmary there is a laminated specimen in which bone earth alternates with uric acid. A peculiarity of calculi composed of pure phosphate of lime is that while they require alkaline urine for their production, it must not be alkaline from ammonia, since, if it were, the triple phosphate could not fail to form, and to make up a large part of their substance. But even in urine which is alkaline from fixed alkali they arise very seldom.

9. *Carbonate of lime* ($CaCO_3$).—Concretions of this substance are very rarely seen in the human subject. Roberts says that when they do occur they are generally small very hard bodies, varying from the most minute size up to that of a hazel nut; grey, yellowish, or bronze-coloured; smooth on the surface, sometimes with a metallic lustre. Dr Haldane, of Edinburgh, once found a number of little calculi consisting of carbonate of lime in the dilated pelvis of the kidney of a man who had died from spinal abscess. Some years before, Roberts had met with a case in which immense numbers of precisely similar bodies were passed during life. The largest of them

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were of the size of mustard seeds, they were translucent and of an amber colour, and showed a laminated structure under the microscope. They were probably also of renal origin. The urine in which they were found was ammoniacal.*

10. *Mixed calcic and ammonio-magnesian phosphates* (" *fusible calculus* ").—We saw how the decomposition of urea into carbonate of ammonia during the alkaline fermentation of urine inevitably leads to the formation of the " triple " phosphate of ammonia and magnesia; and how this salt and the amorphous phosphate of lime come down together as a precipitate, which has a strong tendency to agglomerate into a mortar-like mass. It sometimes concretes upon the surface of the inflamed mucous membrane of the bladder, and accumulates upon any foreign body which is exposed to its action, particularly upon calculi, of whatever nature, which are washed by putrid urine. It rarely forms the starting-point of a stone, whether in the kidney or in the bladder; but it often converts a small nucleus into a concretion of enormous size. It is a soft friable substance; in the blowpipe flame it melts into a kind of enamel, whence the term " fusible calculus." In the bladder it may form stones weighing as much as twenty, thirty, or even forty ounces. In the kidney it often takes the shape of the pelvis and calyces, each branch with an expansion at its end.

Dr Gee has recorded ('Med.-Chir. Trans.,' vol. xxxix,) a case in which such a concretion weighed thirty-six and a quarter ounces; it, or rather the remains of the kidney stretched over it, had been felt during life as an abdominal tumour of stony hardness. As is often the case, its surface was covered with brilliant crystals of pure triple phosphate, but these were of exceptional size and beauty, some of the prisms being half an inch long. The whole of the calculus was very hard and dense. It had a dark brown nucleus, which consisted mainly of oxalate of lime; whether this also had a branched form is not stated. Such " coral-like " masses, sending prolongations into the several renal calyces, are only found as a result of ammoniacal decomposition of the urine in the renal pelvis. They are probably always composed of mixed phosphates. Dr Ord and Mr Wagstaffe have published similar cases in the 'Pathological Transactions.'

Ætiology.—It will be apparent from this account of the different kinds of urinary calculi that, in a patient whose urine is, and has been, acid, the only kinds frequently met with are those mainly composed either of lithic acid, or of oxalate of lime, or of alternating layers of lithic acid, lithates, and oxalate. Phosphatic calculi rarely occur except when the urine is ammoniacal, and only by deposition on a pre-existing nucleus, and this (when not a foreign body) is either lithic acid or oxalate of lime. The ætiology of primary calculus, therefore, resolves itself into the causes that favour the precipitation of uric acid or calcic oxalate from the urine.

The eastern counties of England, and especially Norfolk, are well known to yield a much larger proportion of cases of stone—at least of stone in the bladder—than any other districts of the country. But, in his address on surgery delivered before the British Medical Association in 1874, Mr Cadge, of Norwich, estimated that lithic acid and the lithates make up nine tenths of the calculi observed there, whereas for the whole of England a lower

* In vol. xix of the 'Pathological Transactions' a large renal calculus is described as composed of carbonate of lime, but in vol. xxviii it is stated that on a re-examination of the specimen it proved to consist of other materials.

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proportion (usually five sixths) is generally given. Consequently, it seems not improbable that these ingredients may alone be concerned in producing the excessive number of cases observed in the eastern counties, the frequency of calculi formed of oxalate of lime, cystine, and other substances being perhaps no greater there than elsewhere. Mr Cadge could only call to mind three cases in which he had removed an oxalate of lime stone from an adult. In one of these cases, only the outside shell consisted of oxalate, the central part being uric acid; the patient had been recently living in North Wales, having previously left Norfolk, probably with a uric acid stone already in his bladder. Another case occurred in a soldier who had only been for a short time in this county. The third was in a Norfolk man, but he also had resided elsewhere.

Why calculous deposits, or why deposits of lithic acid, should occur so frequently in East Anglia is unknown; but it is generally believed that hardness of drinking water is one cause of calculus, and the soil of Norfolk and Suffolk is mostly cretaceous.

At first sight it seems difficult to understand how this "hardness," which means abundance of earthy salts, can affect the concretion of calculi, since calcic oxalate and lithates do not occur as minerals, and chalk is scarcely deposited in the body except upon a previously existing calculus. But distilled water will dissolve lithates, oxalates, or other more or less "insoluble" substances better than water already holding other salts in solution. Hence if the blood and urine of patients, who habitually drink well-water from a calcareous soil, contain more salts in solution than in the case of those who drink rain-water—a supposition which has not yet been verified—it would follow that the small quantity of oxalate of lime or lithic acid, which was held in solution in the urine in the latter case, would begin to be deposited in the former. The chalk formation, however, is far from being confined to the eastern counties.

Calculi are said to be less common in Germany than in France and England, and to be remarkably rare in Sweden.

The hereditary influence sometimes seems to show itself in a very marked way. But persons who come to reside in Norfolk are said sometimes to form a calculus very rapidly, while others who leave the district lose the proclivity.

Another point on which Mr Cadge insisted is that in children stone is almost entirely confined to the poor, and he was inclined to think that a deficient supply of milk as food had to do with it. The late Sir William Fergusson is said to have remarked, that he only once received a fee for cutting a child for stone; whereas in a large London hospital lithotomy on boys under puberty is one of the most common operations. The only child of well-to-do parents whom he had treated for stone was said by the mother to have differed from all her other children in having never been able to take milk.

Sex and age.—Stone is far more frequent in males than in females; but this applies to stone in the bladder rather than in the kidney. In fact, the difference is probably not in the less frequent formation, but in the earlier and easier escape of calculi from the female bladder.

As regards age, vesical calculi are well known to be much less common in young adults than in children and in old people, but it is not certain whether the same rule applies to renal calculi.

Symptoms and effects of renal calculi.—The morbid conditions produced by

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the presence of gravel or calculi in the kidney may be classed together as "nephrolithiasis." They depend upon irritation of the renal pelvis, or upon mechanical obstruction of the ureter, and may be arranged as follows—the first three are symptoms, the rest pathological effects:

1. *Pain in the loins*, or renal lumbago.
2. *Hæmaturia*, the most constant effect of calculi.
3. *Renal colic*, consisting of nephralgia with vomiting, and produced by the passage of a stone into the ureter.
4. *Obstructive anuria*, or *suppression of urine*, caused by impaction of a calculus in one ureter, when the opposite kidney is from any cause already incapable of secreting urine.
5. *Unilateral atrophy of the kidney*, from obstruction of its ureter.
6. *Hydronephrosis*, from the same cause.
7. *Pyelitis*, with *pyonephrosis* and *perinephral abscess*.

1. *Renal lumbago.*—In the mildest form of nephrolithiasis the principal symptom is a dull aching pain in the loins, commonly called *lumbago*. Such a pain is often the result of irritation of the kidneys by the urine, or by something deposited from it. It is practically difficult to distinguish such cases from those of myalgia (or, as it is vaguely called, muscular "rheumatism") affecting the lumbar muscles, for in both cases the back may feel stiff, and stooping may aggravate the pain. When, however, the urine is strongly acid and deposits lithates, relief is usually given by rendering the urine alkaline. Often, in the author's experience, the pain has been at once removed by a few full doses of the citrate of potass, repeated at short intervals. It may be a question whether the symptoms are due to an over-acid state of the urine alone, or whether there is formed in the renal pelvis an actual sediment of uric acid, which is re-dissolved when the secretion becomes alkaline. The dull aching sensation is sometimes experienced only when the patient first wakes in the morning, and ceases after he gets up, and "the alkaline tide" sets in after food (p. 413).

2. *Hæmaturia.*—The presence of blood in the urine can often be recognised at once from its colour. When hæmorrhage is profuse, the urine may look like pure blood. From this every gradation of colour may be observed down to the palest pink or the faintest brown tinge. The pink and red shades are due to the urine being neutral or alkaline, and are seen as a rule in cases of hæmorrhage from the bladder; the brown and smoky tints are caused by the hæmoglobin being changed into acid hæmatin, and therefore appear with an acid state of the urine. In many instances, when the colour is not in itself distinctive, there is a sediment which cannot be mistaken: on tilting the chamber vessel from side to side, a granular-looking reddish-brown substance may be seen lying just within the edge of the fluid, and following its movements.

In any case the microscope will at once reveal the presence of blood in a drop of urine taken from the bottom of a vessel in which it has been standing for a little while, or sometimes from the bulk of the urine directly after it is voided. It is impossible to insist too strongly upon the importance of microscopical examination in all cases in which hæmaturia may be suspected; without it the absence of blood should never be asserted. The only cases in which hæmaturia may fail of detection by the microscope are when the urine is of very low specific gravity or in a state of ammoniacal

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decomposition, for in such cases the blood-discs may be rapidly dissolved. In urine of acid reaction and a normal density they remain visible for days.

Albumen must always be contained in urine in which there is blood. But the ordinary tests often fail to show it, when the discs are at once seen with the microscope.

Blood-discs in urine do not always retain their form: they often have crenated edges, and sometimes they are shrivelled; in dilute urine they are often represented by delicate globular corpuscles slightly larger than natural, produced by endosmosis, from urine being of much lower specific gravity than blood. Minute discoid forms of oxalate of lime might be mistaken for blood-corpuscles in urine, but they are more refracting and have no yellow tinge. Dr Beale speaks of cases in which spirilla looked so like blood-discs that great care was required to distinguish them. The microscope at once distinguishes hæmaturia from hæmoglobinuria (*supra*, p. 431) by discovering the presence of abundant corpuscles. But the two conditions may be associated by each replacing the other in the same patient.

In the exceptional cases in which the microscope fails to reveal the presence of blood, the spectroscope may be used (see p. 431) or chemical tests applied. One test is known as Heller's; it consists in rendering the urine alkaline by the addition of caustic potass or soda, and then boiling, so as to precipitate the earthy phosphates, which carry down with them any blood-colouring matter that the fluid may contain. Salkowski says that this reaction is very delicate, but that it also occurs with the colouring matter of rhubarb or senna. Another test much employed in this country is adding to the suspected urine a drop of freshly prepared tincture of guaiacum, and shaking it up with a few drops of ozonic ether; if blood is present, a brilliant blue colour appears in the layer of ether that collects on the surface of the fluid when it has stood for a minute or so. The chief sources of fallacy lie in the fact that saliva, nasal mucus, and iodide of potassium give the same reaction. According to Dr Mahomed ('Med.-Chir. Trans.,' lvii) this guaiacum test is even more delicate than that of the spectroscope; but it is too uncertain to be of much practical value.

It must not be forgotten that blood may be added to urine for purposes of deception. In one such case the microscope showed that the corpuscles present were the oval blood-discs of a bird.*

Its causes.—In the first place hæmaturia may be merely a symptom of a general disease, such as purpura, scurvy, smallpox, or malarial fever. Whether hæmaturia is ever vicarious to menstruation (blood may of course be present in a healthy woman's urine during menstruation), or to a hæmorrhoidal flux, or to asthma is doubtful; such cases do not occur at present.

Secondly, and more frequently, hæmaturia is a symptom of some disease of the kidneys. The commonest are calculi and gravel. Next to these come acute nephritis, tubercular ulceration, and new growths.

Thirdly, bleeding of the bladder may be caused by stone, acute cystitis, *e. g.* from gonorrhœa, tubercular or cancerous ulceration, villous growths, or the presence of *Bilharzia hæmatobia*.

When blood is poured into the urinary passages in sufficient quantity to coagulate, symptoms of various kinds may result, which may simulate some antecedent disease to which the hæmorrhage was due. Thus a clot in the

* I once checked an attempt of this kind by remarking that it was necessary for me to see the urine passed; the patient, who had had a railway accident, subsequently confessed that he mixed with his urine blood that came from a cut upon the wrist.—C. H. F.

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ureter may produce an attack of "renal colic" just like that caused by a calculus; and clots in the bladder may give rise to dysuria, or to complete retention of urine.

Seat of hæmorrhage.—When blood-clots are passed in the urine, they often clear up all doubt as to the place of the hæmorrhage. Sometimes they are flat, and were evidently formed on the floor of the bladder; sometimes cylindrical, having come from a ureter. Blood-casts of the uriniferous tubes prove of course that the hæmorrhage is derived from the renal cortex. They are rarely found except in acute tubal nephritis.

Bleeding from the substance of the kidney is seen when the organ has been lacerated by violence, in cases of poisoning by oil of turpentine or by cantharides, and in very rare cases in which quinine, by a curious idiosyncrasy, produces a like effect ('Brit. Med. Journ.,' Jan., 1870). The renal affection which in some patients is set up by the application of a blister sometimes produces hæmaturia; sometimes only fibrin is exuded, but in such quantity as to form transparent gelatinous clots in the bladder, and obstruct the outflow of urine. A case of this kind occurred in the writer's practice in 1886, and two are related by Bartels.

We may be helped in discovering the seat of hæmaturia by the way in which the blood escapes during micturition. Should bleeding occur into the urethra, the blood precedes the stream of urine, and is washed out by it. On the other hand, when the bladder is the seat of disease, it is towards the end of micturition that the urine is most deeply discoloured. Dr Beale speaks of cases in which persons, apparently healthy, day after day pass small quantities of blood, just as micturition is ceasing; it would seem that "the effort required to expel the last drop of urine causes the rupture of a few capillaries about the membranous part of the urethra or the neck of the bladder." In some instances hæmaturia has been caused by undue sexual indulgence. It usually ceases after a time if the patient rests. When there is hæmorrhage from the renal pelvis, the blood is always intimately mixed with the urine; but so it is likewise in many cases in which its origin is from the bladder.

When the urine is of red or pink hue this is due to the reaction being alkaline, and hence the seat of hæmorrhage is most likely the bladder.

If the urine, instead of being bright red, has a brownish hue ("like tea"), this shows that the blood-corpuscles have been acted on by acid, and their hæmoglobin turned to acid hæmatin; the probability is that it came from the kidney. But whenever the hæmorrhage is profuse the urine remains red; for it then neutralises the smaller amount of acid urine.

After all, it must be confessed that in many cases, including the most severe, there are no certain indications as to the seat of the hæmorrhage, except from other symptoms, such as pain or dysuria.

In practice, however, almost the only *vesical* affections that give rise to profuse hæmaturia, as their sole symptom, are villous tumours and other forms of new growth, generally malignant. Sir George Humphry believes that the occasional cessation of hæmaturia for a long period affords an indication in favour of the diagnosis of villous disease of the bladder. Such diseases occur chiefly in patients who have already reached middle age or passed beyond it; but in 1865 a child, only eighteen months old, died in Guy's Hospital of the effects of a polypoid growth from the right side of the neck of the bladder. The writer had once sent him from Mr Bryant a minute clot which was passed with the urine of a little girl in apparent health, and

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only five years old; on microscopic examination a beautiful tuft of villous growth was apparent. In 1877 an autopsy was made in the case of a man aged thirty-four, who said that ever since he was twelve years old he had suffered from hæmaturia, recurring at intervals of weeks or months, with greater or less severity. There was found to be a soft spindle-cell sarcoma, growing as a flat, slightly lobulated tumour from the base of the bladder on one side. It is a point of some importance that in a case of Murchison's ('Path. Trans.,' 1869), villous growths from the pelves of both kidneys were associated with a like affection of the bladder. Mr Davies-Colley once succeeded ('Clin. Soc. Trans.,' 1881) in removing through a perinæal incision a villous growth from the bladder of a man aged thirty-two, who had suffered for eight years from hæmaturia, and was completely cured by the operation. This patient sometimes passed blood at the beginning of micturition, sometimes at its close, the explanation no doubt being that the villi were occasionally nipped in the prostatic part of the urethra. Sir Henry Thompson has published several similar cases.

Hæmorrhage from the *renal pelvis* may be due to various causes. When a patient passes blood in the urine without there being other symptoms to throw light upon the nature of the disease, the presumption is generally in favour of the presence of a renal calculus; but the possibility that tubercle or cancer of the kidney may be developing itself must never be left out of consideration. It is, however, surprising how often one meets in practice with instances in which profuse hæmaturia causes for the time the utmost alarm, without there being any clue as to its source, and ceases after two or three days, leaving the patient as well as ever.

In 1881 a man aged sixty-three was admitted into Guy's Hospital with extreme wasting and cachexia. After a few days he was attacked with severe hæmaturia. This, however, quickly subsided, though afterwards pus appeared in the urine, and three weeks later he died. At the autopsy it was found that the cause of the wasting was cancer of the œsophagus. In one of the calyces of the left kidney a calculus was impacted. The lining of the renal pelvis was much thickened, œdematous, and of a deep purple colour from ecchymosis, looking like velvet. Probably a like condition is generally present when hæmaturia is the main symptom.

It is an interesting question whether bleeding ever occurs as a result of the irritation produced by mere granular deposits of uric acid or oxalate of lime, or whether the presence of a larger concretion is necessary. A certain answer to this question can hardly be expected; but Sir Benjamin Brodie and others taught that "red sand" (lithic acid) is capable at times of causing hæmaturia. A point of considerable clinical importance is that jolting movements of the body are exceedingly apt to bring on, or to aggravate, hæmaturia in persons who have even small calculous concretions in the kidneys. Not only does this occur after horse exercise, but also after riding in a carriage with springs. Even when no blood is obvious to the naked eye, it is always worth while to make a microscopic examination of the urine passed under such circumstances by a patient in whom renal calculus is suspected. In most cases of this kind there is lumbar pain, or pain referred to the front of the abdomen on one side, or to the groin. Another circumstance, to which Brodie drew special attention, is that the symptoms are sometimes referred mainly to the bladder. Micturition may be frequent, and accompanied by a cutting pain in the neck of the bladder and in the urethra, so that the presence of a vesical calculus is suspected.

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Treatment.—Whatever the cause of hæmaturia, rest in bed is essential. If cold is applied, it should be as near the seat of hæmorrhage as possible; if it be from the kidney, ice-bags to the loin; if from the bladder, ice-bags to the hypogastric region of the abdomen, or injections of iced water into the rectum or into the bladder itself. Prout found the injection into the bladder of a solution of alum (twenty to forty grains in a pint of water) very effectual when the hæmorrhage was vesical. As internal styptics, gallic acid, acetate of lead, ergot, or alum may be employed. In some cases the tincture of perchloride of iron is particularly serviceable. Oil of turpentine, too, may succeed when all other drugs have failed; it would probably be injurious if the blood came from the renal cortex, but in cases of hæmorrhage from the pelvis of the kidney there is no objection to its use. The hæmaturia caused by cantharides is treated by cupping to the loins, warm poultices, diaphoretics, and purgatives.

On the whole, drugs have less effect on hæmaturia than on most other forms of hæmorrhage,—less, for example, than ergot on hæmoptysis, or opium and lead on bleeding from the bowels.*

Hæmaturia, though so important as a symptom, is not a serious occurrence in itself. It is far less dangerous than intestinal, gastric or pulmonary hæmorrhage; indeed, few physicians have seen a fatal case of bleeding from the kidneys.

In the treatment of recurrent or persistent hæmaturia attributed to renal calculus the first thing is, if possible, to get rid of the cause of the disease. This will be considered at the close of the present chapter (p. 540).

3. *Nephralgia.*—The passage of a calculus down the ureter into the bladder is attended with special symptoms, which are commonly known as *renal colic.* They often set in with extreme suddenness and violence, and constitute one of the most painful of diseases. The patient is sometimes awakened from sleep by an attack; sometimes it is brought on by the jolting of a vehicle, or by some muscular effort, such as sneezing, coughing, running, jumping, or riding on horseback.

The *pain* is generally described as running from one loin downwards in the direction of the ureter; but it may also spread over the whole of the abdomen, or radiate to the chest or to the shoulder-blade, or appear to run along the costal cartilages or the iliac crest. Very commonly it shoots down into the corresponding testicle, which is drawn up towards the inguinal canal, and is distinctly swollen as well as tender. There may be pain, too, along the inner side of the thigh, with numbness and tingling of the skin there. The suffering is often intense; the patient grows faint and cold, and breaks out into a profuse sweat; the pulse becomes very rapid and small,† the breathing is quickened, and the temperature may presently rise.

* "I do not lay any great stress upon the use of internal astringent remedies (for hæmorrhages), because it does not appear likely from reasoning that they should do any service, and I am far from being convinced by experiment that they ever do, except perhaps in hæmorrhages of the primæ viæ. They may sometimes have appeared to be attended with success, because there is but a small proportion of hæmorrhages, not owing to internal violence, which would prove fatal though no means were used to stop them" (Heberden). A recent expression of opinion agrees with that of this wise physician more than a hundred years ago: Dr Saundby says of the use of drugs in hæmaturia, "My experience has been that they are all very untrustworthy, and I hesitate to give the preference to any one" ('Brit. Med. Journ.,' Dec. 17th, 1887).

† Traube, however, recorded a case in which, during repeated attacks of renal colic, the pulse was slow, full, and remarkably tense. Heberden had remarked the same fact.

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Vomiting and nausea are marked symptoms, the matters rejected from the stomach often becoming bilious after a time. Epileptiform convulsions have sometimes been observed. In pregnant women abortion frequently takes place; cases are even recorded in which successive pregnancies have been brought to a termination at considerable intervals of time by the supervention of attacks of renal colic.

In the hope of relieving pain, the patient adopts the most extraordinary positions. A medical friend of the author found that the only attitude which seemed to give him any comfort was kneeling with the head bent over, so as to touch the ground. But some persons prefer to lie on the side with the knees drawn up. Movements of the body generally increase the pain, and yet the restlessness is so great that it is often impossible for the patient to remain in one position. The paroxysm, if it lasts long, is generally interrupted by remissions of the pain, which soon, however, becomes as bad again as ever. The duration is very variable; it may be over in the course of a few hours, or it may last for several days. Its termination is sometimes quite sudden; the patient, perhaps, during a violent fit of retching, may experience a sensation as though he were stabbed, and from that moment the acute suffering ceases, for the stone has slipped into the bladder.

Micturition during an attack of renal colic is generally frequent, and sometimes there is severe strangury, with burning pain in the urethra or at the end of the meatus. Often only a few drops of urine are voided at a time, and they are often deeply tinged with blood; but if the kidney on the opposite side is healthy, it may go on pouring out a normal secretion. Ebstein remarks that in cases of calculous pyelitis, in which the urine is habitually discoloured by blood and pus, the fact that it becomes normal when a stone is impacted in the ureter affords valuable evidence that the other kidney is not affected in the same way. But it often happens that calculi are present in both kidneys, so that the urine from that which is unobstructed is purulent or blood-stained, or perhaps of low specific gravity and albuminous, as the result of consecutive Bright's disease (p. 499). Those cases in which at the time when an attack of renal colic occurs the opposite kidney is absent, or so atrophied as to be unable to secrete any urine, will be specially described in the next section.

It is doubtful whether the passage of a calculus is dangerous to life when the other kidney is healthy. Ebstein speaks of the possibility of its ulcerating through the walls of the ureter and escaping, so as to set up fatal peritonitis. But the case which he cites from Allan Webb ('Pathologia Indica,' 1846) was not uncomplicated, for "the vermiform appendix and the ureter were found ulcerated and adherent to one another and to the surrounding structures, and a large amount of pus had escaped from the ulcerated ureter into the abdominal cavity."

On the other hand, it does not seem that the subsidence of an attack of renal colic is a complete proof that the stone has passed into the bladder. Possibly it sometimes undergoes disintegration in its course downwards, and the fragments escape unnoticed in the urine. In other cases it remains permanently impacted, while the kidney undergoes atrophy or passes into a state of hydronephrosis, as will be presently described.

The fact that the pain may cease while its apparent cause is still there, affords an argument in favour of an opinion held by Traube that nephralgia really is due, not so much to the direct irritation of the mucous

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membrane by the calculus, as to the peristaltic movements which take place in the over-distended ureter above.

A ureter which has given passage to a stone may be found considerably wider than natural when death occurs from some other cause, after the lapse perhaps of many years; in one case the vesical orifice of the tube was large enough to admit a thick probe. The occurrence of such dilatation of the tube explains the fact that when attacks of renal colic are frequently repeated, the later ones are comparatively slight and of much shorter duration; but of course a great deal depends upon the shape and the size of the calculus on each occasion.

Sometimes the affection recurs at tolerably regular intervals. Sometimes a calculus is passed by a patient who never before showed any symptoms of urinary disorder; and this is especially frequent when the concretion consists of oxalate of lime. An attack of renal colic occasionally puts an end to pyelitis which had existed for a considerable time; in such a case one may infer that the renal pelvis contained a solitary stone, which was the cause of the previous symptoms, and which has now escaped into the bladder. Prout, however, remarks that after the passage of an oxalate of lime calculus painful sensations often continue to be experienced for some time, so that he had found difficulty in convincing his patient that other calculi did not remain behind.

In making a *diagnosis* we must not suppose that the occurrence of renal colic is in itself proof of the passage of a stone into the ureter. In some cases (as, for example, in one recorded by Traube, in which the pain for some time returned every night, lasting about five hours, but in which no calculus was ever voided) it seems more likely that the attacks are due to the presence of a stone in the renal pelvis, just as hepatic colic seems sometimes to arise from a gall-stone which has never left the gall-bladder. Again, precisely similar seizures may arise from the ureter becoming blocked in other ways; as, for instance, by small hydatid cysts, or even by clots of blood. Thus, in cases in which there is profuse hæmaturia, the occurrence of violent pain along the course of the ureter must not be taken as establishing the fact that the bleeding is due to a calculus rather than to cancer. Again, in a remarkable case recorded by Dr Dickinson in the 'Pathological Society's Transactions' for 1875, an abdominal aneurysm over which the ureter was stretched gave rise for a long period to paroxysms of pain exactly like those of renal colic, and once attended with swelling of the testicle. In the immense majority of cases, however, renal colic is a symptom of stone in the pelvis of the kidney.

The *treatment* of renal colic consists mainly in the administration of anodynes, though something may perhaps be done to facilitate the release of the calculus. Thus, if the abdomen is not too tumid, friction may be used in the course of the ureter; or while the affected side is being rubbed, the patient may be held head downwards, as was done by Sir James Simpson with success in two cases, the concretion apparently falling back into the pelvis of the kidney. Venesection was formerly had recourse to, and numerous leeches were employed; but it is better to avoid such methods of relieving what is essentially a transitory affection. A hot bath is often of service, or hot fomentations to the loins. But our chief reliance must be placed on the free use of opium. The hypodermic injection of morphia is to be preferred on account of the rapidity and certainty of its action, but if this should be objected to, and vomiting prevent a draught being retained,

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fifteen or twenty minims of tincture of opium may be thrown up into the rectum. The dose will probably have to be repeated at rather frequent intervals. The inhalation of chloroform often answers better than anything else, but a hot bath is almost always a useful adjuvant.

4. *Suppression of urine.*—In the chapter on cholera (vol. i, p. 283) the fact was mentioned that the secretion of urine may, for a time, be completely suppressed, the patient voiding none, and none being found in the bladder when a catheter is passed. Poisoning by turpentine may produce a like effect; and in some remarkable instances it has been observed after an operation upon the urethra, or even after passing a catheter. It is also a symptom of suppurative nephritis, and of the most acute form of Bright's disease. Such cases of "*non-obstructive suppression*" (as Roberts terms them) end fatally in a few hours or in a day or two, unless the kidneys resume their function. When the affection is recovered from, the urine that is first passed is scanty, high-coloured, and generally albuminous or even bloody. The best treatment appears to consist in the use of the hot bath, or the application of hot mustard poultices to the loins, and the injection of hot gruel into the rectum.

In other cases, a temporary suppression of urine, without obstruction of the ureters, occurs as part of the general *shock* or collapse produced by a large dose of an irritant poison, or by sudden lesions, such as perforation of the stomach, acute intestinal strangulation, or rupture of the uterus. The renal affection is then relatively unimportant, passing off when the patient rallies, and generally leaving no ill-effects behind it.

According to Charcot, hysterical women are liable to a very different kind of suppression of urine, which he terms *hysterical ischuria*, and which may continue for a long time, almost without interruption, without seriously disturbing the health. He describes one of his patients as voiding less than a teaspoonful on an average each day for weeks together, whereas in the matters which she vomited urea was present. It is difficult to believe that fraud was not practised in this and other like cases, although Charcot is convinced that he completely guarded himself against it.

A complete contrast to these cases is presented by *obstructive suppression*. When this occurs, the patient, instead of dying within a day or two, goes on for seven or eight days without obviously grave symptoms, so that both he himself and his friends find it difficult to imagine that there is danger. He is calm and free from distress, with an unclouded intellect, and with natural pulse, respiration, and temperature. He may be able to take food, the tongue may be clean, and there may be neither nausea nor vomiting. The muscular strength, however, begins to fail, and there is often marked sleeplessness. There is no desire to micturate, and sometimes no urine at all is voided. Generally, however, at irregular intervals, the bladder discharges a few ounces, or sometimes a pint of urine. This is always pale and watery and of very low specific gravity; and, unless tinged with blood, it is usually free from albumen. At the end of about a week symptoms appear, which are commonly followed by a fatal termination within two or three days at the latest. The most distinctive of these are muscular twitchings; and contraction of the pupils is no less constant. The muscular weakness now rapidly increases; and, as a result of its involving the respiratory muscles, the breathing is slow, panting, and laborious. The appetite is entirely lost, and the tongue and the palate become dry. There is

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increasing drowsiness, with short fitful snatches of sleep, and a little rambling delirium. Convulsions and coma rarely set in, the intellect being commonly preserved to the last, so that the patient has in more than one instance spoken sensibly shortly before his death. Diarrhœa is of quite exceptional occurrence; and so is severe vomiting. The skin is moist, and often sweats profusely; there is never any ammoniacal or urinous odour from the surface of the skin or with the breath; nor does the body give off such odours after death. In one instance slight general anasarca was observed when the suppression first took place, but it passed off entirely on the third day.

The duration of life is stated by Roberts to be, as a rule, from nine to eleven days, and he remarks that the passing of a few ounces, or even of two or three pints, of a dilute urine does not seem to prolong it by more than a few hours. He knows of only three instances in which the patient survived beyond the eleventh day. In one of those cases, that of a man aged sixty-four, recorded by Rayer, death did not occur until the lapse of twenty-five days; another, that of a man aged seventy-three, recorded by Sir James Paget ('Clin. Soc. Trans.,' vol. ii), did not prove fatal for twenty-one days; the third, observed by Roberts himself, in a woman aged fifty-six, ended in death on the fifteenth day. The age of the patient does not appear to have any influence in accelerating or retarding the progress of the affection. Recovery has been known to occur in two or three cases in which there had been nearly complete suppression of urine for nine or ten days; in one of them the pupils had become contracted, and there was some mental confusion, but muscular twitchings had not made their appearance.

It is to Sir Wm. Roberts that we are indebted for the first complete account of the symptoms and causes of "obstructive suppression" of urine. But such cases had, of course, been observed before, although their characters had not been distinguished from those without obstruction. The case recorded by Sir Henry Halford, and cited in 'Watson's Lectures,' must have belonged to this category, although it was much more rapid in its course, having apparently proved fatal in about three days. Sir Thomas Watson remarked that patients affected with suppression of urine are chiefly persons who are advanced in life and inclined to corpulency.

An instance of obstructive suppuration of urine occurred at Guy's Hospital in the year 1876. A man aged forty-six received a blow on the left side of the abdomen, which was followed by hæmaturia; two days later the urineb ecame entirely suppressed and remained so until he died, seven days after the injury. In the course of the last twenty-four hours the muscles of his face were noticed to twitch, a profuse sweat broke out, and he became unconscious. At the autopsy one unusual feature was observed, namely, suppurative nephritis; and probably this accounts for its having reached a fatal termination more rapidly than usual. But the cause of the suppression of urine was found to be exactly what is stated above, viz. obstruction of the ureter of one kidney by a calculus, when the other one is incapable of secreting urine, owing to some antecedent lesion.

It is conceivable that both ureters might simultaneously be plugged with calculi; but the only other condition that can interfere with the flow of urine through both ureters at once (the two kidneys being healthy) and so cause obstructive suppression of urine, is obliteration of their channels by pressure from without, as by cancer of the uterus or by some other disease of the pelvic organs, such as we shall find to produce hydro-

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nephrosis. Roberts relates a few cases of this kind which ended fatally, and one, in which, after no urine had been secreted for seven days, it flowed again naturally during the remaining four weeks of the patient's life. But in most cases due to external compression, the renal cortex becomes atrophied or destroyed by hydronephrosis or consecutive Bright's disease before complete obstruction of the ureters occurs, so that the symptoms and course of obstructive suppression are seldom typical.

In the usual form of obstructive suppression dependent upon blocking of the ureter of the only functionally active kidney possessed by the patient, it is to be noted that the renal pelvis does not become much dilated, and that the quantity of urine accumulated by it is by no means large. The substance of the kidney in one of Roberts's cases was found to be much congested, but in another it was rather anæmic-looking, though dotted on the surface with numerous blood-spots. No pathologist seems to have noticed in the human subject œdema of the kidney, and a deeply ecchymosed state of its pelvis, which Cohnheim described as the usual consequences of ligature of the ureter in animals. The kidney, however, is generally of about twice the normal size, having undergone hypertrophy as the result of the overwork thrown upon it by the obsolescence of the opposite kidney during the months or years that may have passed since the latter became unable to share in the excretion of the urine.

As already remarked, whatever urine is formed by a kidney of which the ureter has been blocked, is pale, of low specific gravity, and contains but a small percentage of urea. This is, perhaps, contrary to what one might have imagined to be the probable effect of such an occurrence, but it accords perfectly with the results of the experiments of Hermann upon animals. He showed that in dogs the secretion of urine appears to cease entirely under a pressure of 2·4 inches of mercury, and that when the pressure is removed the result is that a large quantity of watery urine is poured out, in which very little urea is present. Bartels relates the case of a young man who had suffered from previous attacks of renal colic, and who in one such attack had suppression of urine for five days. When this passed off, he voided in twenty-four hours more than 3000 c.c., having a specific gravity of 1001, and containing numerous hyaline casts as well as albumen. Most observers seem now to think that the cessation of the activity of the kidney as soon as the pressure in the ureter and renal pelvis reaches a certain point is more apparent than real, the urine being really secreted, but reabsorbed as fast as it is formed. We can see what an important bearing this view has upon the theory of uræmia, when taken in connection with the absence of the usual uræmic symptoms in obstructive suppression.

In the *treatment* of suppression of urine, when it appears to be due to impaction of a calculus in one ureter, recourse should be had to those measures which we have seen to be sometimes effectual in aiding its expulsion downwards into the bladder, or its return upwards into the pelvis of the kidney, when there is renal colic. The abdomen in the course of the ureter may be well rubbed and kneaded, while the patient is in various positions,—standing, or lying, or inverted with his head downwards. But it is to be feared that the absence of pain in such cases means that the peristaltic movements of the ureter itself have ceased; so that there is little chance of success from such means. In two of Roberts's cases it is expressly noted that soon after the secretion of urine ceased, the pain of which the patient had been complaining disappeared entirely. Consequently it does not appear

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hopeful to employ hot baths, or chloroform inhalations, or anodynes of any kind, for the purpose of relaxing spasm.

There is, however, one method of treatment which seems never yet to have been attempted, but which deserves a trial. It is that of cutting down upon the kidney in the loin, and incising the ureter in the renal pelvis, so as to allow whatever fluid may be collected there to escape. The removal of pressure would probably at once be followed by an abundant secretion, and it is possible that a permanent fistulous opening in the loin might be created. Such an operation may not indeed be justifiable during the first few days after the suppression of urine has set in, on account of the possibility of spontaneous recovery; but there certainly can be no objection to it when at the end of a week muscular twitchings begin to appear. It is also a question whether it may not be practicable for the surgeon to remove an impacted calculus from the ureter. In the case that occurred in 1876 (see p. 525) the stone was found after death impacted four inches from the kidney; but in two cases recorded by Roberts it lay just within the vesical orifice of the ureter. Might it not be felt in such a position *per rectum*, and possibly be set free by an incision?

A remarkable case of obstructive suppression of urine occurred at Guy's Hospital in 1885, under the care of Mr R. C. Lucas. The patient, a woman of thirty-seven, was first admitted in June, 1885, with a history of frequent hæmaturia, and pain and swelling on the right side of the abdomen. Mr Lucas removed the right kidney, which proved to be a mere shell filled with calculi, with excellent results. The following October the patient was seized with violent left nephralgia and suppression of urine, followed by vomiting, muscular weakness, and at last somnolence, with feeble pulse and temperature of 99°. On the fifth day of suppression Mr Lucas cut down on the left kidney and removed a large impacted calculus; the patient recovered, and was well five years afterwards ('Med.-Chir. Proc.,' Jan. 13, 1891).

5. *Unilateral atrophy of the kidney.*—It is not at all uncommon in the *post-mortem* room to find one kidney shrunken to a mere thin flat relic, scarcely if at all bigger than the adjacent adrenal body, and weighing about an ounce or an ounce and a half. Twenty cases of this kind were collected by the author from our records at Guy's Hospital; and this number has since considerably increased. In several instances the cause of death was some disease entirely unconnected with the urinary organs. The secretion of urine, in fact, takes place quite naturally under such circumstances, because the opposite kidney undergoes a compensatory enlargement, becoming as heavy as the two organs together normally should be.

The nature of the process by which this enlargement is effected has been studied by different observers with discrepant results. Perl ('Virchow's Arch.,' vol. lvi) found increase in size of the convoluted tubes and epithelium, but not of the glomeruli. Beumer (*ibid.*, vol. lxxii) could find no demonstrable increase in size, whether in the glomeruli or in any of the tubes, in a case in which one kidney was congenitally absent, so that, according to the strict terminology of Virchow, the compensatory change would be a *hyperplasia* rather than a *hypertrophy*, *i. e.* a multiplication rather than an overgrowth of the individual parts. There can be little doubt that the latter view is correct. There is more renal tissue, but the renal tissue is not composed of gigantic elements.

Such a condition is so far dangerous that everything depends on the hyper-

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trophied organ. If its ureter should from any cause become obstructed, the result is suppression of urine instead of a mere attack of renal colic. Again, laceration of the region by violence is very likely to be followed by fatal results, as happened some years ago in the case of a boy admitted into the accident ward of Guy's Hospital, although during life it appeared a mystery why a unilateral injury should have such a serious effect. It may also be that the tissue of a kidney enlarged by compensating hypertrophy is particularly liable to Bright's disease; at any rate, in about one fourth of the above twenty cases there was chronic nephritis of the hypertrophied kidney. Perhaps the compensation is not always perfect, and so the enlarged organ cannot for an indefinite length of time do the entire work of secreting urine without danger. The period of life at which the atrophy occurs might naturally be supposed to make a difference in the completeness of the compensating hypertrophy. Among forty-eight instances of congenital absence of one kidney, collected by Beumer from different sources, there were no fewer than twenty in which the opposite kidney was found diseased. It most often was the seat of "chronic inflammation," but in many instances it contained calculi in the renal pelvis.

Acquired atrophy of a kidney is due to various causes. In three of our twenty cases a calculus was found impacted in the corresponding ureter, and in two other cases calculi were present in the renal pelvis. In none of the remaining cases was any concretion found, nor was there any obstruction to the outflow of urine from the renal pelvis. Yet the pelvis and the calyces were dilated in no fewer than nine of them, and in two the ureter was dilated and thickened all the way down to the bladder. It seems difficult to avoid the inference that there had at some former period of the patient's life been a renal calculus, which either escaped through the natural passages or underwent disintegration, but which had deranged the kidney sufficiently to cause it to waste. This conclusion is greatly strengthened by the fact that in two other cases there was a history of lithotomy many years previously; in one of them the operation had been performed by Sir Astley Cooper when the patient was aged thirteen, forty-five years before his death. Lastly, in one instance in which neither the renal pelvis nor the ureter was enlarged, the vesical orifice of that tube was considerably lower than that on the opposite side, and lay nearer to the prostate, as though it had been forced downwards in the expulsion of a concretion.

5*a*. A very rare effect of the presence of a calculus in the renal pelvis is the replacement of the substance of the kidney by a mass of adipose tissue having the shape of the healthy organ, and of about the same size, as in a case described and figured by Dr. Rickards, of Birmingham, in the 'Brit. Med. Journ.' for July 7th, 1883.

6. *Hydronephrosis.*—We have seen that plugging of a ureter by a calculus, or partial obstruction of both ureters as the result of morbid processes of various kinds, does not necessarily lead to any considerable accumulation of fluid in those tubes or in the renal pelvis. There are, however, cases in which such an accumulation occurs, and for these the term *hydronephrosis*, originally suggested by Rayer, appears to be the most suitable.

Anatomy.—The earliest indication that the organ has been subjected to pressure from within is shrinking of the mammillary apices of the pyramids, which, instead of nearly filling the calyces, become separated from each other by broad intervals, and ultimately flattened, or even hollowed out. With

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the microscope it may be seen that the tubes in the remains of the pyramids are bent into a regular series of wave-like curves. As this change in the pyramids goes on, the calyces and the renal pelvis undergo dilatation. Sometimes the calyces stretch out of the hilus of the organ, so that the pelvis forms a sac situated nearer to the middle line of the body than the kidney itself, into which it sends finger-like processes; in a case that occurred at Guy's Hospital in 1876 such a sac lying beyond the kidney was found to hold a pint of fluid. Much more frequently, as the calyces and pelvis yield before the pressure of their contents, each calyx forms a somewhat egg-shaped cavity, communicating with the pelvis by a smooth orifice, and separated from the adjacent calyces by a tough fibrous membrane; the surface of the organ acquires a lobulated appearance, the lobules corresponding in number with these cavities; or, if the sac is very large, the septa between them may become perforated, so that they may at last break down and form a huge single cavity.

In the meantime the secreting substance of the organ passes into the condition described as consecutive Bright's disease (p. 499), or undergoes atrophy, until at length no trace can be discovered, or at most only a few scattered relics here and there in the walls of the sac. The ureter, too, may be dilated until it is as large as the finger of a glove, or a coil of small intestine; so that it has actually been supposed during life to be an abdominal tumour.

Secretion.—The nature of the fluid contained in the sac of hydronephrosis varies in different cases. When the enlargement is but slight, as in most instances in which both kidneys are affected, it is still more or less dilute urine, which, however, may contain albumen, or be mixed with pus or blood. In those extreme cases which are generally unilateral the fluid is sometimes pale and clear, sometimes stained with blood. It is usually of lower specific gravity than normal urine, being in this respect like the fluid secreted in cases of "obstructive suppression." But in a remarkable case operated on by Czerny it must have had the same characters as healthy urine, for that passed by the patient was in all respects natural; yet extirpation of the hydronephrotic organ was followed by complete and fatal anuria, and on *post-mortem* examination it turned out that the opposite kidney had undergone atrophy, and that its ureter was obliterated.

The solid matters dissolved in this fluid are generally urea, uric acid, and salts of the same composition as those that are found in urine. But in a case that came under the author's observation in 1876 neither urea nor uric acid could be detected in the fluid removed by tapping from a tumour believed to be hydronephrotic; and Sir Spencer Wells and Mr Cooper Rose ('Lancet,' 1868) have also met with instances in which urea has been absent. Mr Henry Morris ('Med.-Chir. Trans.,' 1876) cites cases in which the contents of hydronephrotic sacs in the fœtus have been devoid of urea. Albumen is commonly present in greater or less quantity. In some cases the fluid has been purulent, as in one described by the writer ('Path. Soc. Trans.,' xxiii) in which six and a half pints of an opaque reddish fluid were drawn off by a trocar; the disease may then be called pyonephrosis, if a special name is needed. Dr Dickinson ('Path. Soc. Trans.,' xiii) has recorded a case in which a very large sac contained a gelatinous or colloid substance.

Ætiology.—The causes of hydronephrosis often affect *both kidneys* simultaneously. Among these may be enumerated stricture of the urethra, enlargement of the prostate, and various vesical affections (including villous disease of the bladder); also pregnancy, prolapsus, or retroflexion of the

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uterus, and pelvic tumours, especially cancer of the womb, involving the surrounding tissues of the iliac glands.

Cohnheim has recorded a remarkable case in a rachitic boy of eleven with contracted pelvis in whom double hydronephrosis was produced by the pressure of an enormously dilated rectum and sigmoid flexure. In such cases the renal affection is usually more marked on one side than the other. But there is almost always so much interference with the secreting action of the two kidneys that death occurs from such interference (if not from the primary disease) before the sac has become large enough to constitute an abdominal tumour capable of recognition during life. Mr Morris, however, relates a case of villous disease of the bladder in which a rounded swelling, of the size of the head of a small fœtus, was felt in the right loin. As a rule, the only clinical evidence of the renal affection is a pale watery condition of the urine, until perhaps convulsions or other uræmic symptoms set in, and rapidly bring about a fatal termination. For example, in 1871, a woman aged thirty-six was lying in the uterine ward of Guy's Hospital with cancer, when she began to complain of severe headache; after two days she screamed out violently in the night, and became unconscious; and in this state she remained until her death three days later. The autopsy showed that the cause of her symptoms was not cerebral hæmorrhage (as had been suspected), but uræmia: each kidney had its pelvis greatly dilated, its pyramids flattened, and its cortex pale, though not decidedly narrowed. In 1869 a woman aged thirty-eight was admitted into our clinical ward shivering violently and very cold, with a dry brown tongue and other typhoid symptoms, but with her mind clear. She was said to have had prolapse of the uterus for a year, and her urine was found to contain pus. She died two days later, her temperature having been very low throughout. On *post-mortem* examination it was found that the womb had dragged down the vesical extremities of the ureters, and compressed them against the pubic arch. There was hydronephrosis on both sides, and the cortex of each kidney was greatly atrophied.

If the cause of the hydronephrosis is so situated as to affect the ureter leading from *one kidney* only, it may produce a tumour of very large size. The opposite kidney then undergoes hypertrophy, and, as it may carry on the secretion of urine perfectly, there is nothing to prevent the development of the hydronephrosis to any conceivable extent.

Of the lesions that may affect one ureter so as to cause unilateral hydronephrosis, the most obvious is obstruction by a calculus. Thus in 1877 a man aged forty-six died in Guy's Hospital of dropsy due to Bright's disease affecting an hypertrophied left kidney; in the right ureter there was impacted a mulberry concretion an inch and three quarters in circumference; the right kidney was converted into a shining loculated cyst with a smooth lining, upon which there was one little patch of renal substance about as large as a shilling still remaining. It is of course impossible that, after both ureters have been completely blocked by stones, the patient should live long enough to admit of the development of double hydronephrosis. But in 1874 a boy aged six was in the hospital for stone in the bladder, when he died of tonsillitis. Each ureter was greatly dilated, and also the pelvis of each kidney. The right ureter was blocked by a second small calculus about an inch above its orifice; the left was free, so that the distension on that side had to be attributed either to interference with the downward flow of urine resulting from the vesical calculus, or else to the

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passage of that calculus at a time when the right ureter was free, or at least not entirely obstructed. In 1857 there died in Guy's Hospital a woman aged fifty-six, who had a large fluctuating swelling in the left loin, and a smaller one in the right loin. Hydronephrosis was found to be present on both sides, and the pelvis of each kidney contained calculi, but it was only on the left side that impaction of a calculus in the ureter had taken place. This patient had been passing blood and pus in her urine all the while she was in the ward; but in most cases of hydronephrosis due to impaction of a calculus the urine is perfectly normal, though a history of former attacks of renal colic may perhaps be elicited, sometimes very far back. Rayer recorded a case of hydronephrosis in a man aged sixty-four, who had for a long series of years enjoyed perfect health, but who at the age of twenty-two had suffered from pain in the right kidney and along the ureter, attended with hæmaturia.

Another cause of unilateral hydronephrosis is compression of the ureter, generally near the brim of the pelvis, by a thickened peritoneal band, the result of inflammation of the serous membrane. Sometimes again the ureter is thickened and narrowed by changes in its own coats, the origin of which is no longer discoverable when the case comes to an autopsy, probably many years after their occurrence. In a case of the writer's ('Path. Trans.,' xxiii), the ureter was found obliterated about an inch and a half below the pelvis of the kidney, and this appeared to be clearly traceable to a kick from a horse about two years previously; the injury had been followed at the time by hæmaturia. A similar instance in which hydronephrosis in a boy of twelve was directly traceable to a fall, has been recorded by Mr Croft ('Trans. Clin. Soc.,' xiv). In 1873 an autopsy in the case of a boy aged four, who died with a calculus in his bladder in Guy's Hospital, showed the ureter as thick as a lead pencil, and completely occluded by an oblique cicatrix about an inch from its origin.

There are, however, many instances in which no cause for the hydronephrosis can be made out, the ureter appearing perfectly free from obstruction in its whole course from the renal pelvis to the bladder. In most such instances there was probably at some former period a calculus, which has in the meantime undergone disintegration, or has been voided.

With regard to this question, the facts adduced at p. 528, in reference to the origin of atrophy of the kidney, are of much importance. It is possible that some conditions generally regarded as occasional causes of hydronephrosis are not really so, but that here also the true cause is a calculus. One of these hypothetical causes is compression of the ureter by a supernumerary renal artery. Another is obliquity of the origin of the ureter from the renal pelvis, causing a valve-like impediment. That such an appearance is not infrequently met with is certain, and Dr Hare recorded ('Med. Times and Gaz.,' 1857) a case of hydronephrosis in which the ureter on each side was coiled on itself—like a turn and a half of a cork-screw brought closely together—and adherent to the lower part of the sac. There can be no doubt that a similar condition of the ureter is the cause of the "intermitting" character of many renal tumours, as well as of the fact that after tapping the cyst the ureter sometimes becomes for a time pervious. What seems doubtful is whether cases of this kind are congenital, or whether the twisting of the tube is not a secondary result of its distension, just as one finds the duct of the gall-bladder distorted to an even greater extent and bound down by adhesions, as the result of the passage of gall-

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stones. The author twice saw such a valvular condition of the upper orifice of the ureter when there was obstruction of the lower urinary passages: once in the case of an old man who died of the effects of stricture of the urethra, and in whom, although the ureter was not dilated, the pelvis of the left kidney formed a large pouch full of dark brown fœtid fluids; and again in a fatal case of lithotomy, complicated with stricture. Dr Sainsbury has described a case of hydronephrosis from two valvular folds of mucous membrane at the origin of the ureter ('Path. Soc. Trans.,' 1886, p. 296). The writer has observed similar folds more or less markedly developed, but not sufficiently to make certain that they would have formed a mechanical obstruction during life.

A point upon which Cohnheim lays stress is the origin of the ureter from the side of the renal pelvis instead of from its lower end. The result of this, he says, is that so long as the patient is in an upright position the bladder receives only so much urine as overflows from the pelvis of the kidney; and he mentions the case of a woman so affected who for a long time passed scarcely any urine during the day, whereas she voided large quantities at night. This, however, surely proves too much, unless, indeed, the hydronephrosis was large enough to hold several hours' urine; and it must not be forgotten that in renal cirrhosis (including the consecutive form of Bright's disease) the nocturnal flow of urine is often excessive. Cohnheim himself observes that before such cases come to an autopsy the conditions are so altered by the dilatation of the pelvis of the kidney as to render it impossible to say how the affection began. That hydronephrosis itself is sometimes congenital is well known; some cases in which the abdomen has been large from the time of birth have been prolonged for years, although they far more often terminate fatally within the first few days or weeks. But congenital hydronephrosis is traceable to some definite malformation, such as occlusion of a ureter, or more rarely of the urethra. The cases in question do not lend support to the view that obliquity or twisting of the upper end of the ureter, occurring as a malformation, can give rise to hydronephrosis.

One point worthy of notice, with respect to the origin of the congenital form of hydronephrosis, is that it is often associated with harelip, imperforate anus, club-foot, and other defects of development. The fact that closure of the outlets of the kidneys causes during intra-uterine life an accumulation of fluid seems to show that their secreting function must already be active; and in a paper read in 1876 ('Med.-Chir. Trans.,' vol. lix, p. 98) Mr Henry Morris has argued for the view that the fœtal kidneys normally pour urine into the *liquor amnii*, whence it is absorbed into the blood of the mother, to be afterwards again excreted by her urinary organs.

In a case that occurred at Guy's Hospital in 1868 Dr Moxon suggested a new cause for hydronephrosis. The left kidney was found by him after death to have the pyramids flattened, the pelvis and the calyces dilated. The patient was a man aged twenty-two, who suffered from a lumbar abscess, and lay constantly on his left side with his pelvis raised upon an air-pillow, so that the tendency of fluid to gravitate within the ureter must have been from the bladder to the kidney, and not in the reverse direction.

Diagnosis.—Many cases are on record in which *single hydronephrosis* has been mistaken for a large *ovarian cyst*, or even for *ascites*. The most remarkable of them all is perhaps one related by Mr Glass in the 'Philosophical Transactions' for 1747. The patient was a woman aged twenty-three at

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the time of her death, who had been dropsical from birth; the abdomen then measured 6 feet 1 inch in circumference, and the sac contained thirty gallons of fluid. In several other instances many pints have been taken from a hydronephrotic tumour during life, or have been found in it on *post-mortem* examination. Among the points which should distinguish such a tumour from an ovarian cyst are its having first made its appearance in the loin rather than near the pelvic brim, its having no pelvic connections, the presence of the colon in front of it, and the absence of resonant intestine in the loin. It has, in fact, all the characters of a renal tumour. Fluctuation is generally well marked, and the outline of the swelling is sometimes lobulated. It may occupy a large part or the whole of one side of the abdomen, extending across beyond the umbilicus, and downwards into the iliac fossa.

In a patient under the author's care in 1883 there was a prominence in the epigastric and in the left hypochondriac region, while in the loin the bulging was but slight; and as there was obvious pulsation, with an audible bruit, the case looked like one of abdominal aneurysm. In that instance the history given by the patient himself contained one point which, if duly attended to, would have cleared up the diagnosis. He said that on more than one occasion after the first appearance of the swelling it had undergone a great diminution in size; he had not, however, noticed that at those times there was any increased flow of urine, nor that the urine was altered in appearance. This spontaneous subsidence or disappearance of the tumour when it is observed is by far the most important clinical character of hydronephrosis. If associated with an excessive discharge of fluid from the bladder it may be said to be pathognomonic. Even without that corroborative evidence the only cases in which a similar occurrence is likely to be met with are those in which an ovarian cyst ruptures into the uterus or into the intestine; and such events are probably always indicated by the escape of fluid through the vagina in the one case, or the entrance of air into the cyst in the other.

Two other affections may be mistaken for single hydronephrosis, namely, *hydatid* of the kidney and a large single *renal cyst*. Each of them is very rare, at least as giving rise to a palpable swelling. The former is almost certain to be set down to hydronephrosis, unless its nature is revealed by the escape of daughter-cysts through the urethra, or by the characters of the fluid removed by paracentesis. The latter could probably be distinguished only after extirpation or on *post-mortem* examination. Two striking instances are recorded by Mr Cæsar Hawkins ('Med.-Chir. Trans.,' xviii) and by Dr Hare ('Path. Soc. Trans.,' iv); in each of them the tumour filled the right side of the abdomen. Three or four other cases are cited by Czerny in his list of cases of nephrectomy ('Trans. Internat. Congress,' 1881). The writer has lately met with what was probably a case.

The patient was an Irish county-magistrate accustomed to long journeys and much exposure, a strong, healthy man of forty-five. In June, 1890, he discovered a large lump below the liver, and more or less moveable. On examination in the following November, a round, smooth, tense, and elastic tumour was found behind the hepatic flexure of the colon, and apparently in contact with both liver and kidney. The diagnosis was a hydatic cyst, but on aspiration in February, 1891, the contents were yellowish and albuminous, and coagulated into a gelatinous mass. The urine was healthy, but in the spring of 1890 he had an attack of hæmaturia.

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Neither pain nor tenderness is necessarily present in hydronephrosis, though when the swelling is large it often causes a distressing sensation of fulness or distension. In some cases pricking or shooting pains are complained of, which are perhaps due to local inflammatory changes in the peritoneum covering the sac. The colon is sometimes tightly stretched over the tumour, in such a way as to interfere with the free passage of its contents; thus in a case recorded by Roberts the chief symptoms were at first those of intestinal obstruction, which recurred again and again during several years.

Double hydronephrosis can only be surmised when the conditions which produce it are present, along with symptoms like those of advanced Bright's disease. Two lumbar tumours are very rarely felt, except in cases of hypertrophic cystic degeneration of the kidneys (p. 505).

Prognosis.—When hydronephrosis is *bilateral*, the patient is always in danger, since the structure as well as the functions of the secreting tissue of the two kidneys is inevitably interfered with; and in many cases the primary disease that has caused the obstruction to the escape of urine would in itself prove rapidly fatal, without such complication.

On the other hand, the course of *unilateral* hydronephrosis is commonly very chronic, and it scarcely ever brings life to an end by itself. In the case recorded by Mr Glass, death was apparently due to pressure on the diaphragm and displacement of the thoracic viscera. In one observed by Mr Thompson, of Nottingham ('Path. Trans.,' xiii), it resulted from peritonitis set up by escape of the contents of the sac through an ulcerated aperture. In the writer's case of traumatic stricture of the ureter (p. 531) there had been communication with some part of the intestine, for the sac had suppurated and contained a mass of vegetable fibre, with bits of apple-core and part of a clove. In other fatal cases that have been recorded the cause of death has generally been either an independent disease (as, for example, acute tuberculosis in a case of Dr Hillier's) or else the supervention of some morbid process in the hypertrophied kidney on the opposite side of the body. Consequently, it is not advisable to interfere actively with hydronephrosis until the patient is unable to bear the pain and discomfort to which its presence may give rise.

Treatment.—In some few instances one can succeed in emptying the sac by rubbing the abdomen. Roberts relates the case of a girl of eight, who came under his care with a soft fluctuating tumour in the left side, of about the size of a child's head. This was diligently manipulated in every direction, with the aid of a lubricating ointment, on alternate mornings. After the third time she suddenly passed abundant urine, the tumour forthwith subsided, and did not reappear while she remained under observation. A somewhat similar result was attained in a case recorded by Dr Broadbent ('Path. Soc. Trans.,' xvi) of double congenital hydronephrosis in an infant.

But when the sac is tense, little can be hoped for from such a procedure; and there is often so much tenderness that it cannot be adopted. The only treatment then is to puncture the sac with a trocar. On the left side this may be done at a spot just anterior to the last intercostal space. But on the right side Mr Morris has shown ('Med.-Chir. Trans.,' lix) that there is danger of wounding the liver, and he advises that a point should be selected halfway between the last rib and the crest of the ilium, and from two inches to two and a half inches behind the anterior superior spine.

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After the operation, fluid like that which has been withdrawn from the tumour sometimes passes for a time with the urine, showing that the ureter has again become pervious. But the sac almost always rapidly fills again, and may soon regain the same size as before. Thus, in a case of double hydronephrosis, which was three times tapped by Fränkel, the patient did not micturate at all during from twelve to forty-eight hours after each tapping, the whole of the fluid secreted in the interval having doubtless accumulated in the two sacs. It is true that unilateral hydronephrosis is commonly attended with such extreme destruction of the renal cortex that the organ can hardly be supposed still capable of forming urine. But experience seems to show that even in such cases fluid continues to be poured out into the sac by a process of transudation like that which occurs in extreme cystic degeneration (p. 503). One of the few cases in which repeated puncture has led to permanent shrinking is that of Mr Croft already referred to ('Trans. Clin. Soc.,' xiv). In that instance, within fifty-four days of the accident which caused the disease, seventy-nine ounces of fluid had already collected. Paracentesis was performed eight times altogether, from three to four pints being removed each time. After the eighth operation, which was performed at three months' interval from the first, no further accumulation took place. In a case observed by Sir Spencer Wells ('Dubl. Quart. Journ.,' 1867) the patient, two months after a second tapping, passed two calculi *per urethram*, after which the tumour completely disappeared and did not return. On the other hand, there does not appear to be much fear of setting up suppuration in the sac by paracentesis, though this result has been known to follow the attempt to cure the disease by making a fistulous opening ('Path. Soc. Trans.,' xiii, Dr Little's case). Czerny mentions ('Trans. Internat. Congress, 1881') the case of a man in whom Gustav Simon had two years previously made such an opening, and who in 1881 was still acting as an attendant in the hospital wards at Heidelberg.

When tapping proves ineffectual, the bolder operation of removing the distended kidney has of late years been frequently performed.

Czerny, in his statistics of nephrectomy (*loc. cit.*, p. 249), gives twelve cases in which that operation has been performed for hydronephrosis or for cyst of the kidney. Seven of them ended fatally, but this high mortality may perhaps be in part attributed to the fact that in five an erroneous diagnosis of ovarian tumour had been made. According to Mr Barker a lumbar incision is in cases of this kind preferable to one in the front of the abdomen. Czerny's case above referred to (p. 529), shows the importance of ascertaining that the opposite kidney retains its functional integrity. Probably this may be best done by making a preliminary opening into the sac and allowing it to drain, so that after one can be certain that no fluid from it any longer descends the ureter, one can measure and test the urine passing into the bladder from the other organ.

7. *Pyuria and pyelitis.*—The presence of a stone in the kidney causes pus to appear in the urine by giving rise to suppuration of the renal pelvis. But, as in the case of hæmaturia, it will be well to consider *pyuria* generally, and briefly to indicate the various affections of the urinary organs that may lead to this symptom.

The presence of pus in urine commonly gives it a turbid, opaque appearance, and on standing there is precipitated a dense whitish-yellow sediment which somewhat resembles the white gravel formed by amorphous phos-

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phate of lime, but may be readily distinguished by the microscope. It remains undissolved after the addition of an acid, and when a caustic alkali is added it forms a transparent gelatinous mass, which hangs in long strings when poured from one vessel to another. In urine which has undergone the ammoniacal fermentation pus has this character; it forms a viscid, tenacious substance, which glides out as a coherent mass when the vessel is emptied. This tenacious alkaline pus often causes much pain and distress in passing through the urethra.*

It is an interesting fact that leucocytes in urine, even when it is alkaline and full of bacteria, sometimes retain their amœboid movements.

Whenever there is pus in urine, there is also albumen, derived from the liquor puris. But if the quantity of pus is small, the albumen may not be discoverable by ordinary tests. It is often an important practical question to determine whether the amount of albumen observed in purulent urine is or is not greater than the pus itself accounts for; since, if it is greater, it affords evidence of the existence of Bright's disease in addition to the affection causing the pyuria. In various surgical affections of the urinary organs the propriety of operative interference depends largely upon this point.† If heat gives a decided precipitate after the pus has been allowed to subside, it is likely that the albumen is due to some additional cause. Tube-casts should also be carefully looked for, as a sign of renal disease.

Apart from calculus, pyuria may be due to a great variety of affections. The possible presence of *gonorrhœa* must never be forgotten; nor, in females, that of *leucorrhœa*, which, however, is indicated by a large number of squamous epithelial cells, as well as of leucocytes, under the microscope. Again, *cystitis* is a frequent cause, and one should remember that there may sometimes be pus in the urine from this cause without the patient complaining of much pain or having to micturate very frequently, especially if he has a stricture or an enlarged prostate.

Whatever may be the origin of cystitis, it is apt sooner or later to lead to an extension of inflammation upwards to one or both of the renal pelves. Thus pyelitis is of frequent occurrence as part of the wide-spread change in the lining of the urinary organs that results from an ammoniacal decomposition of the urine within the body. It may also accompany various surgical diseases of the lower urinary passages.

Pus may appear in the urine as the result of nephritis from certain poisons, of which cantharides and turpentine are the chief. Slight forms of pyuria are seen in connection with Bright's disease, and also (it is said) as the result of diabetes, or during the course of enteric fever and of other specific diseases. Again, it sometimes follows a blow or other injury to the loin; and, as Kaltenbach has particularly stated ('Arch. f. Gynäk.,' iii), it may develop itself after parturition, as the result perhaps of extension of inflammation from the pelvic organs.

None of these causes, however, produce persistent and severe pyelitis independently of a like affection of other parts of the urinary tract of the

* This chemical test for pus is commonly associated with the name of Dr Babington, though Leube attributes it to Donné.

† Leube having added to urine 2 per cent. of pus, he found that in every microscopic field, prepared with fluid that had not been allowed to settle, there were from ten to fifteen leucocytes, and that the amount of albumen precipitated by boiling occupied about one tenth of the bulk of the urine. His conclusion is that a coagulum of even one twentieth or one twenty-fifth is more than can be attributed to pus, unless at least some few pus-corpuscles are visible in each microscopic field.

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mucous membrane. So that if we exclude tubercular cases (which will be described in the next chapter) we need perhaps admit no other cause than gravel or calculus for such forms of pyelitis as require special clinical recognition. It is true that one occasionally fails to discover any concretions in cases which have ended fatally after having been of long standing, or in which a surgical operation affords an opportunity of thoroughly exploring the diseased organ. But we have already found grounds for the belief that either unilateral atrophy of the kidney or hydronephrosis may result from calculi which subsequently disappear (pp. 528 and 531); and the same may also be true of pyelitis.

The symptoms that characterise "calculous pyelitis" are more or less severe *pain* in the loin or in the abdomen, *hæmaturia* which generally recurs from time to time, and more or less constant *pyuria*. A case in point was under the author's observation for several years. The patient felt, in 1876, a slight pain or uneasy sensation in the left loin, for which no cause could be found. A short time afterwards he noticed some blood in his urine; but, on taking medicines which rendered it alkaline, the hæmaturia ceased. Yet from that time the urine almost constantly contained pus in small quantity, with apparently an excess of albumen; and crystals of oxalate of lime were usually to be detected, sometimes crystals of lithic acid. In 1880 he passed a small oxalate calculus, after which he was more free from pain than he had been for some years previously. All along the general health was good, and the patient was able to discharge responsible duties.

It very rarely happens that the disease assumes so mild a form as this. Generally speaking rigors recur from time to time, sometimes with regular quotidian periodicity. There is often considerable pyrexia, which may assume a hectic type. Diarrhœa may be persistent and intractable; or there may be obstinate constipation from adhesion of the colon to the anterior surface of the affected kidney. When pyelitis runs on for a length of time the renal pelvis often becomes dilated into a large sac, which may be felt as an abdominal tumour, and may bulge into the loin as an elastic fluctuating mass, very painful and tender to the touch. If the ureter becomes from time to time blocked, this swelling may present great variations in size on different occasions, and there may be converse variations in the degree of pyuria, the urine being clear when the swelling is largest, whereas a subsidence of the tumour is accompanied by the escape of several ounces of pus into the bladder. In such cases of "pyonephrosis" the renal cortex probably always undergoes atrophy, or becomes shrunken by a process of consecutive Bright's disease. If there are calculi in both kidneys, as is often the case, this morbid process is of itself sufficient to destroy life, with symptoms of uræmia. And even when the affection is limited to one side, the opposite kidney may, after undergoing hypertrophy, become affected with Bright's disease, either as the result of lardaceous changes in it, or independently of any such changes; but in other cases, after lasting a certain length of time, the inflammation subsides, and the kidney shrinks and dries up into a putty-like mass.

Again, many cases of pyelitis end fatally by the supervention of *perinephritic abscess*. Inflammation probably never affects the renal pelvis for any considerable length of time, nor with any great degree of severity, without extending to the surrounding structures, which become indurated and matted together by new fibroid material; but in many instances, after a while, the mucous membrane undergoes ulceration, and perforation with escape of urine and of pus takes place into the connective tissue. When

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this occurs, there is usually a marked increase in the pyrexia and in the other general symptoms that have previously resulted from the pyelitis. A fluctuating swelling may appear in the loins, with extreme local tenderness; and ultimately the skin may become reddened, and the abscess, if not opened by the surgeon, may point and break of its own accord. In other cases the course taken by the suppuration is different: it may enter the sheath of the psoas muscle and make its way downwards into the groin, and even penetrate the hip-joint. A point on which Trousseau laid stress is that when the psoas is affected the thigh is kept more or less rigidly flexed upon the pelvis; this is true, but the psoas abscess may be due to vertebral caries or other cause than renal suppuration. Sometimes the pus may extend in front of the iliacus muscle, and point above Poupart's ligament; if it rupture into the intestine, gas and fæcal matter often escape into the abscess cavity; subcutaneous emphysema may develop itself in the back, as was twice observed by Trousseau. Lastly, a perinephritic abscess may burrow through the diaphragm and the lung, and discharge itself by the bronchial tubes.

Conversely, an abscess starting from a vertebra, a lymph-gland, or some other abdominal viscus may make its way into the urinary passages.

When there is free discharge of pus in the loin the inflammation sometimes gradually subsides, and recovery ensues. But, as a rule, the prognosis of perinephritic abscess is unfavourable, the patient becoming worn out by the drain of pus, by severe pain and hectic fever, or by lardaceous disease.

8. *Suppurative nephritis.*—This consists in the presence of more or less numerous foci of inflammation in the cortex and medulla of the kidney, which appear upon the surface as minute round or irregular dots, and upon section of the organ as streaks or lines, traversing the cortex to a greater or less depth, or running continuously through its whole thickness, as well as through the corresponding medulla. At an advanced period there is well-formed creamy pus; when the disease is earlier fatal, there is often only a soft pinkish-white material, which consists of kidney-tissue infiltrated with leucocytes, but not yet completely destroyed. Surrounding the infiltrated or suppurating tracts there is much vascular injection. Sometimes only one or two points of even commencing suppuration are discoverable, so that they are not unlikely to be overlooked. There is reason to believe that the recognition of even a single point of acute inflammation in the renal cortex at an autopsy proves the existence of a morbid change sufficient to account for death.

The *causes* of suppurative nephritis vary. Sometimes it occurs as part of general pyæmia; and Dr Moxon noticed that in cases of pyæmia resulting from perinæal section or lithotomy, abscesses in the kidneys were more apt to occur than when pyæmia was due to lesions unconnected with the urinary organs. This observation is interesting because some pathologists have been disposed to refer suppurative nephritis in general, even when obviously traceable to an inflammatory process spreading upwards from the bladder along the ureters, to an infection with some septic poison from without. Thus Dr Goodhart, in vol. xix of the 'Guy's Hospital Reports,' endeavoured to trace a connection between this affection and the presence of erysipelas in other cases in the same ward and at the same time. Moreover, as a matter of fact, the remarkable decline in the frequency of pyæmia in our wards of recent years has been accompanied by a corresponding decline in the frequency of suppurative nephritis.

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In some cases suppuration of the kidney seems to occur as a primary morbid process. Dr Goodhart's paper recorded three instances, in each of which, although some degree of cystitis was found at the autopsy, it seemed doubtful whether this was sufficient to account for so severe an affection of the kidneys; one was a case of enteric fever, another of mitral disease, and the third of extensive burns.

But in the vast majority of instances suppurative nephritis is secondary to an affection of the urinary passages; either to some one of the common surgical diseases of the urethra or of the bladder, or else to paralysis of the bladder from some spinal lesion, or to compression of the ureter as the result of cancer or other disease of the uterus. As might be expected, most cases of this kind are also marked by the mechanical effects of obstruction to the outflow of urine from the renal pelvis, on one or both sides, which have been fully described already. The suppurative nephritis itself is not always bilateral. In some instances the whole length of the urinary tract is obviously affected with inflammation from the bladder to the mucous membrane covering the renal pyramids. But in others the lining of the ureter and of the renal pelvis is normal. Dr Dickinson is of opinion ('Med.-Chir. Trans.,' lvi) that the exciting cause of the nephritis is really the ammoniacal state of the urine resulting from its decomposition within the urinary passages, and holds that suppurative nephritis as a secondary affection never occurs except when the urine has undergone this change. How rapidly the disease may develop itself is well shown by a case which he narrates of an old woman admitted into the hospital for a fracture of the femur, who two days later became unable to pass her water, so that a catheter had to be used. The urine drawn off was then natural, but very soon afterwards the urine became offensive, and death occurred within a week of the accident, three days after the urine had changed its character. Both kidneys were found to be suppurating.

The *symptoms* of suppurative nephritis are obscure. Dr Dickinson describes rigors as an early and frequent symptom; febrile symptoms rapidly follow, with vomiting, great prostration, feeble pulse, dry, brown tongue, and sometimes profuse sweating or diarrhœa. Convulsions are rare, and the case ends in stupor rather than coma. But the disease is often latent, so that the patient may die quite unexpectedly, without febrile or cerebral symptoms.

The state of the urine throws but little light upon the *diagnosis* of suppurative nephritis. There may be a large quantity of pus present, but this is probably the result of the pyelitis and of the cystitis which are so generally present at the same time.

It might be thought that the determination of the amount of urea in the urine would throw light upon the state of the kidneys in these doubtful and obscure cases. This expectation, however, appears not to be verified by experience. Dr Goodhart ('Guy's Hosp. Rep.,' xix) records two instances in which he made quantitative analyses shortly before death, and found that the renal secretion contained thirteen or fourteen grains of urea to the ounce; a third patient passed in the twenty-four hours three pints of urine, with a total quantity of 592 grains of urea; and a fourth patient thirty ounces with 328 grains of urea. On the other hand, a man, who afterwards went out well, having had his bladder punctured *per rectum*, passed thirty ounces in the twenty-four hours with only 295 grains of urea, or less than ten grains per ounce.

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Treatment of renal calculus.—It is remarkable that the improved knowledge of the actual composition and chemical properties of renal and vesical calculi, which marked the first half of the present century, was not accompanied by improved chemical therapeutics. On the contrary, previous attempts to dissolve calculi were almost entirely given up. In the fourth volume of the 'Medico-Chirurgical Transactions' there is a report on the solvent treatment of calculi by the Rev. Stephen Hales and Mr David Hartley; the experiments, which were suggested by Cheselden, were carried on by Mr Sharpe at Guy's Hospital and by Mr Gardiner at St George's Hospital.* But this seems to have been nearly the last of such experiments, which were frequent in the eighteenth century.

Sir Wm. Roberts has reintroduced a *solvent method*, which at least for calculi of lithic acid deserves a full trial before more radical measures are entertained ('Med. Chir. Trans.,' 1866). With calculi composed of lithic acid he began by making a careful series of experiments outside the human body, exposing them to the action of a slow stream of a solution of carbonate of potass, which proved to be more effective than the carbonate of soda. With a liquid containing from forty to sixty grains of the alkali to the pint, he found that stones lost from 15 to 20 per cent. of their weight in twenty-four hours. Even with liquids containing twenty or thirty grains to the pint the solvent action was considerable. But what is very remarkable is that above the strength of sixty grains it ceased, in consequence of the formation of a tenacious white crust of alkaline biurate upon the surface of the concretion. The next step was to ascertain what doses of the vegetable salts of potass would give to the urine an alkalinity equivalent to about fifty grains of carbonate in the pint; and it was found that this could be effected in adults by the administration of forty to sixty grains of the acetate or citrate, dissolved in three or four ounces of water, every three hours; in children by about half the quantity. Some patients find that the acetate agrees with them better than the citrate, in others the reverse is the case. As the citrate of potass of the shops is apt to be impure, Roberts advises that it should be prepared by neutralising a solution of the bicarbonate with crystallised citric acid; the following formula yields sixty grains of the citrate to the ounce:—℞ Potass. Bicarb. ʒxij; Acid. Citric. ʒviij gr. xxiv; Aq. ad ℥xij.

It is not to be supposed that the urine passed by patients taking such doses of the potass salts can be maintained at an absolutely constant degree of alkalinity. On the contrary, it varies from hour to hour, but generally within the limits which correspond with the highest solvent action upon calculi; and Roberts has found experimentally that such urine, when it is allowed to pass over a uric acid stone outside the body at blood-heat, dissolves it at the mean rate of twelve and a half grains in the twenty-four hours. Clinically, it is obvious that the best proof of the power of urine, when rendered alkaline in this manner, to act upon calculi within the body, is to be obtained in the case of vesical calculi, of which the presence and the approximate size can be determined by sounding before treatment is begun, and which can, if necessary, be removed by lithotomy afterwards. In one case Roberts, after thirty-nine days' treatment, obtained the clearest evidence that a solvent action had been exerted; at the end of that time lithotomy was performed and the stone was found to be eroded to a con-

* Injections of soap-ley into the bladder was commonly practised in the eighteenth century (Heberden), and soft soap was the chief ingredient of the nostrum for which Parliament paid £5000 in 1739 (cf. p. 514).

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siderable extent, so that an incomplete layer of oxalate of lime was exposed, part of which was actually undermined. The proof of the efficacy of such treatment in the case of renal calculi is necessarily less complete; but there is a strong presumption in its favour.

The following case occurred to the author. A man came with a number of little uric acid calculi which he had been passing frequently. A vegetable salt of potass was prescribed, and a fortnight later he brought a single concretion, the only one he had passed, coated over with a white layer, which looked like phosphates, but which may have consisted of the biurate of potash. There seems to be little doubt that many other concretions must have been dissolved, for all the renal symptoms which had been troubling him disappeared, and he voided no more calculi.

The great drawback to this solvent treatment is that it is unfortunately altogether ineffectual when a stone consists of oxalate of lime, and that it fails even in the case of mixed calculi as soon as a complete layer of the oxalate is reached. It has been supposed that by rendering the urine alkaline one runs a risk of bringing about a deposition of phosphates, and so of actually augmenting the size of a calculus. But Roberts has shown that so long as the alkalinity is due to a fixed base there is no danger of this result; and as a matter of experience, he has found that after the treatment has been continuously carried out for three months, an oxalate of lime calculus in the bladder has remained entirely free from phosphatic incrustation. On the other hand, in the experiments already referred to, in which uric acid calculi were exposed outside the body to a slow stream of urine rendered alkaline by fixed alkali, and in which the calculi underwent solution, it was ascertained that as soon as ammoniacal decomposition of the urine occurred, a layer of mixed phosphates was deposited, and all further solvent action ceased. It is therefore useless to attempt a solvent plan of treatment unless the urine is acid. But even when putrefaction of the urine within the urinary passages has begun, the administration of benzoate or salicylate of soda may sometimes succeed in arresting this change, and in restoring the natural acid state of the fluid, so as to bring the case again within the scope of solvent remedies.

Unfortunately, among adults of middle age, the frequency of oxalate of lime calculi compared with those of uric acid is greater than would appear from the statements usually made. Perhaps these statements are based upon museum specimens obtained by lithotomy, or upon other cases of vesical calculi. When the presence of an oxalate calculus is suspected, the only prospect of cure apart from surgical operation lies in the possibility that it may either pass down the ureter and be voided, or else become "encysted," so as to cause no further symptoms. The possibility of the latter occurrence was especially insisted on by Dr Rees in the Croonian Lectures for 1856.

Operative treatment.—As in cases of intestinal obstruction and of gallstones, so in those of renal calculi and pyelitis, abdominal surgery has, since the introduction of antiseptic precautions, become justly bolder than before, and the results obtained are scarcely less brilliant than in ovariotomy. The operations are *nephrotomy*, or incision of a suppurating kidney with subsequent drainage; *nephrolithotomy*, or removal of a renal calculi from the pelvis; and *nephrectomy*, or removal of the entire diseased kidney.

In all cases of protracted and severe pyelitis the question of surgical interference must be taken into consideration, and it should not be delayed too

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long; for the chances of recovery are much greater at an early period of the disease than when it is far advanced. If there is an abscess in the loin, there is no doubt of the advantage of thoroughly exploring it, and of searching for and removing any calculi that may be present; and even when there is no evidence of more than pyonephrosis it is almost always advisable to cut down upon the kidney in the loin, or to lay open the suppurating cavity, so as to allow of its thorough drainage. How successful this operation of *nephrotomy* may sometimes be is well shown by a case related by Rosenberger, of Würzburg ('Trans. Internat. Congress,' 1881). The patient, a medical man, had during the previous year been incised above the ilium, with discharge of several pints of offensive pus, and he was reduced to a skeleton when the lumbar operation was performed. Yet, the cavity having been washed out with carbolic acid and a drainage-tube inserted, he gradually regained his health and resumed his practice. When, however, a large branched calculus is found occupying the renal pelvis, the attempt to extract it may be as dangerous a procedure as the complete removal of the kidney. A case of this kind, which occurred to Mr Morrant Baker ('Trans. Internat. Congress, 1881'), proved quickly fatal by shock and by hæmorrhage from the walls of the dilated renal pelvis.

Nephrectomy, or excision of the kidney, is often a formidable operation. When there is a large pyonephric sac, a lumbar incision may fail to give room for its extirpation—as has been pointed out by Mr Howard Marsh ('Trans. Clin. Soc.,' 1882). Another difficulty is illustrated by two cases of Mr Barker's ('Med.-Chir. Trans.,' lxiv); in each of them the kidney was found to be surrounded by a mass of dense vascular tissue, which could not be removed. Moreover, we cannot be sure of the condition of the opposite kidney. Cases have been recorded in which fatal suppression of urine has occurred; for the organ affected with calculous pyelitis, of such severity as to justify its extirpation, was nevertheless the only functionally active kidney which the patient possessed. To obviate this risk Czerny has proposed to make two stages of the operation, first producing a urinary fistula, and after an interval proceeding to nephrectomy. Another suggestion, made by Simon, of Heidelberg, a pioneer in renal surgery, is that in female patients, after dilating the urethra, it may be possible to catheterise each ureter separately; and Dr Teichmann, who for several years carried on investigations on this point in the *post-mortem* room at Guy's Hospital, believes that even in the male subject he can, with an instrument introduced along the urethra, nip up the mouth of each ureter in turn, and so withdraw from the bladder the secretion of each kidney separately.

Nephrolithotomy.—When no lumbar abscess or even pyelitis is present, cutting down upon a kidney with the object of removing a calculus—a procedure which was condemned by Sir Benjamin Brodie as dangerous and absurd—is now proved to be feasible. The first successful operation was by Mr Henry Morris at the Middlesex Hospital ('Clin. Trans.,' xiv) in 1880. The patient, a girl aged nineteen, was admitted under the care of Dr Coupland. She had for several years been liable to severe paroxysmal pain in the right lumbar region, which made her life as a domestic servant unendurable, and for at least two years her urine had often contained blood. Mr Morris cut down upon the kidney, and with his forefinger almost at once detected "something rounded, about the size of the uncut end of a pencil, causing a slight irregularity of the surface of the organ at a spot just a little behind the hilus." With a bistoury he incised

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the kidney at this spot, and succeeded in removing a calculus, which weighed thirty-one grains, and consisted of oxalate of lime. The patient made a good recovery. In vol. xv of the 'Clinical Society's Transactions' two similar cases are recorded, each of which was no less successful than that of Mr Morris. One, by Mr Marcus Beck, is that of a young man aged nineteen, who had suffered for twelve years from symptoms of stone in the left kidney, some pain in the loin increased by movement, hæmaturia, and great irritability of bladder. Mr Beck exposed the kidney, and on thrusting a darning-needle into the organ a stone was at once felt and extracted. It consisted of alternating layers of uric acid and of phosphates. In the other case—Mr Butlin's—the symptoms were neuralgia of the right testicle, which had continued for ten or twelve years, with some pain in the loin; but the urine never contained either pus or blood, though crystals of oxalate of lime were almost always present, and often a trace of albumen. The kidney having been exposed, a hard body was felt with the finger, and removed; it proved to be an oxalate of lime calculus. In vol. xvi of the 'Clinical Society's Transactions' there is a fourth case of successful nephrolithotomy (the stone weighing 473 grains) by Mr May, of Birmingham, and a fifth by Mr Howse. (See also the same 'Transactions' for 1884, 1885, and 1887.)

The chief difficulty is that of diagnosing with sufficient certainty that the stone is present, and that it is too large to pass down the ureter into the bladder. Mr Morris cites seven instances, in each of which an incision down to the kidney has been made without any stone being detected. See cases of this operation and of nephrectomy by Prof. Czerny, Mr Baker, Mr Lucas, Mr Barwell ('Internat. Cong., 1881,' pp. 242—279), and Mr Thos. Jones, of Manchester ('Brit. Med. Journ.,' June 2nd, 1883); also Sir Spencer Wells's remarks in his treatise on 'Abdominal Tumours' (p. 199), Mr Henry Morris's monograph on the subject, and the report of a discussion on Renal Surgery at Leeds in 1889 ('Brit. Med. Journ.,' ii, 1881).

One of the most remarkable examples of this branch of surgery was Mr Lucas's nephrolithotomy of one kidney following nephrectomy of the other and ending in the patient's recovery; it was referred to above (p. 527).

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TUBERCLE, CANCER, PARASITES AND ABNORMALITIES OF THE KIDNEYS

"When death's pale horse runs away with a person on full speed, an active physician may possibly give them a turn; but if he carries them on an even slow pace down hill too, no care nor skill can save them."—JOHNSON.

Tuberculous pyelitis—Its pathology—Symptoms, causes, and treatment.
Malignant disease—Sarcoma—Carcinoma—Symptoms—Nephrectomy.
Hydatids of the kidney—Chyluria and Filaria sanguinis—Endemic hæmaturia and Bilharzia—Renal malformations—Floating kidney.

THE present chapter completes the subject of renal diseases by dealing with degenerations and new growths, parasitic affections, and displacements of the kidney.

TUBERCULOUS DISEASE OF THE KIDNEY.—In cases of acute tuberculosis, miliary tubercles are found after death in the kidneys, as in other organs, but they are generally few in number, so far as the naked eye can judge. Even when numerous they are not known to produce any physiological disturbance, nor do they affect the course of the disease. Ebstein says that the epithelial cells of the convoluted tubules are generally in advanced granular degeneration; but the urine is free from albumen, and there are no symptoms which point to the kidneys during life.

The affection now to be described is of a different nature. It consists in the gradual destruction of the kidney, generally of one side only, by the formation of cavities with caseous walls, which may fairly be called *vomicæ*, from their resemblance to vomicæ in the lungs. Precisely the same differences of opinion have prevailed about this "nephro-phthisis" as about the corresponding pulmonary disease. Some observers have refused to recognise it as tuberculous, regarding it as a mere "caseous inflammation," and declare that if tubercles are found they are secondary and accidental formations. But there was always strong evidence, from the close likeness which exists between this renal affection and those in the lungs and in other organs, that they have all the same "specific" origin; and now, since the discovery of the tubercle bacillus, the truth of this view has been established not only as concerns the so-called "caseous" and "pneumonic" forms of pulmonary phthisis, but also for the corresponding "scrofulous" inflammation of the lymph-glands, the testes, and the kidney.

Anatomy.—There have been some differences of opinion as to whether tuberculous disease of the kidney has its starting-point in the cortex or in the medulla of a pyramid. The following observations of the author's show that it may begin in either situation, and also that, as a rule, tubercles are present from the first, and that even when this is not obvious the character of the morbid process is yet unmistakable.

(1) In 1874 a woman aged twenty-six died in Guy's Hospital of

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phthisis; one kidney had in its cortex a cluster of yellow tubercles, from which a yellow streak extended down the corresponding pyramid. (2) In 1873 a girl aged six died of tubercular peritonitis; in one kidney, near the apex of a pyramid, was a round tubercle just beginning to soften; the mucous membrane of the pelvis of the other kidney was covered with tubercles, as was also the lining membrane of the bladder. (3) In the same year another girl, aged four and a half years, died of acute tuberculosis, the bronchial glands being caseous; in a pyramid of one kidney, not quite reaching either its free surface or its base, there was a well-marked vomica, with an indurated caseous border. (4) In the same year a man aged forty died of phthisis, with caries of the spine; in one kidney there was early tuberculous disease, ulcerating so as to form a conical cavity; in the adjacent part of the cortex there were caseous nodules up to the size of swan-shot, some extending to the surface of the organ. (5) In 1863 a boy aged fifteen, who had been admitted for vesical symptoms, died of tubercular meningitis; both kidneys contained "softening tuberculous matter as well as distinct tubercles;" in the pelvic mucosa of the right kidney there were well-marked isolated tubercles, and also in that of the corresponding ureter and of the bladder, near its neck. (6) In the same year a man aged twenty-two died of acute general tuberculosis and tubercular meningitis; the kidneys were stuffed with soft yellow tubercles, in some places collected in groups, and apparently about to soften into abscesses; the pelvis of the right kidney was lined with a layer of granular lymph, and this extended down the ureter into the bladder, which itself was affected with tuberculous ulceration; all round the opening of the right ureter into the bladder the mucous membrane was covered with isolated tubercles of various sizes. (7) In 1876 a man aged thirty-four died of bronchitis and emphysema, the lungs being quite free from tuberculous lesions; in one kidney a single pyramid was eaten away at the tip, and the rest of it was changed into a gelatinous material of sulphur-yellow colour. (8) In 1875 a man aged twenty-three died of pleurisy and of tubercular disease of the lung; in the substance of a single pyramid of one kidney there was an early patch of caseous infiltration. (9) In 1878 a woman aged twenty-eight died of phthisis: one kidney contained a circumscribed cheesy mass the size of a damson, and two of its pyramids were also affected with early tuberculous lesions. (10) In 1879 a youth aged nineteen died of spinal disease with psoas abscess; one kidney showed several early tubercular masses excavating the cortex and forming vomicæ with cheesy walls; in the mucous membrane of the pelvis there were also scattered grey tubercles and caseating patches. (11) In 1880 a woman aged twenty-five died of bronchitis; in one kidney there were two typical vomicæ with cheesy walls. (12) In the same year a man died of lardaceous disease of the viscera, the result of caries of the spine; in one kidney there were two vomicæ, one in the cortex, the other in a pyramid, with opaque caseating tubercles round them. (13) Another man, also in 1880, died of phthisis; in one kidney a pyramid was eroded by a single tuberculous ulcer, and beyond this, in the cortex, there were opaque white tubercles.

These cases show that while the characters of tuberculous disease of the kidney vary within certain limits, the morbid process is always fundamentally the same, and the result to which it tends identical. They also indicate clearly how close is the relationship between this affection and

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undoubtedly tuberculous lesions of other organs. When the renal disease has time to run its course and to prove directly fatal, it is obviously less likely to have been preceded by tuberculous lesions elsewhere; but even then they often develop sooner or later, not only in other parts of the urogenital apparatus, but also in remote organs.

In a single case, inspected in 1874, an ulcer which excavated a single pyramid of one kidney had a hard calcareous wall, which seemed to indicate that the affection was arrested in its progress, and might have remained stationary had the patient lived. All the author's other observations tend to confirm the usual opinion, that when once tuberculous disease has begun, it goes on to destroy the whole kidney. The vomicæ, which correspond more or less accurately with the affected pyramids, keep increasing in size, their caseous walls spreading further and further into the renal tissue, until they lie close beneath the capsule, and touch one another on all sides, or communicate by lateral openings. At the same time the capsule undergoes great thickening, and may become almost as hard as cartilage. The mucous membrane of the pelvis, from an early period, is converted into a thick whitish-yellow layer. Any parts of the cortex that escape removal by ulceration are converted into a tough, white, fibrous material that shows no trace of a tubular structure, as was well illustrated by a specimen which was shown by Mr Lucas to the Pathological Society in 1875. Almost always there is enough of this material to provide septa by which the excavated organ is permanently divided into a series of sacculi, more or less completely shut off from one another; and these may at length lose their caseous walls and become bounded by a smooth lining membrane. Their contents vary in consistency in different cases; sometimes they are full of a caseous pulp, sometimes of a substance like putty or mortar, with abundant earthy salts; occasionally some of them ultimately come to contain a transparent yellowish fluid, in which may be seen crystals of cholesterine.

It is obvious that such matters could hardly accumulate in large quantity were there a way freely open for their escape. The fact is that from an early period in the course of the disease the *ureter* is commonly blocked and impervious. Its mucous membrane undergoes the same change as that which affects the renal pelvis; its other coats are indurated, and it becomes converted into a hard cord, which may be as thick as a pencil or thicker, and has but a narrow lumen left in its centre.

Tubercular ulceration of the *pelvis* of the kidney is generally secondary to similar disease of the bladder or testes, or both; and it usually leads to vomicæ in the substance of the kidney. It is sometimes, however, secondary to calculous pyelitis, or at least the two coincide.

The *bladder*, in its turn, shares in the morbid process. Sometimes an excavated ulcer forms round the orifice of the ureter. Sometimes the whole of the vesical cavity becomes lined with patches of cheesy material, and more or less extensively ulcerated. In some cases, however, the vesical affection begins at the same time with, or even before, that of the kidney.

Not infrequently the morbid process extends into the *urethra*. In three cases examined after death by the author it reached the external orifice, where the rough greyish-yellow appearance of the mucous membrane might easily have been seen during the patient's life. In one of these cases the canal was remarkably widened, so much so that a No. 16 catheter was required to fill it. When the affection was advancing along the urethra up to the time of death, we sometimes find at an autopsy obviously

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recent grey tubercles, apparently situated upon the surface of the mucous membrane.

In many cases the *genital organs*—the prostate, the vesiculæ seminales, the vasa deferentia, and the testicles, some or all—take part in the disease. For example, apart from cases in which the patient complains of anything that the surgeon would recognise as "strumous disease of the testicle," one or more hard nodules can often be felt in the epididymis if careful search is made for them. As a rule, the vas deferens retains its natural size when affected with tuberculous inflammation, but in one case the whole spermatic cord appeared obviously indurated during life. In the prostate the general result of tuberculous disease is to cause moderate enlargement, with the formation of vomicæ having caseous walls; and the same may be said also of the vesiculæ seminales, except that the cavities which are seen in them are formed out of those that are naturally present.

It is a remarkable fact that in women tubercular nephritis is seldom attended by tubercle of the pelvic organs. Exceptionally, however, the internal genitalia, and particularly the Fallopian tubes, may become tuberculous along with a like affection of the kidneys.

Altogether, among some thirty-four cases of tuberculous disease of the kidney from our hospital reports of autopsies in which the renal affection was so far advanced as to have a more or less important share in the result, hardly a single instance occurred in which the other parts of the urogenital apparatus were free from tubercular lesions. Of the kidneys themselves one alone is often affected, while the other shows no trace of tuberculous lesions: among the thirty-four cases this occurred in twenty-two, while in the other twelve the disease was bilateral, but always much older and more advanced on one side than the other. It has been stated that the right kidney is less liable to become tuberculous than the left; and in the thirty-four cases the relative frequency of the disease on the two sides was as three to four.

Symptoms.—The indications of tubercular nephritis during life are often remarkably slight. There may be pain in the loins, occasionally paroxysmal; and in some few cases there is tenderness on pressure.

The diseased organ very rarely forms a tumour that can be felt by manipulation of the abdomen. In Mr Lucas's case, already referred to, which occurred in a little girl of seven, a circumscribed tumour was detected in the right hypochondriac and lumbar regions; after death the kidney was found to be six inches in length, and eleven in circumference. In another case at Guy's Hospital a tumour is said to have been felt, but at the *post-mortem* examination the kidney only weighed eighteen ounces. In a third case there was a swelling which for a time led to the suspicion that the disease was malignant; but this proved to be an abscess behind the kidney, without much enlargement of the organ itself. As a rule a tuberculous kidney is but little above the natural size.

The *urine* is occasionally normal, no doubt owing to the ureter of the diseased kidney being blocked, as already described. It is thus possible for tuberculous disease to go on to complete destruction of a kidney without any discharge from it reaching the bladder. But in most cases the urine is seldom free from either pus or blood, or both.

Hæmaturia is neither constant nor profuse; among eighteen fatal cases with notes of the symptoms during life, which occurred at Guy's Hospital, in only ten is blood said to have been at any time observed in the urine;

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and in most of these cases the bladder was likewise affected with tuberculous disease, so that the exact source of the hæmorrhage was after all doubtful. The most striking case is one of a man who a year before his death was stated to have one day passed a pint of blood by the urethra, after straining his back in lifting a heavy weight.

Pyuria is a more constant and distinctive symptom. The quantity of pus in the urine is often so considerable as to form a thick deposit. Under the microscope a large proportion of the pus-cells are often found to be undergoing disintegration. The sediment may also contain granular amorphous masses insoluble in acetic acid, and even shreds of connective tissue, the presence of which is very significant. The *bacillus* of tubercle is frequently to be recognised in the pus-cells when successfully stained.

In many cases the urine retains its acid reaction throughout the whole course of the disease, but sometimes—probably always when the bladder is also affected—it becomes ammoniacal and fœtid. There is then severe dysuria. Sometimes pain in micturition, and strangury have been conspicuous features of a case during life, but on *post-mortem* examination the bladder has been found apparently healthy.

Pyrexia is generally present if there are other marked symptoms, and it may assume a hectic type. It is attended with loss of appetite and often with nausea and diarrhœa. Usually emaciation ensues and brings the disease to a close.

Course and event.—The duration of these cases from the time when the patient is first discovered to be ill is commonly from six months to two or three years. But it would be a mistake to suppose that this fairly represents the course of the malady. On the contrary there are many instances in which its progress is so slow that the opposite kidney has time to become hypertrophied, and probably carries on its function with perfect efficiency. Ultimately this healthy kidney may in its turn suffer from the effects of pressure upon its pyramids, as the result of tuberculous disease of the bladder; and an ascending suppurative nephritis may set in and rapidly bring the case to an end; or the hypertrophied organ may become affected with Bright's disease, as mentioned at p. 528; or the drain of pus from the tuberculous kidney or from other parts of the urogenital apparatus may lead to the development of lardaceous disease, both in the opposite kidney and in the viscera generally. In the last-mentioned event the urine may be albuminous and may contain tube-casts, when from obstruction of the tuberculous ureter no pus is for a time being discharged.

In one case at Guy's Hospital the immediate cause of death was the extension of ulceration from the tuberculous kidney into the peritoneal cavity. Others have ended in the formation of perinephric abscesses, which have pointed in the loin, or have burrowed down in the sheath of the *psoas* muscle, until they made their way into the hip-joint or appeared in Scarpa's triangle. Lastly, the disease often terminates by the supervention of phthisis, or tubercular peritonitis or meningitis, or general miliary tuberculosis.

Ætiology.—With regard to the causes of tubercular disease of the kidney, apart from those concerned in the production of tubercle in general, we know nothing. It shares the hereditary tendency of phthisis.

It is at least twice as common in men as in women.

Of twenty-nine cases at Guy's Hospital in which the renal disease was the principal cause of death, in three death occurred between the ages of ten and twenty, in twelve between twenty-one and thirty, in eight between

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thirty-one and forty, in five between forty-one and fifty, and in one between fifty-one and sixty.

Treatment.—There is little to be done medically beyond placing the patient under favourable conditions and giving cod-liver oil and steel.

At an early stage, if the diagnosis could be made with certainty before the bladder had become affected, it would probably be advisable to run the risk of nephrectomy. Mr Morrant Baker ('Trans. Internat. Congr.,' 1881, vol. ii, p. 262) performed this operation in the case of a girl aged seven, with the result that five months afterwards the child had greatly improved in health, could play all day long, and go out of doors for a walk. Her illness had begun with hæmaturia about twenty-two months before the kidney was excised. In vol. xv of the 'Clinical Society's Transactions,' Dr Goodhart and Mr Golding Bird have recorded a case of nephrectomy for tuberculous disease which proved fatal about four hours after the completion of the operation. The patient had been seriously ill for about eight weeks, but had complained of pain in the back for eighteen months. At the autopsy the ureter was found to be diseased in its whole length, and the mucous membrane of the bladder was thickened and opaque. In the prostate also there was some cretaceous material. It is the liability of the disease to affect other parts of the urogenital apparatus that renders nephrectomy a doubtful expedient in most cases where the symptoms are enough advanced to render diagnosis certain. For this reason it will perhaps be found that the prospects of an operation are more favourable in women than in men (p. 547). In a recent case in Miriam Ward, Mr Jacobson removed the kidney from a girl of eighteen, and she made an excellent recovery. There were several large tubercular vomicæ in the organ when excised.

SARCOMA AND CARCINOMA OF THE KIDNEYS.—When a malignant growth, in whatever part of the body it may begin, gives rise to secondary nodules in distant organs, it not infrequently happens that some of them are seated in the kidneys. But in such cases the renal affection is seldom of clinical significance, except sometimes by causing more or less hæmaturia, or (if the primary growth is melanotic) by giving to the urine a brown or black colour.

Primary malignant tumours of the kidney are decidedly rare. Statistics from various sources are cited by Ebstein (in 'Ziemssen's Handbuch') in proof of this fact, and they are fully borne out by the reports of *post-mortem* examinations at Guy's Hospital, where only fifteen cases occurred during a period of twenty-two years.

Anatomy.—Two distinct affections were formerly included under the name of "malignant disease," or "cancer" of the kidney. One of them occurs chiefly in infants and in children up to the age of eight or nine years, though we have had two cases in boys aged respectively eleven and seventeen. This, although Ebstein speaks of it as carcinoma, is, as a rule, *sarcoma*, like most of the malignant growths of other parts that are met with in children. It forms a smooth rounded mass, which sometimes reaches an enormous size, weighing ten, twenty, or even thirty pounds, so that at one autopsy it was said that instead of the tumour being removed from the child's body, the body was removed from the tumour. Such a growth is commonly very soft and elastic, so that it may appear to fluctuate; it is therefore likely to be punctured by the surgeon, a procedure which is gene-

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rally harmless, but which may be followed by a sharp attack of peritonitis. Sometimes, besides blood, a small piece of sarcomatous tissue is brought away in the orifice of the trocar. These sarcomata of the kidney are very vascular, and interstitial hæmorrhage often takes place, causing a sudden increase in their size.* They grow, too, with great rapidity, destroying life sometimes within a few weeks, and almost always in less than a year from the time when they are first discovered. They commonly affect one kidney only, but Dr Abercrombie showed to the Pathological Society (in 1880) three cases, occurring in young children, in each of which both kidneys were invaded at the hilus by sarcoma.

A few rare and interesting cases have been recorded of renal sarcoma containing striated muscle; they probably have their origin during embryonic life before the protovertebræ are fully differentiated (see 'Path. Trans.,' 1880).

A sarcomatous tumour in the position of the kidney is not always seated in that organ. Some years ago Dr Dickinson brought before the Pathological Society (vol. xxi, p. 397) a specimen in which the growth occupied the lumbar glands, and merely pushed the kidney before it; and in a case of Dr Day's it affected the capsule of an otherwise healthy kidney.

The other form of primary malignant disease of the kidney is true *carcinoma*. It is seen chiefly in persons past middle age; in almost every instance that occurred at Guy's Hospital during twenty years the patient was more than forty-five years old. It is much more common in men than in women. The tumour is not generally very large, but it may sometimes occupy all one side of the abdomen, or even appear to fill the whole cavity, like a great ovarian tumour in a woman. In such cases, however, a good part of its bulk is commonly made up of hollow spaces containing a blood-stained fluid; or there may be a large accumulation of a similar fluid in the dilated renal pelvis, which has been shut off from the ureter.

Carcinomata of the kidney are often spoken of as scirrhous, but they seldom deserve that name. They appear to be usually of only moderate firmness, and of the glandiform type (*adenoma malignum*). In very rare instances they are found to have undergone colloid degeneration.

In 1876 the author showed to the Pathological Society (vol. xxvii, p. 204) a *carcinoma lipomatosum* (of Cornil and Ranvier) growing in the kidney. It looked like adipose tissue, but had extensively invaded the renal veins, as well as the substance of the organ, and possessed a typical alveolar structure, the alveoli being filled with large cells loaded with oil-drops. The specimen had no clinical interest, for the patient died of another disease.

Sometimes carcinoma of the kidney causes enlargement of the whole organ, the distinction between cortex and pyramids being still traceable in the tumour; sometimes only certain parts are affected, rounded or irregular masses of growth being separated by tracts of healthy tissue. Following Waldeyer, most pathologists now believe that the renal epithelium is the starting-point of the growth.

Like sarcoma, carcinoma, though as a rule it affects only one kidney, has occasionally been found in both.

Ætiology.—With regard to the causes of primary malignant disease of

* For an account of the histology see Dr Paul's paper with figures in the 'Pathological Transactions,' vol. xxxvii.

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the kidney nothing definite is known. In a few cases it has appeared to be the result of a blow or of a kick in the loin; but one may doubt whether it was not really present before, and whether the injury did more than bring on symptoms such as hæmaturia, by which attention was first drawn to it.

In the following two cases cancer appeared to supervene on another disease of older date. One occurred in a man of forty-five, who was said to have been troubled by passing a gelatinous substance in his water for twenty or thirty years. The right kidney was found to have its calyces dilated into a number of chambers, and in the pelvis lay a large, irregularly branched calculus, "like a knotted branch of a tree." Growing from the upper part of the organ was a cancerous mass, which also extended upwards behind the liver, and penetrated through the diaphragm into the lung. The other case was that of a man aged sixty-six, who had an attack of hæmaturia twenty years previously, and who came under treatment for a recurrence of this symptom ten months before his death. The lower part of the kidney showed the ordinary appearances of hydronephrosis, with "sacculation" from distension of the calyces; but into many of the sacculi soft masses of carcinoma were projecting, and the upper part of the organ formed a solid tumour. Apart from the anatomical appearances, it is almost inconceivable that the cancer could have dated back as far as the earliest attack of hæmaturia, although Ebstein speaks of a case of renal cancer which was believed to have lasted for eighteen years. The occurrence of a calculus in the kidney as a complication of malignant growths seems not to be very rare; it may probably be the primary source of irritation, as with cancer of the gall-bladder (p. 353); but sometimes a phosphatic stone may be of later development than the tumour, especially if pyelitis happen to be present.

The ordinary *duration* of cases of carcinoma of the kidney in adults is probably from six months to two years after the first appearance of symptoms.

The symptoms of a malignant growth in the kidney, whether sarcomatous or carcinomatous, are mainly three: the presence of an abdominal tumour, hæmaturia, and pain.

1. The *tumour* has the usual characters of tumours seated in the kidney. It occupies one side of the abdomen, having its centre opposite the lumbar region, between the lower ribs and the iliac crest. It often bulges into the loin, and one can move it slightly forwards by pressing the loin with one hand, while the other is placed over the front of the abdomen. It does not descend during inspiration, and the fingers can be inserted between it and the ribs, showing that it is not seated in either the liver (if on the right side) or the spleen (if on the left). It is sometimes perfectly smooth and uniform, sometimes more or less uneven or lobulated. Overlying it in front there is commonly a part of the colon (the hepatic or the splenic flexure, according to the side affected), which either may be felt as a ridge, or may be traced by its tympanitic percussion-sound. The dulness obtained on percussion over the tumour is continued into that of the lumbar region behind, whereas a splenic, ovarian, or other tumour, not growing from the loins, would be bounded externally by the resonance of the ascending or the descending colon. Sir Spencer Wells has proposed, in doubtful cases, to inflate the gut with air, so as to render the position of the bowel more conspicuous. In one case the author could feel several coils of small intestine in front of the growth beside the colon; they were freely moveable, and slipped away from the finger under manipulation.

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Roberts notices a remarkable instance in which not only was the stomach made out during life to lie in front of a cancerous left kidney, but the spleen could be distinctly felt as a separate mass in the iliac fossa, lying over the lower and inner part of the tumour. Mr Holmes recorded, in vol. xxiv of the 'Pathological Transactions,' a case in which a malignant growth of the kidney pulsated, and was attended with a bruit, so that aneurysm was suspected.

There is often considerable distension of the superficial abdominal veins, which probably may be due to compression of the inferior vena cava by the tumour, or by enlarged glands; but in many instances the growth fungates into the renal vein, and it may even protrude into the cava, so as to narrow its calibre. As a result the feet and legs may become œdematous; and Rindfleisch speaks of embolism of the pulmonary artery as being sometimes caused by the detachment of portions of the cancerous thrombus. In a case that was observed at Guy's Hospital in 1871 the disease made its way into one of the veins of the colon, and thence into the portal vein and its branches within the liver; ascites was the result.

2. *Hæmaturia* is by no means a constant symptom. Ebstein found it absent in twenty-eight out of fifty-two cases collected by him. Very often it is the earliest indication that anything is amiss with the patient. Sometimes it is directly brought on by a blow or a fall. It may recur again and again at regular intervals for a considerable time before any tumour can be detected. It is then apt to be set down to a renal calculus, but one distinction is that the hæmaturia is not generally attended with a marked increase or aggravation of pain. In many cases the bleeding comes from portions of the growth that protrude into the pelvis of the kidney. But occasionally its source is from a tumour within the cortex, and if such is the case tube-casts containing blood-corpuscles may be found in the urine, as stated by Ebstein.

3. The *pain* produced by a malignant tumour of the kidney is very variable in degree; sometimes it is altogether absent. Its usual seat is in one lumbar or hypochondriac region, but it may radiate widely over the lower part of the chest to the front of the abdomen, or to the crista ilii, and even down the thigh. It may be either constant, dull and aching, or paroxysmal, sharp and cutting in character. Sometimes there is much tenderness to pressure. The pain is seldom, if ever, attended with retraction of the testicle, in which respect it differs from the pain due to calculus. If, however, clots of blood formed in the pelvis of the kidney should become impacted in the ureter, the pain may assume a different character, and become exactly like that which accompanies an attack of renal colic.

Other symptoms that may be present in cases of malignant growth in the kidney are anorexia, nausea, vomiting, and constipation or diarrhœa. The patient usually becomes wasted, anæmic, and cachectic. The temperature remains normal or subnormal, and the pulse may be slow. Death is usually by exhaustion, and is sometimes preceded for a few days by stupor or insensibility. In a case recorded by Bright in the first volume of the 'Guy's Hospital Reports,' the tumour gave way into the abdominal cavity, causing a large extravasation of blood. Some years ago a woman was admitted into the hospital for paraplegia, which had been coming on during two months, but she was said to have had hæmaturia four months before, and to have been ill for a year. At the autopsy it was found that there was a primary cancer of the left kidney, and that the growth had extended into

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the spinal canal. A like case has been observed by Cornil. In a patient who died in Guy's Hospital in 1870 all the symptoms were cerebral, and the immediate cause of death was the presence of secondary tumours in the brain.

Diagnosis.—It can be easily understood from the foregoing description that it is often difficult or impossible to recognise the presence of malignant disease of the kidney. A man aged thirty died in Guy's Hospital many years ago of wasting and anæmia, whose case excited great interest from there being no discoverable local symptoms. At the autopsy it was found that the right kidney was the seat of a primary growth, which had destroyed nearly its whole substance, but did not reach its pelvis. When a tumour in the lumbar region is accompanied by cachexia and emaciation in an elderly patient it is probably malignant, and if hæmaturia is present the diagnosis becomes nearly certain. But there are no anatomical signs to distinguish between a renal growth and one starting in the lumbar glands, the adrenals, or the vertebræ.

Treatment.—It is obviously improbable that any large measure of success will ever be attained by nephrectomy in cases of sarcoma or carcinoma of the kidney. But some few cases have already turned out much more favourably than could have been anticipated. In 1877 Mr Jessop, of Leeds, removed an encephaloid tumour of the kidney from a boy two and a half years old; rapid recovery took place, but about eight months afterwards the disease returned, probably in the lumbar glands, and the case ended fatally a few weeks later. In 1878 Dr Martin, of Berlin, extirpated a sarcoma of the kidney, weighing twenty-eight ounces, in the case of a woman aged fifty-three; she was up on the eighth day, went home on the seventeenth; and Czerny speaks of her as being still well two years afterwards. In 1879 Lossen, of Heidelberg, performed nephrectomy in a woman aged thirty-seven, for an "angio-sarcoma" of the right kidney, which, being moveable, was mistaken for an ovarian tumour; she recovered in six weeks, and she continued to be in good health eighteen months later. In 1881 Czerny removed a large vascular sarcoma from a man aged fifty-three, who at the time of the operation was very cachectic, and suffered greatly from vomiting; two months later he left the hospital in complete health, with the proportion of red discs in his blood twice as great as it had been previously. Against these successes, however, must be set many cases in which extirpation of malignant growths in the kidneys has either been attempted ineffectually, or has proved quickly fatal by shock or by peritonitis (see Czerny's tabular statement in the 'Trans. Internat. Congress,' 1881, vol. ii, p. 249).

Apart from surgical interference, the only treatment is to relieve the symptoms as they arise.

Hydatid of the Kidney.—According to Davaine the kidney comes next to the lungs in the order of frequency among the organs liable to be infested with the echinococcus in its encysted state. In our records of autopsies at Guy's Hospital there are, however, only three or four instances; in one the parasite was the size of a plum; in another, of an orange; in a third it formed a bulging elastic swelling extending from the left hypochondrium into the loin, and containing two pints of fluid.

Clinically the diagnosis must generally be based upon the recognition of an abdominal tumour, having the characters above given of tumours of the kidney, more or less tense and rounded in form, painless, and possibly yield-

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ing fluctuation or palpable *fremitus*. The diseases for which it is most likely to be mistaken are hydronephrosis, soft sarcoma of the kidney, and (in the female) cystic disease of the ovary, as in a case in which Spiegelberg performed an operation which he intended for ovariotomy. A young woman was admitted into Guy's Hospital under Dr G. B. Babington, in 1854, with an abdominal tumour which had been wrongfully supposed to be a pregnant uterus. She passed first "skins and little bladders," and afterwards blood and pus from a hydatid of the right kidney. After many months she went out in good health, but still passing the echinococci and pus. It is instructive to notice that the tumour dated from a blow. A point which may sometimes aid in the diagnosis is the discovery of a second hydatid in the liver; this was the case, for example, in a patient who died at Guy's Hospital with a hydatid in the kidney holding two pints of fluid.

In many instances an echinococcus in the kidney probably remains for years—perhaps from childhood or middle age throughout the entire life of the host—without affecting the health. Or it may die, and dry up into a pultaceous or caseous mass, which henceforth has no power of doing damage. But in the majority of cases (if one may judge from hospital records) it sooner or later ruptures into the pelvis of the kidney, after which the daughter-cysts and scolices pass down the ureter and are expelled with the urine. Such cases present the symptoms of renal colic (see p. 521).

The presence of cysts or hooklets in the urine is not in itself proof of the existence of a hydatid in the kidney. For the pelvic pouch of the peritoneum is nearly, if not quite as frequent a seat of the echinococcus (cf. p. 405), and in such cases it sometimes opens into the bladder from behind, as in a case in which Mr Birkett (1885) drew off hydatids from the bladder during the patient's life. In two cases of pelvic hydatids there was a tumour in the hypogastric region of the abdomen, having exactly the shape and the other characters of a distended bladder.

In cases of renal hydatid, the passage of daughter-cysts down the ureter is often the first indication that anything is amiss with the patient; and when the parent cyst is small, no tumour may be discoverable in the loin. Not infrequently the rupture of the cyst is directly produced by a blow or by a fall; or the symptoms may appear to be brought on by riding or by driving, as in cases of renal calculus. Sometimes there is only a single discharge of daughter-cysts in the urine, and the patient afterwards remains perfectly well; sometimes the same thing recurs again and again, at long intervals, during a period of ten, twenty, or even thirty years; sometimes suppuration within the capsule of the cyst occurs, and blood and pus may be voided in the urine. Roberts, however, says that the ultimate prognosis is generally favourable. Of sixty-three cases which he collected, only nineteen were known to have ended fatally; and in nine of these the cause of death was some disease not directly connected with the renal affection. In some instances there has been ulceration through the diaphragm, with escape of daughter-cysts into a bronchial tube and expectoration through the air-passages; the prognosis is then very unfavourable.

The proper *treatment* of hydatid of the kidney, when a tumour can be detected, is puncture with a tubular needle, fitted to an aspirator. In two cases Roberts found that the withdrawal of only a drachm or two of fluid sufficed to destroy the life of the parasite, and caused it to pass very gradually into obsolescence, just as we found in the case of the liver (p. 402).

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CHYLURIA.—Prout first described a very remarkable condition of the urine, in which it looks white and milky when passed, and soon afterwards sets more or less completely into a soft jelly, like *blanc-mange*, which takes the shape of the vessel that contains it. Sometimes it solidifies within the bladder, and the result may be that there is pain and difficulty in micturition from obstruction of the urethra. The coagulum after a little while liquefies again; a material, somewhat like cream, collects upon the surface, and there falls a deposit which is generally of a pinkish colour, from the presence of a small quantity of blood. Prout recognised that the characters of the affection were exactly such as might be due to the admixture of chyle with the renal secretion. This view is confirmed by microscopical examination, which shows that the cause of the opacity is a finely granular material, not large fat-globules as in milk; and also by the application of chemical tests, for by ether a large quantity of fat may be extracted, and the urine also contains albumen in considerable amount. Occasionally glycose has been also present. Beside the "molecular base of chyle," granular leucocytes and sometimes a few red blood-discs are seen under the microscope.

This peculiar state of the urine is often far more marked a few hours after a full meal than when the patient has been fasting for some time.

It is to be noted that *chyle*, in a strict sense of the term, does not exist in the urine in every case in which that fluid is more or less opaline in appearance; if the clot which forms is almost translucent, the cause may be merely the presence of *lymph* that has passed through one or more glands on its way upwards to the thoracic duct. But if the clot is opaque, like *blanc-mange*, and if the state of the urine varies in relation to the patient's meals, there can be no doubt that the obstruction involves lacteals coming from the intestines.

Until recently the pathology of chyluria was a complete mystery, and there is scarcely anything more curious in the history of medicine than the way in which it has step by step been elucidated, until we now seem to know nearly all about it.

Prout noticed that a large number of those affected were born, or had lived for many years, in hot climates. Next, in 1866 Wucherer, in Brazil, detected in chylous urine certain minute living organisms, evidently the embryos of a nematode worm. Six years later, in 1872, the late Dr T. R. Lewis, in India, discovered similar embryos in the blood; and the parent worm (*Filaria sanguinis hominis*) has since been discovered, with the complete history of its development, which has already been set forth at length in the chapter on Entozoa (*supra*, p. 300).

The probable method by which the immature ova form obstructions in the lymphatic vessels has been also described (p. 303). The result is rupture of some of the lymphatics and extravasation of their contents. If a lacteal vessel thus bursts into the peritoneum chylous ascites is produced (p. 327).

The only hypothesis of the way in which the filaria produces chyluria is one put forth by Dr Manson, in a paper in the 'Pathological Transactions' for 1882. His idea is that so long as the discharge of embryos goes on after the manner above described, the parasite is innocuous to its host. But, from some cause or other, it happens in certain cases that, instead of the larval filariæ enclosed in their sheaths, ova in a much earlier stage of development, with unstretched shells, are extruded from the maternal vagina. Dr Manson has twice obtained such ova from the lymphatics; and probably they have been found in the urine also. Now,

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according to Dr Manson, they measure $\frac{1}{750}''$ in breadth by $\frac{1}{500}''$ in length; according to Dr Cobbold $\frac{1}{1650}''$ by $\frac{1}{1000}''$. In either case their transverse diameter is far greater than that of the embryos; and thus they fail to pass along channels which the embryos would find no difficulty in traversing. Dr Manson supposes, for instance, that when they are carried by the lymph-stream to a gland, they become impacted in the smaller lymph-channels in the cortex.

If the obstruction affects the abdominal or pelvic lymphatics, the result will be more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, or in the scrotum. In the former case the chronic œdema with hypertrophy which is produced is called *elephantiasis* (*Arabum*), or Barbadoes leg; in the latter it is known as *lymph-scrotum*. Roberts has related a remarkable case of intermittent chylous urine, together with lymphatic dilatation and lymph or chyle-vesicles scattered over the abdomen, which completes the chain of connection between these several disorders (loc. cit., p. 377).

Elephantiasis will be considered under diseases of the skin.

Lymph-scrotum consists in the formation of vesicles, which are in fact dilated lymphatics, and which discharge fluid either clear or milky in appearance, according as it is derived from a peripheral vessel, or from one which has already passed through a lymph-gland. The tissues of the scrotum are thickened, but feel soft and spongy. Sometimes similar vesicles form on the inner side of the thigh. In the course of years the flow of lymph commonly ceases, and the scrotum passes into a state of elephantiasis. But the elephantiasis may be developed in the first instance without the formation of vesicles or escape of fluid.

When *chyluria* occurs, there can be no doubt that distended lymph-vessels open upon the surface of some part of the urinary mucous membrane; but whether this takes place most often in the bladder, or in the ureter, or in the renal pelvis, has not yet been determined.

One peculiarity of the affection is that it is often intermittent, the urine from time to time losing its abnormal characters for days or weeks together. In a case recorded by Ackermann ('Deutsch. Klin.,' 1863) the patient always passed normal urine after he had been lying on his right side.*

The relations now ascertained to exist between the filaria and the chyluria of hot climates leave undetermined the pathology of the disease when it occurs in persons who have always resided in Europe. Among well-authenticated instances may be mentioned: Prout's original case, Dr Beale's, Dr Dickinson's, and Dr Morgan's at Manchester. In every case there is no doubt a definite fistulous communication between the lymphatics or thoracic duct and the urinary passages, whether caused by the filaria or any other agent.†

Chyluria as an endemic disease is found in Bombay and other parts of India, in China and Japan, in Mauritius and the Ile de France, in the Bermudas and the West Indies, in Guiana and Brazil, and in Queensland.

The autopsy upon Dr Stephen Mackenzie's patient ('Path. Trans.,' 1882, pl. xxii, p. 394) showed a large mass of dilated lymph-sinuses and glands, extending from the bifurcation of the aorta below to the diaphragm above, and occupying the whole of the space between the kidneys. The lower part

* For the sequel of this case see 'Brit. Med. Journ.,' 1864.

† See a case of chylous ascites admirably worked out and recorded by Dr White, of Belfast, in the 'British Medical Journal,' May 30th, 1885.

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of the thoracic duct was sinous and pouched, varying in diameter from three eighths to half an inch. About three inches above the diaphragm it became impervious, and was lost in a quantity of tough, dense material, apparently of inflammatory origin. In this case the communication between the lymph and the urine was probably in the kidneys.

It is obvious, from what has been stated with regard to the life-history of the filaria, that the *prevention* of the diseases due to this parasite is quite possible. Dr Manson suggests that wells and water-jars should be covered with a netting sufficiently fine to prevent the entrance of mosquitoes, but it must surely be a better plan to drink no water which has not been boiled or filtered. Care must also be taken to have all raw vegetables thoroughly washed with boiled water before eating them.

In the *treatment* of chyluria, when it is once established, very little can be hoped for from medicines. Bence Jones thought that by giving gallic acid to the amount of two drachms daily he was successful in restoring the urine to a normal state for periods of several months at a time. In Guiana the old women give decoctions of mangrove bark, and not without apparent success. When the loss of chyle is considerable, it sometimes causes emaciation and debility, as well as a craving appetite and urgent thirst. In such cases the patient must be impressed with the importance of rest, for exercise is found to aggravate the complaint.

The duration of the disease is often very long. Roberts cites two instances, in one of which it continued for twenty-eight years, and in the other for more than fifty years. If death occurs during its course the cause is generally some intercurrent malady, such as phthisis or Bright's disease.

PARASITIC HÆMATURIA.—As far back as 1812 Chanotin recorded the prevalence of an endemic form of hæmaturia in Mauritius; and subsequent writers afterwards noticed the occurrence of a similar affection in other hot climates. But nothing was made out with regard to its nature, until in 1851 Bilharz, being engaged with Griesinger in investigating the diseases of Egypt, discovered in certain of the veins of the abdominal viscera a trematode worm, to which he assigned the name of *Distoma hæmatobium* (p. 304). It was found that this parasite gave rise, in some cases, to more or less severe urinary symptoms; and Griesinger, in the 'Arch. d. Heilkunde' for 1854, suggested that it might probably be the cause of the endemic hæmaturia of warm countries. Afterwards it was shown to be generically distinct from the liver-fluke, and Cobbold proposed for it the name of *Bilharzia hæmatobia*, which is now generally adopted. In 1863 Dr John Harley detected the ova of the same entozoon in large numbers in the urine of a man who had become affected with hæmaturia at the Cape of Good Hope; and he has since shown (in papers in the 'Med.-Chir. Transactions') that the complaint prevails not only in the Cape Colony, but also along the coast of Natal.

The Bilharzia is a soft, white worm, which differs in shape in the two sexes. The male, half an inch in length, is flattened; but the hinder part of its body acquires a cylindrical appearance from its edges being thinned and folded inwards, so as to overlap one another, forming a hollow channel. The female, three quarters of an inch long, is slender and filiform. The ova are about $\frac{1}{170}''$ in length, and have a sharp projecting beak-like spine, placed usually at one end, but sometimes laterally. According to Dr Zancarol, of Alexandria ('Path. Trans.,' xxxiii), ova with lateral spines are found only when the seat of the parasite is in the veins of the intestine,

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whereas when it occcupies the veins of the urinary tract the spines are terminal; and this statement seems to correspond with previous observations. (See the drawings by Dr Cavafy, *ibid.*, p. 410.) It often happens that empty egg-shells are found in the interior of the human body, so that there can be no doubt that the ova may be hatched while in the tissues. But Dr Cobbold believed that the ova in urine never give exit to the embryos, which are often to be seen fully developed within them, and quite ready to escape. Such ova, however, when placed in water, become ruptured in a few minutes. The embryos are covered all over with cilia, and swim actively about.

The further steps in the life-history of the Bilharzia have not yet been ascertained; but the presumption is that the embryo finds in some fresh-water mollusc an "intermediate host," and there develops a Cercaria-form. Leuckart seems to think that the most probable way in which human beings become infected is by their swallowing encysted cercariæ in the substance of snails or minute slugs eaten accidentally with raw vegetables. Griesinger was of opinion that in Egypt the chief danger lay in the use of fish as food, though he also mentioned the drinking of Nile water as possibly affording the parasite a mode of entrance into the human body, in which case one must suppose that it is capable of developing directly from the free cercaria into the sexually mature condition. Dr Harley inclines to think that bathing in the rivers of Natal may bring the host into contact with the parasite, which may either make its way through the skin, or even pass straight into the bladder through the orifice of the urethra. Males are much more liable to suffer from the Bilharzia than females. The resulting hæmaturia commonly appears during boyhood, but not under five or six years old. It may, however, occur at any age, for one of Dr Harley's patients was a man of seventy-six.

Endemic hæmaturia appears to be vesical in its seat. At the end of micturition, the urine having been quite clear, the patient voids a small quantity—perhaps a teaspoonful or less—of dark blood. Or instead of the blood there may be soft shreds or filaments of mucus, by which the urethra is sometimes blocked for a few minutes; in these shreds the ova of the Bilharzia are found in large numbers. Sometimes a little pain is experienced in the loins or in the perinæum, especially after active exercise, which also increases the amount of blood. The health, as a rule, remains good, though a feeling of lassitude is sometimes complained of, and a more or less marked degree of anæmia may be developed. Boys in Natal appear to take no notice of the complaint, and about the age of puberty it commonly ceases. Long after this, however, Dr Harley has found that ova are still present in the urine, although the patient may imagine that he is quite free from his disorder; and (what is very remarkable) during early adult life he often begins to pass small *calculi*, in the centre of which the remains of ova may be detected. Indeed, in Egypt the affection commonly leads to the formation of stone in the bladder, which has to be removed by surgical operation.

Our knowledge of the lesions produced in the viscera by the Bilharzia appears still to be based almost entirely upon the investigations of Bilharz and Griesinger in Egypt; they found it present in no fewer than 117 out of 363 autopsies. However, it is fair to assume that the state of the bladder in persons who (like the boys of Natal) suffer comparatively little from this parasite, corresponds with only the slightest cases examined by the two German observers. They give a much more serious account of the ordinary symptoms of Bilharzia, which they describe as chronic cystitis

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The earliest morbid change in the bladder was the formation of swollen, hyperæmic, ecchymosed patches, varying in size up to that of a shilling, and generally coated with tough mucus or with a layer of soft greyish-yellow exudation; they were often limited to the posterior wall of the organ. But in many cases there were also thick deposits of a buttery or soft and granular material upon the surface of the mucous membrane, generally incrusted with urinary salts. Sometimes there were raised warty vegetations or fungus-like excrescences, due to a swollen infiltrated condition of the submucous tissue. Ova and empty shells of the Bilharzia were present in large numbers throughout the substance of the diseased tissues, and also in the mucous and other exudations upon the surface of the lining membrane of the bladder. Deeper down lay the parasites themselves, in smooth-walled spaces, which communicated with the veins and evidently were nothing else than altered blood-vessels. The alimentary canal of the worms was always full of blood-corpuscles, which no doubt served them for food.

How the ova effect their escape from the spaces in which the parent worms lie, and how they manage to pass through the substance of the mucous membrane, does not seem to have been made out. Is it possible that the sharp spine with which each of them is provided enables them to work their way gradually outwards, under the influence of the pressure to which they are subjected by the contraction of the muscular wall of the bladder? Cobbold supposed that the spine may act as a "holdfast," giving purchase to the embryo, and so aiding it in the violent efforts which it sometimes has to make before it can get out of its shell.

In a great many cases the ureters were affected as well as the bladder, and sometimes they suffered when it escaped; in exceptional instances the morbid process extended even to the pelvis of the kidney. The lesions in the ureter were identical with those in the bladder, but were far more serious in their effects, inasmuch as they obstructed its channel, leading to hydronephrosis or pyelitis, and at length to complete destruction of the renal cortex. The natural result of such an affection would be the death of the patient by gradual exhaustion, and this was often observed. But in most instances the direct cause of the fatal issue was either pneumonia or dysentery. In regard to the latter disease it was at first a question whether it might not also be dependent on the presence of the parasite. For, beside the veins of the urinary apparatus, the only other vessels in which the Bilharzia was found were the portal vein and its tributaries, including the radicles of the intestinal veins. Ova seemed, in fact, to be scattered through the coats of the bowel exactly as they were through those of the bladder; and the mucous membrane of the bowel showed similar patches of inflammation. But although it is not unlikely that symptoms identical with those of dysentery may sometimes be produced by the Bilharzia, Griesinger soon satisfied himself that the common endemic dysentery of Egypt could not be attributed to this cause. On the other hand, he was inclined to think that in some cases the morbid process set up by the Bilharzia in the urinary apparatus gave rise to acute and rapidly fatal "typhoid" symptoms. If this is the case, they are due perhaps to septicæmia. That the eggs may be carried to distant parts of the blood-stream is shown by a case in which a few empty shells were found in the interior of the heart; and they may act as mechanical or septic emboli.

In the *treatment* of endemic hæmaturia, Dr Harley thinks that he has

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obtained good results by daily injections into the bladder of from twenty to thirty grains of iodide of potassium dissolved in five ounces of warm water. Dr Guillemard, however, has recorded a case in which even a weaker solution than this set up acute cystitis; and Dr Cobbold had previously expressed a strong opinion that such procedures are more likely to do harm than good. Dr Harley has also prescribed, with some apparent advantage, draughts each containing ♏xv of oil of male fern and the same quantity of oil of turpentine. When there appears a tendency to the formation of uric acid calculi it should of course be counteracted by the administration of alkalies or of salts of the vegetable acids.

Malformation and Fixed Malposition of the Kidneys.—One kidney is sometimes congenitally absent or atrophied, usually the ureter remaining. The adrenal body never partakes in this or any other renal abnormality. In some persons the two kidneys have their lower ends *united in* the shape of a horseshoe. When the abdomen is thin and flaccid, this abnormality might easily lead to the diagnosis of an abdominal tumour, a morbid growth in the lumbar glands, or an aneurysm of the aorta. The occurrence of hydronephrosis or of pyelitis in a "horseshoe kidney" would also be very likely to be misinterpreted during life.

In a specimen in the museum of Guy's Hospital both kidneys are united into a single mass, which lay within the pelvis.

Occasionally one kidney—generally the left one—lies at a lower level in the abdomen than natural, over the sacro-iliac synchondrosis, or *even* within the pelvic cavity. The misplaced organ may be mistaken for a tumour, as in a case recorded by Mr Durham in the 'Guy's Hospital Reports' for 1860; or, in the female, it may interfere with parturition.

The persistence of a fœtal condition, called a lobulated kidney, does not in any way interfere with its functions.

Moveable and Floating Kidney.*—In some persons, instead of the kidney being fixed deeply in the loin, it may become loose, so as to move in various directions, downwards, forwards, or inwards. It can be *readily* felt through the abdominal wall, and can almost as readily be pushed back for the time into the natural position. At the same time the lumbar region of that side may be felt to be flattened or even hollowed, and pressure over the quadratus lumborum does not meet with the usual sense of resistance; percussion also may yield a tympanitic note.

This condition occurs much more often on the right side than on the left. Among ninety-one cases collected by Ebstein, in sixty-five the right kidney was moveable, in fourteen the left, and in twelve cases both were moveable. In Landau's 173 cases (many of which, however, were *identical* with Ebstein's) 152 were right, only twelve left, and nine double.

As a rule the place in which the kidney is felt is in the iliac fossa, or somewhere between this and its natural seat. Sometimes, however, it comes in contact with the front wall of the abdomen, and is far more freely moveable. One cannot conceive of a kidney as thus *floating* without its having peritoneum on both sides; and there is anatomical evidence that it is actually provided with a mesonephron. Possibly an imperfect degree of this condition may be a subsequent result of a moveable kidney making for itself

* *Synonyms.*—*Fr.* Reins mobiles, Reins flottants.—*Germ.* Bewegliche Niere, Dislokation der Niere, Wanderniere.

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such a covering by pushing forward the membrane that should naturally be in contact only with its anterior surface.

In vol. xxvii of the 'Pathological Transactions' may be found a case observed by Dr Goodhart, in which the right kidney, while lying on the spine and over the psoas muscle, had become completely turned over, so that its anterior surface looked backwards; both surfaces were covered by peritoneum. As a rule, however, a moveable kidney glides about behind the serous membrane, merely dragging this with it to a slight extent.

The *causes* of the affection appear to be complex. Sometimes, perhaps, the kidney is pushed downwards by an enlarged liver; in one case, in the *post-mortem* room of Guy's Hospital, the liver was cancerous. Cruveilhier thought that displacement of the right kidney was often an indirect result of tight lacing, through its altering the position and shape of the liver. But although a mobile kidney is far more frequent in women than in men, it is believed to occur chiefly in hospital patients who are less likely to wear tight stays; and it seems often to be due to a relaxed state of the abdominal walls in consequence of frequent child-bearing. Sometimes, perhaps, an injury may help in determining the displacement. Thus Ebstein cites a case from von Dusch, in which a woman who had borne eleven children, and whose abdomen was very loose, fell downstairs and struck her right side; soon afterwards she felt a tumour in the right hypochondrium. Becquet has maintained that a principal cause of mobility of the kidney is congestion and swelling of the organ, recurring at the menstrual periods. The objection to this view is that under normal circumstances such congestion is not known to take place. Roberts, however, in his article in 'Reynolds' System' (p. 645), mentions two cases in which a displaced kidney seemed to become larger and more sensitive to the touch each time that the catamenia appeared.

A moveable kidney is most often discovered in patients between the ages of twenty-five and forty, but Stoffen has observed it in children not more than nine years old. In 122 cases collected by Landau, 43 were between thirty and forty, and 79 between twenty and fifty. The same writer found in 97 cases 87 women and only 10 men. Other collectors give the numbers as 61 to 9, or as 82 to 14. The *right* kidney is much more often mobile than the left.

The *symptoms* caused by mobility of the kidney are sometimes slight, or even absent. Walther, of Dresden, some years ago examined a number of persons to decide this point, and detected the affection in many cases in which there were no symptoms whatever. But more frequently it causes a sense of weight and pressure in the abdomen, a feeling of dragging, as though something were loose in its cavity, or more or less intense pain, which may radiate in various directions—to the ribs, the shoulder, the epigastrium, or the external genitalia. Sometimes there is nausea or vomiting, increased by palpation. Such patients are liable to attacks of intense suffering, attended with faintness and collapse, during which the kidney becomes exceedingly tender. Active exercise, whether walking or riding, often brings on or aggravates the pain; some patients are prevented by it from standing upright, or from turning in bed, or lying on one side. There is often an apparently disproportionate degree of anxiety and of depression of spirits, amounting to hypochondriasis.

The *diagnosis* of a moveable kidney is sometimes easy, sometimes difficult and uncertain. We have in practice to distinguish it from fæcal accumula-

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tion, from an enlarged spleen, from a distended gall-bladder, from a mass of swollen glands, or from an ovarian cyst with a long pedicle. Ebstein mentions an instance in which a hydatid cyst in the mesentery was mistaken for it; and in a case which the author saw there were several such cysts, some of them oval in shape, and almost exactly the size of the kidney. The points to notice are the shape and size of the tumour, its smoothness and mobility, and the almost testicular sensation which seems to be produced by moderate pressure. The prone position is sometimes advantageously taken by the patient instead of the supine, in order to facilitate palpation. It must be borne in mind that a floating kidney is not exempt from hydronephrosis, cancer, and other local diseases—perhaps more liable to them; so that it may be a moveable renal tumour rather than a moveable kidney which we have to recognise. It should be added that the secretion of a floating kidney is in all respects normal.

The *treatment* of this affection consists in keeping the patient in bed during the attacks, and applying fomentations and poultices when there is severe pain. Leeches or hypodermic injections of morphia may be required. Afterwards an elastic abdominal belt should be applied, with a pad to maintain the kidney in its proper position. It is often, however, very difficult so to adjust an apparatus as to effect this object.

A suggestion made by Czerny is to inject alcohol into the connective tissue around the organ, while the patient is maintained in a recumbent position, the object being to induce local adhesions.

Hahn cut down upon the kidney by a lumbar incision, and fixed it in its proper place by sutures (*nephrorraphy*). Several cases are recorded by Ceccherelli ('Rivista Clinica,' April, 1884), quoted by Sir Spencer Wells.

In one only of the cases which have come under the writer's care has he advised an operation; in this one, at the patient's urgent request for relief, Mr Jacobson exposed the right kidney, which was brought into its proper place under chloroform, and fixed there with silver sutures. A good recovery followed, but the subsequent relief was less complete than we had hoped. The fact is that most patients with moveable kidney are more or less neurotic, apart from hypochondriacs, who only imagine they have the complaint; so that any treatment is apt to fail.

When, however, a moveable kidney is attended with unbearable suffering it is perhaps justifiable to extirpate the organ. According to Czerny ('Trans. Internat. Congress, 1881') this had then been done in twelve cases. Four of them ended fatally, three by peritonitis, one in consequence of the opposite kidney being diseased; the others were completely successful. No fewer than seven of these twelve cases of nephrectomy occurred in the practice of a single surgeon, Dr Martin, of Berlin.*

* Besides the chapters in the systematic works of Rayer (to whom the first clear account of this curious malady is due), Trousseau, Ebstein, and Roberts, the following monographs on the subject are interesting:—Rollett's 'Pathologie und Therapie der beweglichen Niere' (Erlangen, 1866), and Landau's 'Wanderniere der Frauen' (Berlin, 1881), translated for the New Sydenham Society by Dr Champneys. Valuable papers on the subject have been published in England by Dr Hare ('Med. Times and Gazette,' 1858), Mr Durham ('Guy's Hosp. Rep.,' 1860), by a Committee of the Pathological Society (vol. xxvii), and by Dr Newman ('Glasgow Medical Journal,' August, 1883), with a useful bibliography.

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DIABETES

"Si tibi nulla sitim finiret copia lymphæ,
Narrares medicis."
Hor. Epist., II, ii, 146.

History and definition: polyuria, hydruria, and glycosuria—Detection of glycose in the urine: the cupric, potash, fermentation, and other tests—Quantitative estimate by volumetric and other methods—The amount, specific gravity, and other characters of diabetic urine—Glycosuria.
Symptoms of diabetes—Course and complications: phthisis—coma—acetonæmia—Modes of death and post-mortem appearances.
Physiology of glycogenesis—Glycosuria and glycosæmia—Theories of diabetes—escape of sugar—over-formation of sugar—exalted glycogenetic function of the liver—diminished destruction of sugar—experimental and toxic glycosuria—incomplete conclusion.
General ætiology of diabetes—inheritance—distribution—age and sex—Prognosis—duration—Treatment: by diet—by drugs—treatment of complications.

It was known from early times that the quantity of urine daily passed may sometimes be greatly increased, without previous excess in drink or other obvious cause; and the Greeks named this disorder Diabetes.* We have seen that polyuria is a frequent sign of the most chronic form of Bright's disease, as well as of hydronephrosis, cystic degeneration, and probably all forms of renal atrophy. But, after death from diabetes, the kidneys are found to be healthy.

Although renal tissue is prone to compensatory hypertrophy, this is never a primary process. Nor do we ever meet with "a morbid excess of function:" all disease is impaired function. Polyuria is only hydruria, and the total amounts of urea and saline constituents excreted in the twenty-four hours are not increased and are sometimes diminished.

In the latter part of the seventeenth century the anatomist, Dr Thomas Willis, made the remarkable observation that in most cases of diabetes the urine contains sugar. The distinction he made between *diabetes mellitus* and *diabetes insipidus* (cf. p. 408) is no longer necessary, for the former is not only a far more frequent and important malady, but its clinical and pathological characters are very different. It, therefore, is now called diabetes without qualification.

It was early observed that patients affected with this disorder not only

* *Synonyms.*—Diabetes mellitus, as it was named by Willis, D. anglicus (cf. p. 408, note)—Saccharine diabetes.—*Fr.* Diabète sucré.—*Germ.* Zuckerharnruhr.

Diabetes (διαβήτης, a siphon) is used by Aretæus, Galen, Alexander of Trales, and Paulus Aegineta, but does not occur in the writings of Hippocrates, Celsus, or Cælius Aurelianus. The *locus classicus* is in the treatise of Aretæus on chronical diseases (lib. ii, cap. ii), θώνμα τὸ διαβήτεω πάθος, κ. τ. λ. The name is thus explained: "Atque hinc equidem adfectum *diabetem* vocatum esse arbitror, perinde ac si διαβήτην, *i. e. siphonem* hunc dixeris; quia humor in corpore non remanet, sed homine, tanquam canali quodam ad effluendum utitur."

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are continually thirsty and hungry, but grow thin, weak, and dried up, in spite of all they eat and drink. It was supposed that their flesh liquefied, and so ran through them; and we shall see that some of the sugar excreted in the worst cases is probably derived from the tissues.

The abundance of the urine is partly the result of the presence of sugar, partly has an independent, probably nervous, source. Hydruria occurs in diabetes insipidus and other conditions, but glycosuria is not met with otherwise except as a temporary effect of certain drugs, to be discussed hereafter.

Diabetes may, therefore, be defined as permanent glycosuria, with polyuria, emaciation, and thirst.

The pathology of this disease is still obscure, for it depends on a disturbance of the chemical processes called metabolism, the seat and course of which are still in great part secret. We know, however, that its most essential symptom is the passage of sugar in the urine; we know that this glycosuria depends on the presence of sugar in the blood, and this again upon some disturbance in the changes which the food undergoes after digestion and absorption. It is not a disease of the kidneys, of the urine, or of the blood, but is a derangement of the chemical labour of nutrition. Its place in a nosology is at present arbitrary, but as a matter of convenience it seems best to put it separate from other urinary disorders but near them, where it would naturally be looked for.

*Glycosuria.**—There is probably almost always a trace of sugar in the blood and in the urine under conditions of health, but it is far too little to give the reactions to be presently mentioned. In diabetes its presence is readily ascertained, and it is often present in very large amount. Chemically, diabetic sugar is indistinguishable from grape-sugar or *glycose*, also known as *dextrose*, from the fact of its turning a polarised ray of light which passes through it to the right hand. Fruit-sugar, *fructose* or *lævulose*, rotates to the left, while cane-sugar or *sucrose* and milk-sugar or *lactose* have no such rotatory power; when taken as food, sucrose is split up by a special ferment in the stomach into dextrose and lævulose, and lactose into dextrose and galactose. *Maltose*, the sugar formed in the process of making ale, is readily converted into dextrose. Ordinary starch or *granulose* yields by hydration under the influence of salivary or pancreatic digestion maltose, while a vegetable starch called *inulin*, found in the dahlia and some other plants, yields lævulose. Lastly, *glycogen*, the animal starch, readily yields dextrose by hydration.

We shall see that these chemical facts find a practical application in the treatment of diabetes.

Tests for glycosuria.—There are several chemical processes by which we can detect the presence of glycose in the urine, but only a few of them are used in practice.

(1) The chief of these is the *copper test*, which may be applied in several ways; but they are all based upon the fact that diabetic sugar or glycose possesses the property of rapidly reducing the oxide of copper to a hydrated suboxide at the temperature of 212° or lower. The oxide of copper is blue, and liquids containing it in solution have a deep blue colour; while the suboxide is orange-yellow, so that there is no difficulty in seeing whether reduction takes place or not.

Trommer's method consisted in adding a few drops of solution of cupric

* Glycosuria, *i. e.* the presence in the urine of glycose or glucose (from γλύκυς, sweet).

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sulphate to the urine and then an excess of liquor potassæ. The precipitate of the protoxide first thrown down is redissolved if sugar is present, and forms a deep blue clear solution, from which on heating the red hydrated suboxide of copper is precipitated. Liquor sodæ may be used instead of liquor potassæ. This is one of the surest methods.

Barreswil and Fehling introduced the method of keeping the copper in solution in excess of potash by means of tartrate of potash or potassio-tartrate of soda (soda tartarata), so as to have the test-liquid ready for use.

The following is Dr Pavy's modification of Fehling's formula :—640 grains of neutral tartrate of potass and 1280 grains of caustic potass are dissolved in ten ounces of distilled water, and 320 grains of sulphate of copper are dissolved in other ten ounces; the solution of sulphate of copper is then poured into that of the potass salt, and forms a clear blue liquid.

The way to use this test is as follows :—About a drachm of the liquid is placed in a test-tube, and heated until it begins to boil. A drop or two of the urine is then added, and if no change is observed, a further quantity of urine, until this equals that of the copper solution. The test-tube is then again heated until the liquid in it reaches the boiling-point. It is then allowed to cool, for there is no advantage, but rather the contrary, in continuing ebullition. If sugar is present, the liquid, before it cools, will deposit a yellow or red sediment of the suboxide of copper.

Dr Pavy has since devised an ammoniated cupric solution. The precipitated suboxide is kept in solution by ammonia, and the test becomes one of colour only.

There are several points which require comment in the application of the copper test.

In the first place, one reason for applying heat to the copper solution first, and not to the urine, is that when the solution has been kept for some time exposed to light it is sometimes found to undergo a slight reduction when boiled by itself. In a hospital ward, in assurance practice, or wherever the excellent rule is followed of testing the urine of every new patient, there is no real risk of this fallacy, particularly if the bottle in which the test solution is kept is of dark glass. If long kept in the light gradual reduction takes place even at an ordinary temperature. In any case, the addition of a fragment of caustic potass to the liquid, when it has become deteriorated by keeping, will render it as fit for use as ever.

Another reason for heating the liquid before adding the urine is that healthy urine, if it happens to contain a large proportion of lithates, possesses when boiling the property of decolourising the copper solution. The mixed liquids have then an amber-yellow colour, and often contain flocculi of phosphates—precipitated by the alkali of the test. This change is often regarded as proof of the presence of sugar; but this is a mistake. Indeed, when the copper solution has thus lost its blue colour, any sugar that might be present could no longer precipitate the suboxide; and this would often make the careless application of the test to diabetic urine misleading, were it not that such urine generally contains too small a proportion of the urinary solids to decolourise the copper solution.

The reasons for adding urine in the exact way prescribed are thus given by Sir Wm. Roberts, who has devoted much pains to the elaboration of the details of the process. In the production of the deposit of the suboxide it is necessary that the sugar should not be in great excess; for unaltered glycose

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has the property of dissolving the suboxide of copper. Hence, if urine containing a large proportion of sugar be added to the copper solution in considerable quantity, no precipitate will result, but merely an opaque yellow solution. But if only a drop or two of the saccharine urine is added, a deposit is produced of a characteristic orange-red colour.

On the other hand, when the urine contains only a small proportion of sugar, it must be added in larger quantity. Then, as soon as the boiling-point is reached, the liquid changes to an intense opaque yellowish green, and a bright yellow deposit is slowly formed.

Roberts has determined the exact limits of the test. He finds that one tenth of a grain per fluid ounce can be detected with certainty.*

It is well known that many substances beside sugar are capable of reducing the oxide of copper; but few of them are present in the urine.

One is *chloroform*, and hence reduction of the copper solution in the case of a patient who has recently undergone an operation under chloroform is no proof of glycosuria; the drug is, however, rapidly eliminated by the lungs as well as the kidneys. The same effect may be produced in the urine of patients who are taking chloral hydrate.† Leucin acts in the same way, but it never occurs in the urine except in cases of acute yellow atrophy of the liver (cf. p. 582).

The only other drug frequently taken which causes a reduction of Fehling's solution is *salicylic acid* and its compounds. This effect is not constant, but frequent enough to be worth remembering. The writer discovered it several years ago, and finds it the rule in patients taking salicylates for rheumatism. The reducing agent is probably salicyluric acid, in which form salicyl compounds are partly excreted by the kidneys.

Another fallacy in the detection of diabetes may be due to excess of *lithates* or of *kreatinin* in the urine. Either of these normal constituents of urine may, if in sufficient amount, produce a slight deposit of the suboxide.

(2) Although the copper test for sugar fulfils all practical requirements in accuracy and delicacy, there are other methods of detecting diabetes which also have their value. One is Moore's, or the *potash test*. It consists in boiling one or two drachms of the urine in a test-tube with half its bulk of liquor potassæ. The ebullition must be kept up for some little time; and as it goes on, the liquid becomes darker, passing through a series of colours which are almost exactly like those of different kinds of sherry wine, until it at last becomes brown. If nitric acid be added, an odour of caramel may be perceived. The colour deepens on cooling. This test is not a very delicate one; it does not succeed with urine containing a quantity of sugar less than a grain and a half or two grains in the ounce. Moreover, urine containing albumen, and generally all high-coloured urines, become somewhat darker when boiled with liquor potassæ, though they contain no sugar; and if the potash solution contains lead, as is often the case, albuminous urine may give a dark porter-brown colour, which has been mistaken for that which sugar produces. Moore's test is therefore chiefly valuable as a preliminary test. But it is well adapted to assurance practice; for if it gives a negative result, glycose is not present in serious amount.

* According to Dr Pavy, the yellow precipitate sometimes fails to be produced when there is albumen in the urine. In that case it may be well to boil and filter the urine before adding it to the copper solution: but the precaution is seldom needed.

† Dr Sherwin ('Boston Medical Journal,' November, 1886), quoted by Johnson.

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(3) A new test has been introduced by Dr George Johnson. It consists in the addition of liquor potassæ and a solution of *picric acid* (carbazotic acid or trinitrophenol) to the urine. Heat is then applied, and when the boiling-point is reached, the picric acid is turned into the deep claret-red picramic acid. Dr Johnson recommends half the quantity of solution of potash to be added to the urine (say a fluid drachm); forty minims of saturated solution of picric acid to be added to this, and the mixture to be made up to half a fluid ounce before boiling. The reaction is produced by kreatinin as well as glycose; but its chief practical drawback is that the change of colour is not so unmistakable as in the case of Moore's test, or of the cupric test in any of its modifications. The addition of caustic potash to a mixture of urine and picric acid always deepens the colour; when heated the tint becomes still darker, and it is far from easy to distinguish between this normal reaction and the production of a somewhat darker and redder tint or even turbidity when a small amount of sugar is present.

(4) Of other tests for glycose, that which depends on the reduction of *bismuth* by carbonate of soda and heat (Böttger's test) was formerly much used in Germany. Like the cupric and picric tests, this depends on deoxidation, and therefore is liable to the same fallacy that any other reducing agent than glycose may produce the same effect.* The two following tests are free from this objection.

(5) The *fermentation test* was first applied by Dobson, of Liverpool, in 1779. A small quantity of yeast, which must be first thoroughly washed, so as to remove any adhering starch or sugar, is added to the urine, and this is set aside in a warm place, with a control glass. When sugar is present, it is gradually decomposed into alcohol and carbon dioxide. The latter, if found in any quantity, is given off as a gas, and may be readily collected. For this purpose all that is needed is that the urine should be made to fill a test-tube, and that this should be then inverted in a saucer, and kept in position by a clamp. After some hours it will be found that the liquid has receded from the upper or closed end of the tube, in consequence of the accumulation of carbonic acid gas. Ethylic alcohol is also produced, and this proof of the vinous fermentation having taken place shows that not only a reducing agent but sugar is present.

(6) The determination of the presence and the amount of grape-sugar by the *polariscope* is scientifically interesting and is also exact; it depends on the property which glycose possesses of rotating the polarised ray of light to the right, whence its name *dextrose*. No other substance at all likely to be in the urine has this dextrogyrate property.

Quantitative analysis.—By the employment of any of these tests, it is easy to determine whether a patient is or is not suffering from glycosuria. But for prognosis and treatment it is desirable to obtain an estimate, not only of the proportion of sugar which the urine contains, but also of the total amount of sugar which is excreted by the kidneys in the twenty-four hours. With this object in view, all the urine which the patient passes must be carefully collected and measured every day, for some days in succession; for the proportionate amount of sugar contained in the urine is by no means uniform throughout the day. It is therefore from the mixed twenty-four hours' urine collected in a single vessel that a sample must be taken for analysis.

* The earliest chemical test was devised by Cruikshank, and published in Rollo's 'Treatise on Diabetes,' 1798. It consisted in converting the dextrose into oxalic acid by the oxidising action of nitric acid.

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There are two or three methods by which the amount of sugar present in a certain quantity of urine can be accurately determined.

(1) One consists in ascertaining how many minims of urine are required to reduce the whole of the oxide of copper in 100 minims of Pavy's copper solution. The solution is first measured by a pipette into a porcelain capsule. Into it is then dropped a fragment of caustic potass, of about twice the size of a pea, this having the effect of causing the reduced oxide afterwards to fall in a dense form, so that the colour of the remaining liquid can be more readily observed. The capsule is next heated by a spirit lamp until it boils steadily; a burette graduated to hold 100 minims, with subdivisions, is in the meanwhile charged with the urine, and this is now allowed to flow drop by drop into the boiling copper solution, which is kept constantly stirred with a glass rod. If sugar be present, the yellow or red oxide of copper gradually appears in greater quantity, but as soon as it is formed it settles, leaving the liquid still blue. At length, however, the blue colour is entirely removed; then the operation is suspended, and a glance at the burette shows how much urine has been used. The copper solution is of such a strength that exactly half a grain of sugar is required to discolourise 100 minims of it, so that there is half a grain of sugar in the quantity of urine that has been dropped from the burette. It is then easy to calculate the amount of sugar that must be contained in each ounce of urine or in the whole amount excreted daily. In his book on diabetes Dr Pavy gives a table by which the trouble of making this calculation may be saved. The process takes a very short time, and after a few trials anyone can learn to do it with sufficient accuracy. If the urine be highly charged with sugar, it is advisable to dilute it with from two to four parts of healthy urine or of water before employing it for analysis, of course making the necessary correction afterwards. The ammoniated cupric solution above mentioned (p. 565) may also be used with great ease for volumetric analysis ('Lancet,' March 4th, 1884), and the results are, for comparison, very accurate, although there is some reason to believe that the presence of ammonia may somewhat alter the reducing power of a solution of glycose.

(2) Another plan is to ferment the urine with a little yeast, and next day to take its specific gravity and to compare it with that of the same urine in its unfermented state. For each grain of sugar per fluid ounce, one degree of density is lost by the process of fermentation. Roberts says that this method yields very fairly accurate results, and its performance requires no technical skill; the only objection to it is the delay, the result being obtained only after the lapse of twenty-four hours.

(3) The *picric acid* test may also be used for a quantitative purpose by comparing the tint obtained with a standard solution made of a definite colour by mixing liquor ferri perchloridi with acetic acid and ammonia; and Dr Johnson has found it convenient as well as trustworthy in practice.

(4) The most accurate and convenient quantitative test of grape-sugar in solution is the *polariscope*, or saccharometer as it is called when so adapted in commerce, for estimating it in large quantity. The only possible drawback would seem to be that the presence of albumen or of oxybutyric acid, which are lævogyrate, might slightly diminish the amount of rotation due to the sugar. Lævulose is said not to appear in the urine.

The *amount of sugar* contained in the urine in diabetes varies from the smallest trace up to forty-eight grains in the ounce, and the total

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quantity of sugar excreted daily shows of course corresponding variations. Dr Pavy believes that this proportion is never exceeded, and that when it has been reached, any further increase in the quantity of sugar requiring to be excreted by the kidneys leads at once to an augmented flow of urine.

Quantity of urine.—A diabetic patient, instead of passing two or three pints of urine in the twenty-four hours, or less, often passes as much as fifteen; and Dr Pavy has himself seen a case in which thirty-two pints were collected and measured in one day. Much larger quantities are recorded, some almost incredible.

Specific gravity.—Since syrup is heavier than healthy urine, it follows that in diabetes the specific gravity of the urine is higher than normal. Instead of being between 1010 or 1015 and 1025, it is from 1030 to 1040 or 1045. Some writers have said that it may reach 1060 or 1070; but according to Dr Pavy, the maximum is but little above 1050. From the specific gravity of the urine in a case of diabetes one can form a rough estimate of the proportionate quantity of sugar it contains; but the relations between them are not constant. If, however, a diabetic patient's urine is diminishing in quantity and also in specific gravity, we may be sure that the amount of sugar is also diminishing.

Some other characters of the urine in diabetes remain to be mentioned. It is generally pale, and the more so the greater the quantity that is passed. When this is very large, the urine may look just like water after standing, though often somewhat opalescent from the presence of torula. Again, it deposits no urates after it has cooled. This fact has a practical value, because it sometimes enables us to form an opinion as to when the disease began. Dr Prout used to ask his patients how long it was since the urine would become thick on cooling; and if such turbidity of the urine had previously been frequently observed, he dated the commencement of the diabetes from the time when it ceased to occur. As diabetic urine dries, it leaves a white crystalline deposit.

Urine containing sugar has a sweet taste—a fact which was first discovered by Willis about the year 1674, when chemical methods of detecting sugar were not known. It is said that flies and wasps are attracted to vessels containing diabetic urine. It has a peculiar odour, which was compared by Dr Prout to that of sweet hay or milk, by Sir Thomas Watson to the faint smell of certain apples, or of an apple-loft. The *Torula cerevisiæ*, or yeast plant, forms in diabetic urine when it is left freely exposed to the air in a warm place; and the sporules of this fungus may be readily detected with the microscope. Formerly its development was supposed to be a proof that a specimen of urine contained sugar; but even in healthy urine sporules may be found, which are, indeed, said to be *Penicillium glaucum*, but which are undistinguishable from those of the torula. Diabetic urine undergoes ammoniacal decomposition more quickly than that of health. *Urea* is not really deficient, though less than normal is found in any given specimen (p. 571).

When a patient suffering from diabetes is attacked by any intercurrent febrile disease, the urine often, but not always, becomes for the time free from sugar. This is a point of theoretical interest, as tending to the view that the disease depends upon a perversion of the glycogenic function of the liver; for it has been shown that when fever arises in healthy subjects glycogen disappears from the liver.

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Glycosuria without diabetes.—If we make it a rule to examine the urine of every patient for sugar as well as for albumen, we shall not infrequently find slight reduction of copper in otherwise normal urine, and in persons free from the symptoms of diabetes. In most cases this is due to one of the causes above described as fallacies (p. 566), but occasionally glycose is undoubtedly present, though in small amount. Minute quantities occur normally in blood-serum, and from its crystalline character and diffusibility one would expect it to appear in minute quantities in normal urine: some authorities deny its presence even in traces; but Brücke and Bence Jones long ago asserted its presence, and Pavy has brought positive evidence to the same effect ('Croonian Lectures,' p. 10). Hence "physiological glycosuria" has been supposed to exist, like physiological albuminuria. It has also been asserted that "dietetic glycosuria" can be produced by eating too much sugar; and "dyspeptic glycosuria" has often been described, chiefly in gouty persons at or beyond middle life. This was taught by Sir Henry Marsh and by Graves, by Trousseau, who called a form of occasional glycosuria *glycosurie alternante chez les goutteux*, and by many living physicians, including Dr George Johnson.

But at present it is safest (as in the case of albumen) to regard the presence in the urine of sugar, if in quantities appreciable by the cupric test without concentration or extraction, as pathological; indeed, excluding the fallacies produced by excess of uric acid and otherwise, which are mentioned above, the conclusion is probably also scientifically accurate.

If small quantities of sugar sometimes appear, and vanish again under dietetic treatment, it is not unlikely that these are incipient cases of what would develop into the slighter forms of diabetes so often met with in persons who are past middle life.

However this may be, there are no other diseases than diabetes which produce glycosuria as albuminuria is produced by cardiac lesions, fevers, and the other morbid conditions enumerated in the chapter on Bright's disease. The only partial and occasional exceptions to this are (1) temporary glycosuria from inhalation of chlororform and ether, said to occur apart from the presence of chloroform itself as a reducing agent in the urine; (2) similar "cyanotic" glycosuria after paroxysms of whooping-cough, asthma, or epilepsy; (3) during pregnancy; (4) after injuries to the head. The last group is of great physiological interest, as we shall presently find (p. 585); but, like the "artificial diabetes" which it resembles, the glycosuria is only temporary. Traces of sugar have sometimes been recorded in cases of fever and of dyspepsia, but they are transitory, and perhaps the reduction observed has not been always due to glycose. (5) Another physiologically interesting form of glycosuria is that observed by von Mering, as the result of feeding dogs on *phlorizin*, a glucoside prepared from the bark of the roots of apple and cherry trees. Poisoning by *curare* has the same effect in animals.

In another class of cases the ordinary symptoms of diabetes are absent, although the presence of sugar in the urine is persistent. But there is no definite boundary-line between such cases and those in which the most marked symptoms are present; indeed, the same patient may come in turn under the one and under the other category.

The distinction, therefore, between mild and severe cases of diabetes, based on the presence or absence of symptoms, is not to be regarded as a natural or safe division; for, as a German writer who adopts it remarks, "fatal complications are not rare in slight cases." The same treatment is

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beneficial in both kinds of cases, and the same name should be given to both; in fact, persistent glycosuria is diabetes.

Symptoms.—The early signs of well-marked diabetes are commonly as follows:—A man finds that his strength is failing him, he knows not why. His appetite is excellent, and, indeed, larger than ever; yet he loses flesh as well as muscular power. Then he notices that he passes an unusually large quantity of water, and that he is always thirsty. His urine is tested, and it is found to contain sugar.

The *muscular weakness* is often extreme. It is by no means to be regarded as a mere result of the wasting of the muscles which generally accompanies the disease. In a series of experiments which Dr Pavy made with various kinds of diet on a man affected with diabetes he found that as soon as the patient was put upon food which caused him to pass an excessive quantity of sugar, he complained that he had no life or energy in him. Another patient when admitted was so weak that he could not stand alone; after about three weeks under treatment he had gained strength, so that he ran to the end of the ward and back to show what he could do.

Loss of virility is another frequent effect of the disease, and in women the suppression of the catamenia. Natural vigour of character may be replaced by feebleness, moral and intellectual. The knee-jerk is sometimes absent, and neuralgia is not an infrequent complaint.

Thirst is another of the earliest and most persistent symptoms of diabetes. The patient generally drinks from eight to twelve pints a day, but sometimes as much as twenty-five or more. Yet even this does not satisfy the craving. The mouth and fauces are also the seat of a sensation of dryness, which causes great discomfort. Dr Pavy says that the way in which the patient keeps rolling the tongue about in the mouth, and the sound which it produces by sticking to the palate from time to time, may be recognised as signs of the disease. The tongue is often "raw," red and unnaturally clean, and sometimes it is fissured. Occasionally a sensation is experienced of a sweet taste in the mouth.

Increased *appetite* for solid food is by no means so constant a symptom as thirst. Sometimes the appetite is enormous, but in the later stages of diabetes there may be a loathing of all kinds of food. The teeth generally become carious, and the gums swollen, loose, and inclined to bleed. Very often the patient complains of a peculiar and distressing sensation of hollowness at the pit of the stomach. Occasionally vomiting and symptoms of dyspepsia are present, but, as a rule, the digestive powers of persons suffering from diabetes are remarkably good.

Owing to the large amount of nitrogenous food taken, *urea* is formed in abundance. It was formerly supposed that the urine of diabetic patients contains much less urea than in health; but it has been shown that this was a mistake, for although each fluid ounce is poor in urea, there are so many ounces passed that the total amount of urea voided in the twenty-four hours is as great as, and often greater than in health. We shall presently see that, at least in some cases of diabetes, a part of the sugar is formed from the proteids of the food, or rather from the peptones into which they pass before they are absorbed from the alimentary canal; and it is probable that these peptones, under such circumstances, split up so as to produce two series of substances, of which the one has its final term in sugar and the other in urea. A part of the urea excreted is no doubt

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derived, not from food, but from the nitrogenous tissues, as in health; but Dr Dickinson has made calculations, allowing a certain proportion for tissue-urea, and he found that in at least one patient although the actual daily quantities of urea and sugar varied greatly, yet the proportion of surplus urea to sugar was almost constantly as 1 to 6·1. Dr Ringer many years ago arrived at the conclusion that during abstinence, or under a non-nitrogenous diet, the *total* amount of urea and that of sugar excreted, whether by different patients or by the same patient at different times, had a constant ratio of about 1 to 2. His results involve the supposition that the disintegration of the nitrogenous tissues gives rise in diabetes to the formation of sugar as well as of urea, which is as yet only probable.

A further question concerns the amount of *uric* (or lithic) *acid* which is excreted by diabetic patients. This also was supposed at one time to be much less than normal; but Dr Dickinson thinks that the deficiency is probably apparent rather than real. He has seen a copious deposit of crystals of lithic acid in urine, of which more than fourteen pints were passed daily. Still, in comparison with urea, it appears to be clear that but little uric acid is passed in diabetes.

Dr Ringer found that in diabetes the ingestion of *non*-nitrogenous food was followed by a marked increase of urea, as well as of sugar in the urine, which may perhaps be correlated with Murchison's doctrine that lithæmia, a state in which nitrogenous principles fail to be fully oxidised, is especially apt to be induced by the ingestion of non-nitrogenous matters in excess, or in such quantities that the digestive organs become overloaded.

Other facts appear to show an antagonism between lithæmia and diabetes. Sir Charles Scudamore long ago showed that whereas gout was less common in Scotland than in England, the relative frequency of diabetes in the two populations was reversed. The same applies also to Ireland. One of Dr Pavy's patients, who had before been a martyr to dyspepsia, said that his digestive troubles ended as soon as diabetes appeared. It has also been noticed that gout has ceased to return in persons who have become diabetic; but this is less conclusive, because it might be merely from increased flow of urine preventing accumulation of lithates in the blood (cf. p. 585).

These facts, indicating that lithæmia and diabetes are inversely correlated, lend support to the view that both these conditions depend on disorder of the hepatic functions.

The *bowels* are usually constipated in diabetes, the fæces being dry and hard. But diarrhœa sometimes occurs, and it may lead to a state of prostration which is the immediate cause of death.

Diabetic patients often complain of chilliness; the *temperature* of the body is as a rule lowered, bearing some proportion to the severity of the disease. In one very severe case recorded by Dr Dickinson it varied from 93·6° to 94·8°, and when fatal pneumonia set in, the thermometer only rose to 97·8°. In another case the same disease was attended with a temperature of 103·2°. We shall presently see that in many cases the approach of death is preceded by a fall of temperature.

The *skin* is usually dry and harsh. The cuticle of the palm is stiff, and the furrows have a peculiar white, mealy appearance. Generally speaking there is no sensible perspiration through the whole course of the disease, but sometimes profuse sweats occur. The latter symptom is difficult of explanation. The fact that the subcutaneous tissue sometimes becomes œdematous may possibly be the result of anæmia. When it occurs, the

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presence of albumen in the urine should be sought for, as Bright's disease is a not infrequent complication of diabetes. But there is often œdema of the ankles, even when the kidneys are healthy.

One of the most marked symptoms of diabetes is *emaciation*. The features acquire a peculiar drawn, pinched look, by which the disease may often be recognised. Sometimes, however, the patient remains well nourished. Roberts says that one of his patients weighed more than fifteen stone, when he had been passing twelve pints of highly saccharine urine for some months; and that one of Prout's patients weighed twenty-three stone.

Many diabetics are fat with smooth skin and pink faces, an appearance which contrasts with the wasted and haggard aspect of the severer form and the latter stages of the disease.

Another symptom of diabetes is that the patient's breath has a peculiar *sweet smell*, like that of the urine but less distinct. Dr Dickinson says that this is connected with a constipated state of the bowels, and he appears to take it as an indication of the near approach of the coma which is often the immediate cause of death. It can be detected by some people even at a little distance; it is said that the late Dr B. G. Babington, when he came down to Guy's Hospital to take in patients on a Wednesday morning, could tell at once whether there was a case of diabetes among the applicants by the characteristic odour.

Latency.—It is rare for sugar to be discovered in the urine of persons who believe themselves to be perfectly well. Of this, however, Bence Jones met with an instance. A gentleman noticed some little white bodies in his urine, and consequently had it tested. They proved to consist of epithelium from the bladder, but there was sugar in the urine, and this continued to be the case whenever it was examined afterwards. He was a stout man, and remained in good health. Another case, mentioned by Griesinger, is that of a medical student, whose urine was saccharine during the whole of one winter, while he was residing in a moist and foggy locality in Switzerland. He never had a single symptom of diabetes, and both before and afterwards the urine was often tested and found normal.

Instances such as these, however, appear to be of most exceptional occurrence. As a rule, when sugar is found in the urine without the ordinary symptoms of diabetes, the patient is far from being well, and complains of symptoms which, though not peculiar to diabetes, so often accompany it that no cautious practitioner ever meets with them without testing the urine for sugar. The affections in question are perhaps best described as "complications" rather than as ordinary symptoms of diabetes.

Complications.—One of these disorders is *pruritus vulvæ* in women, often attended with a lichenous or eczematous eruption. That such complaints are often due to diabetes is mentioned by most writers. A lady aged fifty-two consulted the author on account of an eruption affecting the vulva, and attended with severe pruritus. The parts were reddened, but dry; the disease, in fact, resembled a chronic case of lichen. Medicine failed; but next time her urine was found to contain ten grains of sugar to the ounce. In a few weeks, under proper treatment, her troublesome complaint was removed; and this although the urine still contained a small quantity of sugar. Most writers say that this pruriginous dermatitis is set up by the local irritation of the sugar in the urine; and they remark that the orifice of the male urethra and the glans penis are sometimes excoriated under the same

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circumstances. But pruritus of the vulva is comparatively rare in ordinary cases of diabetes.

Some peculiar forms of papular dermatitis have been occasionally observed in diabetes; the first case was recorded by Addison and Gull among their cases of xanthelasma ('Guy's Hosp. Rep.,' 1851, p. 268); others have been called lichen. It is not certain that they are more than a coincidence; but this will be discussed under the section on xanthelasma.

Again, *carbuncles* and *boils* are apt to arise in patients whose urine is saccharine. Prout stated that in his experience diabetes always accompanied carbuncles and malignant boils or abscesses. But it has since been shown that this is too sweeping a statement. The importance of remembering the liability of diabetic patients to carbuncular affections is well shown by a case of Sir William Gull's, which is related by Dr Pavy. A medical man was suffering from cerebral symptoms, for which it was suggested that he should apply a blister to the nape of the neck. His urine contained sugar, and on this account he was cautioned against doing so. However, the blister was employed, and a large carbuncle soon developed itself, which proved fatal.

Prout also spoke of glycosuria as a temporary accompaniment of affections of this kind. A patient of his, middle-aged, told him that for a long period he had been subject, at intervals of a year or two, to boils and carbuncles, and that during such attacks he always passed a quantity of saccharine urine, whereas at other times the secretion was natural. Later writers also have given cases in which patients have had sugar in the urine only while they suffered from carbuncles or boils.

Gangrene of one of the lower limbs, resembling senile gangrene, is also sometimes associated with a saccharine state of the urine. This is a fact which has been especially insisted upon by the surgeons of Dublin; and several cases of the kind have occurred in Guy's Hospital.

Defective accommodation is another symptom which is common in diabetic patients, due to impairment of the power of the ciliary muscle; in such cases Dr Pavy has found that the application of Calabar bean to the conjunctiva is very beneficial.

Sometimes sight is affected by the formation of *cataract*. Many years ago Mr France published several cases of this kind in the Ophthalmic and the Guy's Hospital Reports for 1859 and 1860 respectively.* Diabetic cataract has acquired special interest from experiments made by Dr Weir Mitchell, in which frogs were immersed in a saccharine solution, with the result that the crystalline lens became opaque. This, however, seems to be only a curious coincidence, for the opacity of the lens in the frogs is due to exosmosis and disappears in distilled water, whereas the cataract of diabetes is due to the same clinical change as in other cases.

Other occasional causes of impaired sight in diabetes are atrophy of the optic discs, or retinitis, or opaque patches in the choroid.

Phthisis.—The most important complication of diabetes, and a frequent cause of death, is a destructive disease of one or both lungs, which is very like the more acute forms of phthisis. The fatal cases of diabetes in Guy's Hospital between 1860 and 1874 were forty in number, and in seventeen the immediate cause of death was phthisis. Dr Addison taught, and Dr Wilks afterwards maintained, that the pulmonary disease in cases of this kind is

* Lecorché's paper on the same subject appeared in the 'Archives générales de médecine' in May, 1861, but cases had been noticed before by von Gräfe.

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not tubercular phthisis, but rather a form of chronic pneumonia. Dr Fagge also was disposed to support Addison's view; for he found that in twelve among seventeen cases there was nothing that could fairly be identified as tubercle in the lungs, and in all of these it was either expressly stated that the larynx and intestines presented no tubercular ulceration, or, at least, no mention is made of these organs. On the other hand, it is said that in four cases the lungs contained grey or miliary tubercles; and in two of them, as well as in the other case, the *intestines* showed tuberculous ulcers. Such a proportion of cases without tubercles in the larynx and intestines is very different from what occurs in any form of phthisis, apart from diabetes; and so far it supports the opinion that the pulmonary affection in this disease is not of tubercular origin.

Sometimes the development of the disease in the lungs is perhaps too rapid for the formation of tubercles in other parts of the body; in several of the above cases the earliest pulmonary symptoms appeared only from two to five months before the patient's death. It is true that in one case the symptoms of disease of the lungs preceded those of diabetes, and began fourteen months before death, yet the intestines were free from tubercle. This, however, was a case of phthisis afterwards complicated by diabetes, and only proves that (non-diabetic) phthisis is sometimes confined to the lungs.

The pulmonary affection generally spreads through the lungs from apex to base, like ordinary phthisis; it very rapidly leads to the formation of one or more large cavities, by which the whole of an upper lobe may be excavated, and which have usually very thin ragged walls. It is generally much more advanced in one lung than in the other, but sometimes attacks both organs pretty equally. A proof of the rapidity with which it advances to a fatal termination is afforded by the fact that in only one of the twenty-eight cases in which death was not the direct result of this form of pulmonary disease did the lungs present any trace of it.

Tubercle-bacilli have been repeatedly found in diabetic phthisis; and the locality of the affection in both apices, the excavation, and the frequent presence of caseous material in the lungs and other organs, render it almost certain that the chronic affection of the lungs in diabetes is true phthisis. It runs a rapid course, and is seldom marked by hæmoptysis or high fever.

Another frequent cause of death in diabetes is ordinary lobar or "croupous" *pneumonia*. This was present in ten out of the forty cases collected by the author. In four of them, the hepatised parts were more or less distinctly passing into a gangrenous state. The onset of pulmonary symptoms in these cases was generally well marked, and occurred two or three days before death.

The present writer was indebted to Mr Kelbe's help in the analysis of the anatomical reports of seventeen cases of diabetes which consecutively proved fatal in Guy's Hospital by inflammation of the lungs between 1881 and 1887. The disease affected one or both apices in ten cases; with vomicæ but no other sign of tubercle in four, with miliary tubercles in the lungs or elsewhere in the other six. In two cases there was acute hepatisation of the base without any sign of phthisis. In the remaining five cases there was less acute pneumonic consolidation not affecting the apices, described as caseous in one case and necrotic in three, in one of which it was caused by the presence of particles of food.

Diabetic coma.—In six of the forty fatal cases before mentioned, the imme-

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diate cause of death was the supervention of *cerebral symptoms*. These generally began with drowsiness, and in a few hours passed into coma. Once or twice there was more or less well-marked delirium, or even convulsions; the pulse was often very feeble, and the temperature low. Indeed, in cases of this kind the state of the patient is often one of collapse quite as much as of coma.

This "diabetic coma" has been ascribed to the presence of *acetone* in the blood. But acetonæmia is not always present in diabetes, nor is it always, when present, accompanied by cerebral symptoms.* Moreover if, as is probable, it is derived from nitrogenous metabolism, it could not be present in sufficient amount to act as an intoxicant dose.

During diabetic coma the following abnormal constituents have been discovered in the patient's urine:

(1) Acetone (C_3H_6O), dimethyl-ketone ($CO,2CH_3$);
(2) Aceto-acetic acid ($C_4H_6O_3$), which yields acetone and carbon dioxide
(3) Crotonic acid ($C_4H_6O_2$);
(4) Oxybutyric acid ($C_4H_8O_3$).

The acetone which imparts its odour to the breath, and which gives a crimson colour to the urine when treated with perchloride of iron, is the product of aceto-acetic acid, which may be extracted from the urine by ether after acidulation with dilute sulphuric acid.

Dr Dreschfeld, in his Bradshaw lecture before the College of Physicians ('Lancet,' August, 1886), recognises three types of diabetic coma: †

1. Diabetic collapse, with coldness, lividity, and subnormal temperature. The pulse is rapid, and there is little dyspnœa. The patients are usually elderly, stout, and long diabetic. The attack comes on after fatigue (as in Prout's cases), and proves fatal rapidly—within twenty-four hours as a rule. The heart is often found fatty, but no acetone or other abnormal constituent but glycose is found in the urine.

2. A rare form, closely resembling drunkenness, with staggering gait, incoherent speech, and disturbed mental faculties. Acetone is often present in the urine and the breath, and sometimes alcohol.

3. The most frequent form, with muscular weakness, drowsiness, rapid breathing, and at last coma. There is the acetone smell in the breath and urine, and not only aceto-acetic but also crotonic and oxybutyric acids can be demonstrated in the urine.

As described by Prout, and afterwards by Kussmaul, the condition of coma (as distinct from collapse) agrees closely with the third of these varieties. "Breathlessness without dyspnœa," occasional convulsions, and a subnormal temperature are its leading features. It occurs mostly in young patients, and early in the disease. It, like collapse, often follows fatigue.

In addition to the six cases mentioned, in which death was preceded by more or less marked cerebral symptoms, there were two in which it was quite sudden, and probably these should be placed in the same category with the last. There was this peculiarity with regard to most of these eight cases, that the fatal symptoms developed themselves very shortly after the admission of the patient into the hospital. In five death took place within five days from the date of admission, and in three it occurred either on the day of admission or on the following day. The cause of the

* See an excellent account of Acetonæmia and Lipæmia in Diabetes, in Dr Gamgee's 'Physiological Chemistry,' vol. i, pp. 168—172.

† These are not identical with Frerichs' three groups (*loc. cit.*, pp. 81, 82, 105).

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sudden fatal termination was, no doubt, the fatigue and excitement which the patients underwent in coming to the hospital. Exactly the same thing was noticed long ago by Prout, who says that four of his private patients sank almost immediately after coming to London from the country to consult him, and one of them was very near dying in Dr Prout's own house.

Dr Pavy has observed that those cases of diabetes in which the disease has been kept under control by treatment are particularly apt to end at last in convulsions, collapse, or coma; whereas when the disease is allowed to run on unchecked, the chances are in favour of the supervention of pulmonary disease.

Since acetonæmia is incapable of explaining diabetic coma, it has been proposed to account for it by oxybutyric, and perhaps other acids, diminishing the normal alkalinity of the blood. The phenomena would then be comparable to those produced by giving dilute mineral acids in large quantity to dogs (Walter and Schmiedeberg). This would prevent the blood dissolving carbon dioxide, which would, therefore, accumulate in the brain and other tissues.

Experience has shown that great caution is necessary in assigning the cause of death in diabetes, unless an autopsy is made. Two diabetic patients in Guy's Hospital died with cerebral symptoms, but in each case death was found to be due to local inflammation; in one the pelves of both kidneys were dilated and inflamed, with commencing renal suppuration; and in the other there was extensive hepatisation of the left lung.*

As above stated, of the forty fatal cases at Guy's Hospital between 1860 and 1874, seventeen patients died of phthisis, ten of acute pneumonia, and six of coma ('Reports,' 1875); in thirty-six reported by Dr Frederick Taylor from the same hospital between 1875 and 1882 (*ibid.*, 1881, p. 152, note; and 'Path. Trans.,' 1883, p. 371) twenty-eight deaths were from coma, three others probably from uræmic coma, and eleven from phthisis and pneumonia. Dr Stephen Mackenzie reported, among thirty-seven fatal cases of diabetes at the London Hospital (1874–83), nineteen deaths from coma, five of these and ten others with phthisis, and eight from accidental causes.

Morbid anatomy.—The viscera of those who have died of diabetes have a decidedly sweet smell, resembling that observable during life in the urine and breath; and it may be noted that in one case Dr Wilks found that this odour was still observable, although the patient died of typhus, and his urine (which also retained the sweet smell) had been free from sugar some days before death.

In some cases of diabetes *the blood* has been found creamy from the presence of fatty molecules, and fat-embolism has been found in the lungs (Sanders and Hamilton, 'Edin. Med. Journ.,' July, 1879). But *lipæmia* (as this condition of the blood has been called) is certainly not constant, and is

* The inflammation of the kidneys in the former of these two cases was so exactly like what occurs in cases of stricture and other diseases of the urethra or bladder that these parts were very carefully examined. The urethra was perfectly healthy; the *bladder*, on the other hand, was greatly hypertrophied. This led the author to consider whether the increased thickness of the coats of this viscus could be due to the augmented work it had had to perform in consequence of the over-secretion of urine. In the second case, therefore, we looked at the bladder with much interest, and found that it also was markedly hypertrophied, and that its mucous coat protruded between the muscular fasciculi, so as to form numerous sacculi. Probably hypertrophy of the bladder may be found to be frequently present in diabetes.—C. H. F., 1882.

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probably rare. It was first noticed by Dr Rollo in 1778, and afterwards by Dr Babington the younger ('Cycl. Anat. and Phys.,' i, 422). Dr Pavy regards it as a physiological effect of the abundant food that is taken.

The *liver* is sometimes large, and Wilks describes it as having a uniform fleshy appearance.

The *pancreas* has frequently been found atrophied or otherwise diseased—a fact which may possibly prove of pathological interest, since von Mering reports permanent glycosuria in dogs which have survived removal of the pancreas.

The *kidneys* are not infrequently large, soft, and fatty; and they are occasionally affected with chronic tubal nephritis or some other form of morbus Brightii, or, still more rarely, with suppurative inflammation.

The *brain* is firm and healthy: in two of our cases dilated perivascular spaces were seen. Dr Dickinson has described certain cribriform spaces mostly around the blood-vessels of the bulb and pons, sometimes with blood-corpuscles. These dilated perivascular spaces do not, however, appear to be characteristic of diabetes, and are probably not even present during life, as seems proved by the observations of Drs Taylor and Goodhart (see 'Guy's Hosp. Rep.' for 1877, and 'Path. Trans.,' vol. xxxiv, pp. 328—396, and the report, p. 397). Dr Hale White (*ibid.*, vol. xxxvi) has also shown that the pigmentation, induration, and other changes of the *semilunar ganglia* reported in diabetes are present in other cases ('Journ of Phys.,' vol. viii, p. 70).

In 44 fatal cases of diabetes at Guy's Hospital, reported by Dr H. J. Campbell ('Guy's Hosp. Reports,' vol. xliv, p. 207), the *kidneys* were usually large, sometimes "coarse" in aspect, and occasionally fatty. The presence of glycogen in the looped tubes, as asserted by Frerichs and Ehrlich, was not clearly demonstrable. Sections stained with iodine, logwood and eosine, silver or osmic acid, showed very often the necrotic degeneration of the epithelium described by Ziegler. The *bladder* was hypertrophied in 13 cases (see foot-note on last page).

The *lungs* were affected in 23 cases; in 18 there was ordinary phthisis of the apex, in 1 gangrene, and in 4 acute œdema or pneumonia.*

We must admit that the results of morbid anatomy do not lend support to the theory of diabetes which places the primary lesions in the central nervous system, the "sympathetic," or in the liver. The morbid changes found are results, not causes of the disease.

* In a series of autopsies on diabetic patients collected by Dr Windle ('Dublin Medical Journal,' September, 1883) the *liver* was reported normal in 84 cases, enlarged in 57, congested in 40, fatty in 15, and tubercular in 2.

The *kidneys* were normal in 115, enlarged in 88, fatty in 35, affected with tubal nephritis in 6, cirrhotic in 10, and lardaceous in only a single case. The *bladder* was normal in 20, and hypertrophied in 13 cases.

The *lungs* were normal in 81 cases, and congested or œdematous in 37 more, making 118 in which they were free from organic disease. In 136 they were phthisical ("tubercular" in 109, "pneumonic" in 27), to which 17 cases with cheesy masses, 12 with vomicæ, 3 with caseous bronchial glands, and 8 with miliary tubercle may be added, making a total of 178. In 24 cases there was acute lobar or catarrhal pneumonia, and in 3 gangrene, while in another 3 fat-emboli were discovered. The *heart* was normal in 70, large in 4, small in 9. The *brain* was normal in 91, and perivascular changes were discovered in 19. The *cord* was normal in 37, and perivascular changes were found in 11. The sympathetic ganglia were normal in 10 cases, and "cirrhotic" in 5.

Valuable information on the morbid anatomy of diabetes will also be found in Frerichs' treatise, where he describes and tabulates (pp. 134—183) 55 cases under his own care; in Dr Saundby's "Bradshawe Lecture" ('Brit. Med. Journ.,' Aug. 23rd, 1890), reprinted in his recent monograph; and in Dr Finlay's report of 20 cases from the Middlesex Hospital ('Path. Trans.,' xxxiv, pp. 336, 341).

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Physiology of glycose.—Although it was known in 1674 that the urine of diabetes contained sugar, this was supposed to be a purely morbid condition, nor was the existence of any carbohydrate in the healthy tissues suspected until in 1848 Claude Bernard published the discovery that a substance which he called glycogen, closely resembling starch, is constantly present in the liver of man and other animals, and that abundance of sugar may be obtained from the liver and the hepatic vein after death. He also stated that there was a much larger amount of this sugar, which had all the properties of dextrose, in the hepatic than in the portal and other veins. Next, he ascertained that though the glycogen of the liver is greatly diminished by starvation, yet that its presence does not wholly depend upon that of carbohydrates in the food. Lastly, he showed that when the liver of a recently killed animal was washed out by the injection of a stream of water through the portal veins, so as to be deprived of its sugar, it reappeared in abundance after a few hours.

Glycogen was isolated as a white powder by Bernard in 1857, and its composition and reactions place it undoubtedly in close relation to, though differing from, vegetable starch.

Bernard's theory was that the sugar which is absorbed from the stomach and intestines in the form of glucose, as the product of all digestible carbohydrates—whether vegetable starch from bread or animal starch (glycogen) from meat, or cane-sugar, fruit-sugar, malt-sugar, or milk-sugar—is conveyed by the portal vein to the liver, and is there changed by dehydration into glycogen, and stored in this indiffusible form; and finally, that it is gradually reconverted into glycose by the action of a special ferment, and carried through the hepatic veins to the right side of the heart, and thence to the lungs, where it is oxidised or burnt off.

The last point of the theory has been modified by increasing knowledge; neither glycose nor any other combustible food is oxidised in the lungs. The process of oxidation and heat-making takes place neither in the lungs nor in the blood, but in the tissues, and particularly in the most active tissues, the muscles and the glands.

Again, it was ascertained by Dr Pavy (and afterwards confirmed by the late Dr Robert McDonnell, of Dublin, and by Ritter and others abroad) that the large amount of sugar found soon after death in the liver is formed after its removal from the body. If immediately on an animal being killed the liver is cut up and thrown into boiling water, so as to destroy the ferment, only traces, and sometimes not even traces, of sugar can be found. This is, in fact, the method on which is based the ordinary preparation of glycogen. Pavy also showed, by passing a catheter down the jugular vein into the right ventricle, that blood obtained straight from the hepatic veins of a living dog or rabbit was approximately free from sugar. These experiments certainly established the conclusion that there is no amount of sugar in the liver during life at all comparable to the large quantity obtained a few hours after death; in fact, the active stream of blood which flows through the portal capillaries would immediately wash out the sugar if present, like the stream of water which Bernard used in the dead liver. But it is doubtful whether Dr Pavy's experiments, multiplied and varied as they have been, have established the conclusion which he drew from them, namely, that glycogen is not reconverted into sugar during life, but undergoes some other transformation—as he believes, into fat. The varying amount of glycogen in the liver, its increase after food and diminution after fasting, seems to

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show that it is constantly undergoing some transformation, and the storage of fat by the liver is in direct, not inverse ratio to that of glycogen. Moreover, Dr Pavy agrees with other physiologists in admitting the normal presence of glycose, though in small quantity, in the blood; and as in the case of urea or the alkaline urates, we must remember that with the considerable mass and rapid circulation of the blood, large renewal and equally large abstraction of its constituents are continually taking place. Fat, sugar, urea, urates and kreatinin, bilirubin and tyrosin, may be excreted so rapidly as to form a very large amount in a short time, and are thus rightly regarded as of great importance in the economy, although it is only possible to demonstrate their presence from any single specimen of blood in quantities so small as to seem at first sight insignificant.

It is certain that the glycose absorbed after every meal is, for the most part at least, stored as glycogen in the liver; otherwise it would be found periodically increased in the blood, which experiments on animals prove is not the case; and if present in the blood it would, from its high diffusibility, equally certainly appear in the urine, so that glycosuria would be a normal and not a pathological event.

Most physiologists, therefore, believe that the glycogen stored in the liver is slowly converted into glycose, and gradually carried off in the blood to be oxidised in the tissues; and that this process is so complete in health that nothing but the merest trace, and perhaps not even that, normally appears in the urine.

The pathology of glycosuria.—We have next to inquire what leads to the enormous excretion of glycose in diabetes, sometimes amounting to two pounds in the twenty-four hours.

The first and natural hypothesis was that the kidneys secreted the abnormal constituent glycose, as they were supposed to secrete the other constituents of healthy or morbid urine. But it is now ascertained that the kidneys do not secrete, in the sense of manufacturing, the urea, uric acid, or kreatinin, any more than the chlorides and phosphates; they only secrete in the sense of separating them from the blood of the renal artery. And it is well established by observation and experiment that the same is true of the secretion of leucin and tyrosin, of oxalates, and of bilirubin, as well as of sugar—in acute yellow atrophy, in the formation of calculi, in jaundice, and in diabetes. The first point, then, established in the natural history of diabetes is that it is not a disease of the kidneys at all, but depends upon some antecedent derangement of the chemistry of the body. In the case of so diffusible a body as glycose, glycosuria is the natural and inevitable result of preceding glycosæmia.

We next inquire to what cause is due the excess of sugar in the blood. Obviously it must depend upon one or more of the following: either the production of sugar must be increased, or it must be saved from being stored away and otherwise disposed of as glycogen, or its destruction by oxidation in the tissues must be diminished.

We shall see that in most cases of diabetes it is possible greatly to reduce the amount of sugar in the urine, and therefore, we infer, of sugar in the blood, by cutting off the supply of carbohydrates in the food; but it is found that animals fed on nitrogenous food alone are capable of forming glycogen in the liver, just as they are capable of forming fat, by splitting up the highly complex proteid molecule into a nitrogenous and a non-nitrogenous moiety. Nevertheless this would be quite insufficient to supply the amount of

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sugar excreted in severe cases of diabetes, unless it were all passed through the liver tissues and kidneys without diminution. Moreover, the fact that no increase in the ingestion of carbohydrates will produce glycosuria in a healthy animal or man proves that if in diabetes there be increased entry of sugar into the general circulation, this depends not on increased absorption of glycose by the portal vein, but on one of two causes: either on glycose not being duly stored away in the liver, or on its increased production from the stored glycogen. On either supposition the excess of sugar in the blood depends on the disturbance of the glycogenic functions of the liver.

We can thus see why, in the worst cases of diabetes, it is of little service to limit the ingestion of food. Whatever carbohydrates are taken appear at once in the blood and run off by the urine, and if all such food is rigidly abstained from, the proteids themselves furnish a considerable amount of glycogen to be excreted; starvation would be the only physiological cure in such cases, and the natural result of diabetes is that a man eating with a good appetite and digesting without discomfort is nevertheless undergoing slow starvation. The defective power of converting glycose into the indiffusible glycogen by the liver is, therefore, theoretically a sufficient cause for glycosuria; and it seems probable that this exists, at least in some cases.

Two facts, however, prove that it is, at all events, not the only cause for the disease: for, firstly, the examination of the liver immediately after death, in artificial diabetes in animals, and soon after death in the diabetes of man, does not show such constant diminution of the amount of glycogen as we should expect; and, secondly, although Bernard obtained glycosuria by ligaturing the portal vein, the same result does not follow obstruction of the hepatic circulation by disease, as cirrhosis, in man.*

Another objection to glycosuria depending upon the diminished power of the liver to put away the sugar which reaches it in the form of glycogen was thus put by Dr Fagge:—"If a patient, whose urine is kept free from sugar by a restricted diet, one day breaks through the dietetic rules laid down for his guidance, and eats an apple or drinks a glass of sweet beer, it will generally be found that the secretion again becomes saccharine. This is no more than might be expected; but now comes a circumstance which is remarkable, and for which some further explanation is evidently necessary. The quantity of sugar that is voided is altogether disproportionate to the amount contained in the apple or the beer; and sugar often continues to be excreted for a very long time afterwards. Dr Pavy mentions the case of a patient in whom the disease had been kept under control by strict dieting, and who drank about a pint of cider. His urine thereupon became loaded with sugar, and remained so for a period of two months, before it again became normal. Now, the only hypothesis which seems capable of explaining such facts as these is that the saccharine or amylaceous article of food exerts some directly injurious influence, so as to cause the blood to contain

* When there is extreme obstruction to the portal circulation in the liver, the blood must needs go in great part by other channels. Very little glycose would then be transformed into glycogen, the large amount absorbed after digestion would be excreted by the kidneys, and thus glycosuria would result. Bernard himself tied the portal vein in a dog, and thus produced artificial *glycosuria alimentaire*. Dr Lépine (following Couturier) observed the same result in three patients with cirrhosis of the liver (verified after death) who were fed on starchy and saccharine diet, and he did not obtain it in cases of cancer of the liver and of fatty liver in phthisis. The result, however, is far from constant; it failed when tried several years ago by the present writer at Guy's Hospital, and sugar ought far more frequently to appear in the urine in cases of cirrhosis, adhesive pylephlebitis, and portal thrombosis. The same negative result was obtained by Frerichs.

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an excess of sugar for long afterwards; and, if we believe that the liver is the organ principally concerned in supplying the blood with sugar, we can hardly help inferring that it is the liver on which this injurious influence is exerted. It seems as if saccharine food were a *poison* to a patient who is affected in this way."

In any case, if we assume that in health the glycose which reaches the liver is stored as glycogen, while in diabetes it runs through the portal circulation unchanged, we must find some other normal destination for glycogen, or else all the sugar absorbed from the food will sooner or later be returned to the blood. Dr Pavy finds this destination in fat, but at present his hypothesis cannot be proved, either chemically or physiologically.

If we still ascribe glycosuria to increase of sugar in the blood, but deduce this increase from a too rapid change of glycogen into glycose, we have an attractive explanation of the way in which this occurs. After Bernard had established the glycogenic function of the liver, he made the equally brilliant discovery that by puncturing a definite spot in the floor of the fourth cerebral ventricle we can produce glycosuria, or, as it is called, artificial diabetes. The explanation offered is that the vaso-motor centre of the liver being paralysed, the hepatic artery dilates, the flow of blood through the liver is increased, and hence the passage of glycose into the circulation is augmented. One objection to the theory is that sugar is carried to the liver by the portal vein, not by the hepatic artery; while the distribution of the portal branches, as well as their greater size and number, makes it probable that it is from the portal rather than the arterial blood that glycogen is formed. Another is that the duration of artificial diabetes is very limited, and that although we shall see hereafter that diabetes has been observed as the sequel of nervous causes, injury or disease affecting the fourth ventricle appears to produce transitory glycosuria rather than permanent diabetes. Again, Dr Pavy has shown experimental ground for supposing that the glycosuria which follows Bernard's puncture is not due to the mere dilatation of the hepatic artery which follows, but to the increased presence of oxygen, for he has obtained glycosuria by causing arterial blood to flow through the portal vein.

A broad ground of objection has been taken to either of the theories which ascribe glycosuria to increased production of sugar; namely, that even if all the glycose which can be produced by digestion of the proteids taken in twenty-four hours by a diabetic patient under strict diet were supposed to pass through the liver without any of it being stored as glycogen, it would not account for the enormous amounts which are passed in the urine. This conclusion seems established by observations upon dogs with artificial diabetes, and in human subjects under strict diet it is certainly true of the most severe cases, when much more sugar is passed by the urine than could be the product of the proteids digested, unless we add to them the fixed proteids of the tissues. But this argument, though used by several writers of repute, seems to prove too much. For if a man or a dog with diabetes really passes a greater weight of sugar in the twenty-four hours than is accounted for by the food he eats, the fact is just as conclusive against the theory next to be considered, that of diminished destruction of sugar. The only explanation, if the facts are as stated, must be that from the fatty elements of food, in some unknown way, more carbohydrates enter the blood and urine as glycose than are ingested as food.

After all, the facts are very doubtful. Külz found that there is never so

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much sugar excreted in diabetes as answers to the amount of carbohydrates ingested; some is always oxidised and burnt off.

Let us now see what may be said for the hypothesis of glycosæmia, and so glycosuria, being the result of diminished destruction of sugar. In health the glycose, which is the product of digestion of carbohydrates, undergoes an imperfectly ascertained chemical dissolution in the tissues, and is afterwards oxidised into carbon dioxide and water, to be thus eliminated from the body, while the chemical energy of the combination is liberated, and reappears as animal heat and the work of muscles, glands, and other less active tissues. That retention of sugar is not the direct and immediate result of deficient oxidation is shown by the absence of glycosuria, except in very small amount and under exceptional circumstances, in cases of cyanosis, of laryngeal dyspnœa, bronchitis and emphysema, cardiac disease and anæmia. In all these cases the antecedent process, whatever it is, which fits glycose for oxidation, must have been performed, and we must suppose that it is in this changed condition that a great part of the glycose of the blood remains unoxidised.

Another proof that it is not lack of oxidation which keeps the proportion of glycose in the blood so high, is the fact that other oxidations go on as usual; for in natural or artificial diabetes fatty food is perfectly assimilated and excreted as carbonic acid and water. Nencke found by experiment that benzole was changed into carbolic acid, Schulzen recovered vegetable salts as carbonates, and Külz showed that certain carbohydrates even were oxidised, as lævulose and inosite. Schulzen has found that in cases of phosphorus poisoning, when oxidation is much hindered, glycose is still split up ready for oxidation, and appears as lactic acid in the urine.

The following argument seems to show that the hypothesis of diminished destruction does not hold for all cases of diabetes. If the liver were storing its glycogen normally, as each fresh accretion of glycose reached it, the diminished destruction of sugar in the tissues would keep the amount in the blood and urine at a pretty uniform quantity, but in diabetics it is well known that the sugar excreted by the kidneys is increased after each meal.*

On the whole, we must admit that the riddle of glycosuria has not yet been solved. It would, of course, be easy to assume, as some have done, several kinds of diabetes: one due to increased production of sugar and diminished deposit of glycogen, another to increased production of sugar from abundant glycogen, a third due to diminished oxidation, and a fourth to diminished preliminary decomposition of the sugar in the blood.

But the distribution to each category would be very arbitrary, and in the case of so well-marked and peculiar a clinical condition as that of diabetes it seems more probable that it has a constant origin.

Artificial diabetes.—Before quitting this part of our subject we must briefly refer to certain conditions known as "artificial diabetes," or rather artificial glycosuria.

* A diabetic patient, Joseph North, under Dr Pavy's care, was placed in succession upon different kinds of food, and his urine was collected and analysed every four hours. As a rule, the amount of sugar excreted in the urine was considerably greater between 5 and 9 p.m. than at any other part of the day; while it was commonly at its lowest point during the night and early morning. These variations were evidently due to the food taken during the day. On one occasion he departed from his instructions, and at 4 p.m. drank some cocoa sweetened with sugar. Between 5 and 9 p.m. of that day he passed 1311 grains of sugar, whereas in the twenty-four hours previously the quantity in equal periods had ranged between 166 and 468 grains; and from 9 to 1 a.m. the same evening it was again only 483 grains.

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The first is the result of Claude Bernard's famous experiment of puncturing the "diabetic centre" in the bulb. *Traumatic* glycosuria is produced; but the experiment fails if from starvation or other cause the liver of the animal contains no glycogen. The sugar derived from its food apparently does not pass on into the circulation, but is stored as glycogen; while the sugar formed from glycogen in the liver is washed out abundantly by the augmented stream of blood—an argument, as far as it goes, for Bernard's rather than Pavy's view. But this glycosuria is temporary only, as indeed we should expect, for it is clear that after the puncture, in proportion as the amount of glycogen in the liver becomes reduced by the excessive demands made upon it, the glycosuria must pass off; and, in fact, after a few hours sugar can no longer be detected in the urine of animals on which this experiment has been performed. It is true that Schiff found that by dividing the anterior columns of the cord in rats he could make the animals diabetic, and that this condition would last for two or three weeks. But even these results scarcely warrant our attributing ordinary persistent diabetes in the human subject to paralysis of the vaso-motor nerves. It seems to be impossible that the excretion of such large quantities of sugar as are passed by patients affected with this disease, at all hours of the day, and in amounts influenced only partially by the meals, can be due to a mere increased rapidity of the conversion into sugar of the glycogen naturally formed by the liver.

The second artificial kind of glycosuria is *toxic* (cf. p. 570). It has been observed after poisoning by chloral, curare, and after inhalation of chloroform and carbonic oxide gas; but, except in the latter case, the presence of sugar in the urine is far from constant. It has, however, been ascertained that feeding animals on phlorizin ($C_{21}H_{24}O_{10}$), a constituent of the bark of cherry and apple trees, will always produce glycosuria.

These interesting facts throw much light and will throw more on the physiology of glycose, but they can only indirectly explain diabetes; for the disease is, at least in most cases, due neither to injuries of the nervous system nor to ingestion of poison.

Theory of the other symptoms.—Given glycosæmia, the other pathological effects seem to follow naturally. With a diffusible crystalloid like dextrose glycosuria will at once follow. That in itself leads to polyuria, and then the flow of urine produces dryness of the tissues, constipation, and thirst; while the loss of oxidisable food leads to hunger, emaciation, muscular weakness, lowered temperature; while dextrose, or rather perhaps concomitant products of altered metabolism—as aceto-acetic or oxybutyric acids—act as poison in producing local gangrenous inflammation in the skin or the lungs, or at least prepare a suitable soil for the growth of the tubercular bacillus.

Clinical ætiology of diabetes.—Some interesting cases have been recorded in which the urine has been found to contain sugar after the brain has been injured, or when it was diseased. As far back as 1854 Dr Goolden published in the 'Lancet' a series of instances of glycosuria following blows or falls upon the head; but in most of them the presence of sugar in the urine was transitory, like that in dogs and rabbits after Bernard's puncture. Dr Pavy, however, mentions the case of a cadet at Woolwich who was attacked with strongly-marked diabetes a few days after being stunned by a blow on the head. He also records two instances of diabetes

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after an attack of hemiplegia, and he has frequently seen it in ataxic patients. Many years ago (about 1867) a little girl was brought into Guy's Hospital dead from fracture of the skull and other injuries in being run over. A patch of hæmorrhage was found by the writer in the floor of the fourth ventricle. Some urine was then collected from the bladder and gave decided evidence of sugar. Frerichs mentions a large number of similar cases. The most striking case in which disease of the fourth ventricle has led to glycosuria in man is one quoted by Trousseau, in which there was a tumour in the floor of this cavity. Similar cases have been since recorded; but, interesting as they are from a physiological point of view, they lend small support to a nervous theory of diabetes.

Sometimes the disease appears to be a result of mental anxiety. Dr Hermann Weber met with the case of a gentleman who became diabetic on two separate occasions, at an interval of nine years, under the pressure of intense anxiety from impending commercial ruin. In other cases recorded by Rayer and Frerichs, diabetes has followed a violent fit of grief or of anger, and Dr Dickinson mentions several similar cases. It has been stated by Dr Maudsley that diabetic parents often have insane children, and Dr Dickinson found glycosuria in 17 per cent. of insane persons. But Dr Hale White found it in only 4 per cent. of the inmates of Bethlem, and in 2·6 at the Surrey County Asylum ('Path. Trans.,' 1883, p. 353). Dr Savage also believes that insanity and diabetes are unconnected ('Brit. Med. Journ.,' 1885, vol. ii, p. 1054).

An *inherited tendency* is clearly present in some cases of diabetes. Pavy gives numerous instances of this: in one family two sisters and two brothers; in another a son, his father, and an aunt; in a third a father and his two daughters; in a fourth a father and a son; in a fifth two brothers; in a sixth three brothers; in a seventh a brother and a sister; and in an eighth the father, mother, and three daughters; and (to mention only one other case) a mother, grandmother, and four out of five children. Roberts knew a family of eight children, all of whom became diabetic, although the parents were healthy. Frerichs found 39 hereditary cases among 400.

One hundred and twenty-two cases have been collected for the writer by Mr J. E. Nevins from the records of Guy's Hospital, and in eight of these there was a history of diabetes in one or more members of the patient's family (6·55 per cent.). Adding cases obtained by the courtesy of the registrars of the three other large hospitals, St Bartholomew's, St Thomas's, and the London, and those recorded by Schmitz and Griesinger, he found 35 hereditary cases in a total of 537 (6·51 per cent.). In one remarkable case the mother and seven of the mother's brothers had died of diabetes.

Many writers, particularly on the Continent where *gout* is rare, ascribe a large share to that disease in the production of diabetes. It is said that gouty diabetes is marked by the patient being stout and ruddy and suffering little, as well as by his being elderly. But this means little more than that gout does not exclude diabetes, and that many physicians call men over forty, with florid complexion and well nourished, gouty without more ado. If we inquire how many diabetics have suffered from inflammation of the great toe, or from chalkstones, we should probably not find the proportion above that of the accidental coincidence of two not uncommon diseases which both affect men rather than women, and those over forty rather than younger persons. It is instructive to observe that some physicians

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with wide opportunities of observation have believed in the antagonism of gout and diabetes (p. 572).

Distribution.—Diabetes is said to be more common in Italy than in Northern Europe, more common in Ireland and Scotland than in England, more common in Saxony and Thüringen than in Berlin, and more common in Ceylon and parts of India than in China, Mauritius, or Berbice. Jews are said to be particularly liable to it,* and negroes to be almost exempt.

Dr Pavy finds that it is more frequent in private than in hospital practice, and that on the average private patients are older than those in hospital. Trousseau used to say that when a sleek, plump, pink-faced patient came into his consulting room, he was sure to be a notary and diabetic.

Age and sex.—Diabetes rarely attacks young children; whereas in men there is no decrease in the mortality from diabetes until the age of sixty-five has been passed. Among 1360 private patients who came to Dr Pavy, diabetes began under ten years old in 8, between ten and twenty in 57, between twenty and thirty in 97, between thirty and forty in 224, between forty and fifty in 339, and between fifty and sixty in 418. Even between sixty and seventy the number was 182—very large considering the smaller number of persons living over sixty compared with those between twenty and forty. Above seventy there were 35 patients, one in whom the disease began when he was over eighty. The youngest patient he ever saw was an infant twelve months old ('Brit. Med. Journ.,' Dec. 5th, 1885).

Diabetes is far more common in men than in women: in 537 cases at *Guy's* Hospital, above two to one; in Dr Pavy's private cases, 2·45 to one. In every decade (excepting the children under ten) the number of males was far greater than that of females, and the total was 966 to 394. It is particularly rare in old women. The predominance of the male sex in this, as in some other diseases, does not appear during childhood.

Course and event.—In its well-marked forms, diabetes tends to the death of the patient, and that in a comparatively short time if he be under middle age. It is true that one cannot often state definitely what is the whole duration of the disease, because its onset is generally insidious; but sometimes the patient can fix the week and almost the day on which he felt unusual weakness and thirst, and found his urine unusually abundant. The important fact is that, for those under forty, when the urine has once been found to be persistently saccharine, the days of the patient are numbered, unless the progress of the malady can be arrested.

Cases are sometimes met with in which death occurs within a few weeks from the time when the first symptoms of the disease appear. Sir George Paget observed a case in which the patient, a Cambridge undergraduate supposed to be in perfect health, took part in athletic sports and came in second in a foot-race, within twelve days of his death. The writer had recently a young man under his care who died from diabetic coma after only two months' illness. Such cases of acute diabetes are, however, rare; more frequently the duration of diabetes is from one to three years. Prout said in 1848 that, among nearly seven hundred patients whom he had seen within thirty years, he then knew of but two in whom the disease had been fully developed ten years before. Dr Dickinson, however, mentions

* Frerichs, for instance, had 102 Jews among his 400 diabetic patients.

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the case of one patient in whom the urine was constantly saccharine for fifteen years, and Dr Pavy a similar case which lasted twenty-five years.

The *age* of the patient appears to have a greater influence than any other condition on the rate with which diabetes advances towards a fatal termination. Children who are attacked by it never live to grow up. On the other hand, old people sometimes pass saccharine urine for years without appearing to suffer from it; but in such patients the urine is seldom excessive in quantity, nor are marked symptoms of diabetes generally present.

At a middle period of life, say between thirty-five or forty and sixty-five, the prognosis of a case of diabetes, when it first comes under observation, must mainly be based upon the degree of severity of the symptoms, particularly emaciation. All writers regard it as a favourable sign if the patient remains well-nourished and florid. If sugar is present without much increase in quantity of the urine the prognosis is better than when there is polyuria. In fact, the older physicians used to call the former cases *diabetes decipiens*. It is generally supposed that the detection of albumen in the urine in addition to the sugar is a serious indication, but Dr Pavy says that he has known a small quantity of albumen to be present for years without apparent harm.

But these considerations, which guide the physician in forming his first impression as to the probable issue of a case of diabetes, require to be combined with, and corrected by, the results of treatment, before a final judgment is pronounced. For medical science sometimes attains more striking results in this disease than in almost any other of equally dangerous tendencies, and the cases which do best under treatment are not always those which appeared the most promising at first. If under moderately restricted diet the amount of sugar rapidly diminishes and the patient's discomforts diminish also, the prognosis is favourable, and particularly if he makes flesh.

Treatment by diet.—Theory and experience agree in teaching that by diminishing the amount of glycose in the urine we diminish it in the blood, and that by diminishing it in the blood we relieve all the symptoms of diabetes. They equally teach that we can in all cases limit, and, in most, prevent the excess of sugar in the blood by removing carbohydrates from the food of diabetic patients. Thus, as Prout long ago laid down, "diet is the first and chief point to be attended to."

Dr John Rollo, at the end of the last century, appears to have first prescribed abstention from starch and sugar; he proposed to confine diabetic patients entirely to animal food. But although the inhabitants of arctic regions and the trappers of North America live for months together without the chance of obtaining anything else, experience shows that in civilised life, when all kinds of food are within reach, there is very great difficulty in keeping patients exclusively on meat; and the more so since in diabetes the appetite is voracious, and the craving for forbidden food all the greater. Of late years, therefore, all those who have studied the subject have striven to include among permitted viands as many of vegetable origin as possible. The result has been the construction of a tolerably copious diet-table.

Almost all kinds of *animal food*, flesh, fish, and fowl, may be eaten by persons suffering from diabetes. But it is necessary to bear in mind that

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the ordinary methods of cooking such food often introduce deleterious matters. Thus soups are thickened, jellies are sweetened, and even roasted joints are basted with flour. There are few alimentary substances of animal origin which produce sugar; but such is the liver of calves or pigs, and such are also the edible molluscs, oysters, cockles, mussels, all of which contain glycogen. It may, however, be noted that in the case of Joseph North (p. 583, *note*), to whom Dr Pavy twice gave four dozen of oysters for two days running, they caused no decided increase in the amount of sugar excreted. *Honey* (yielding dextrose and lævulose) is of course injurious; and so is also *milk* (which yields dextrose and galactose), but not in the degree that might have been inferred from the amount of lactose it contains. Dr Pavy indeed found that in North's case the administration of three pints of milk daily not only caused a marked increase in the amount of sugar, but also brought back the uneasy sensations which the man had experienced when the disease had been uncontrolled by treatment. But Roberts mentions the case of a girl who (her diet being restricted in other respects) continued to gain strength and to improve in health when she was allowed to drink three pints of milk daily.

Cream and butter form important articles of diet in diabetes. Cod-liver oil is useful in a similar way. Though glycerine might be expected to be harmless, since its composition is so different from that of sugar, yet Dr Pavy found that it caused a great increase in the sugar excreted; but the daily quantity which he administered was from six to ten ounces, and it does not follow that smaller quantities are injurious. A better substitute for sugar than glycerine is Saccharine (Benzoyl-sulphonic-imide). It sweetens far more powerfully than cane-sugar, and of course does not yield glycose; but it renders the saliva unpleasantly sweet.

The late Dr A. S. Donkin's plan of treating diabetic patients by a diet of *skimmed milk*, six or eight pints daily, is not theoretically justified, for the lactose is taken and the cream left behind. Dr Greenhow recorded a case in which a patient took from four to six quarts of skimmed milk daily, with the effect of removing his symptoms and of freeing his urine from sugar, and two months afterwards he remained well. But Roberts refers to the cases of several persons treated with skimmed milk, and says that few of them could tolerate it for more than a few days, and then were rapidly reduced, while one case was brought to a fatal termination in three months. Much more likely to be useful is a preparation of milk, which Dr A. E. Wright lately showed,* made by precipitating the casein with dilute acetic acid, and then redissolving the curd in an alkaline solution of the phosphates and other salts of milk. The result is a not unpalatable drink, which can be sweetened if desired with a little saccharine, and is entirely free from sugar. Practically, however, most diabetics do without much milk, and a small amount is not generally found injurious.

Most *vegetables* are injurious to diabetic patients. The most noxious of all are those richest in starch, as potatoes and artichokes; but carrots, parsnips, turnips, peas, beans, Brussels sprouts, cauliflower, broccoli, asparagus, and sea-kale are also forbidden, as containing starch or sugar.

Greens, however, as well as spinach, lettuces, watercresses, and mustard and cress, may be eaten; and even small quantities of radishes and asparagus or celery. The general rule is that all white parts of vegetables, in which

* In the Grocers' Lecture at the University of London, which was published in the 'British Medical Journal,' April 11th, 1891.

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chlorophyll has not been developed by exposure to sunlight, contain sugar, and are harmful. But by boiling in a large quantity of water, even the forbidden kinds of vegetables, if they contain sugar only and not starch, may be rendered much less injurious. According to Prout, there is a direct advantage in the use of such green vegetables as are harmless; for their indigestible part, cellulose or lignin, tends to correct the constipation which is often so troublesome in diabetes.

All kinds of sweet fruits appear to be injurious, although currants and raspberries and other acid fruits are sometimes allowed to diabetic patients. Nuts are harmless; but chestnuts abound in starch.

The greatest difficulty is for the diabetic patient to do without *bread.* Consequently, there have been many attempts to invent a substitute for it. The earliest of these attempts was perhaps that of Bouchardat, who, in 1841, suggested that a kind of bread should be made from the gluten of flour, after removal of all the starch by washing. But this gluten bread is by no means perfectly free from starch, for it at once turns blue if a little iodine be dropped on it. Patients also complain that it is very unpleasant to chew, feeling like india-rubber between the teeth. Dr Pavy says that gluten biscuits are much more palatable, but these will not keep more than ten days. Gluten is also prepared for cooking purposes, by which variety may be given to the meals put before a diabetic patient. A second substitute for ordinary bread is a sort of cake from bran. Prout appears to have first suggested this; and Dr Camplin, who himself suffered from diabetes, greatly improved the method by which it is prepared, so that bran cakes are still made according to his directions. A third substance which may be used by diabetic patients instead of bread is the almond cake which was introduced by Dr Pavy; its only fault is its high price.*

If inulin, the starch of dahlias and several other roots, could be produced in abundance, it might be used generally instead of wheaten flour, for it only yields lævulose on digestion instead of dextrose.

Wheaten flour, as well as that of other kinds of corn, is of course very injurious; and so are rice, arrowroot, sago, tapioca, macaroni, and vermicelli. In all but the most severe cases we may allow a very little toast.

It is necessary to regulate the *drink* as well as the food of diabetic patients. Restriction of the amount of water taken into the stomach is followed by but little advantage, and it adds greatly to the patient's discomfort, if he obeys the physician's injunctions.

The beverages which are admissible are tea, coffee, cocoa made from nibs, dry sherry, claret, burgundy, chablis, hock; also brandy and other unsweetened spirits, with soda and other mineral waters. Chocolate, sweet ales, porter and stout, cider, all sweet and sparkling wines are forbidden; and it is not desirable that alcohol in any form should be freely taken by diabetic patients.

Dr Pavy says that the patient often at first complains that his food is not bulky enough to satisfy him; but after persevering for a few days he finds that his appetite becomes less. Johnson's dictum that it is easier to abstain than to be abstemious is very applicable to the diabetic.

Before a patient suffering from this disease adopts the restricted diet, and also while it is being carried out, the amount of urine passed each day and its specific gravity should be carefully registered, and the quantity of sugar should also be accurately determined at frequent intervals. In no

* Dr Palmer, of Nayland, recommends the *boules de soupe*, little dumplings of gluten.

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other way can the progress of the case be properly watched, or an opinion be formed as to the necessity for subsidiary treatment. Moreover, a daily analysis enables the physician to check the proceedings of his patient, and to tell with considerable certainty whether he is adhering strictly to the prescribed rules. Those who suffer from diabetes are often untruthful, and wilfully deceive their medical advisers. Prout speaks of a mental imbecility and want of fortitude as one of the effects of the disease. This may perhaps be the explanation of the assertions that have been made that diabetic patients sometimes pass a quantity of urine exceeding that of the fluids ingested. Niemeyer mentions a case, in which careful watching proved that the patient had really been drinking a larger quantity of fluid than that to which she had confessed. Patients are often detected in having surreptitiously eaten apples, bread, &c., by the discovery that the urine has on some one day been found to contain a larger quantity of sugar than had been present before. The advantage gained by daily analysis is therefore considerable.

The effect of dieting varies. Sometimes it cures the disease entirely. Roberts gives a case of this kind. A man aged thirty-nine was passing eight pints of urine daily, containing 5680 grains of sugar. Under a restricted diet the mean daily flow of urine went down within a week to 60 ounces; the sugar fell on the third day to 134 grains, at the end of a week to 48 grains, and after a fortnight only a trace of it could be detected. The patient lost all his symptoms, and gained flesh at the rate of three pounds a week. In about eight months all the sugar had disappeared, and nearly four years afterwards he was quite well.

The present writer lately met with a somewhat similar case. The patient, a stout florid man of forty-three, had begun rapidly to lose strength and weight, and to suffer from thirst and frequent micturition. The symptoms dated from early in December, 1890, and sugar was detected soon after. When first seen (January 1st, 1891) he had the raw tongue, constipation, and all the other discomforts of acute, and somewhat advanced, diabetes. The urine was of sp. gr. 1032, and contained a large amount of glycose. He was put on restricted diet with a grain of opium three times a day. He had greatly improved in a week, and the sugar had diminished to about a third of its previous amount. He persevered with the treatment, and three weeks after it was begun he felt perfectly well, and the urine was moderate in amount, of sp. gr. 1021, and nearly free from sugar. On February 26th the urine was perfectly normal, and there was no return of symptoms two months later.

Unfortunately, in the majority of cases the results of treatment are much less decisive. The patient's symptoms may be greatly benefited, or altogether removed; the amount of urine which he passes may be much diminished, or reduced to the normal quantity; it may even cease to contain sugar, so that for the time all signs of the disease are absent; and yet, if he now attempts to return to his previous way of living, the urine again becomes saccharine, and one by one all the symptoms of diabetes reappear.

Indeed, even when the patient under strict diet loses his symptoms, and the quantity of urine which he passes falls to the normal amount, it by no means always ceases to contain sugar. Its specific gravity may be hardly, if at all, lowered; and the percentage of sugar (although not the absolute amount excreted) may be as great as before. Whenever dieting, or indeed

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any other treatment, does good, it appears always to bring the quantity of water down to normal, before seriously affecting the specific gravity or the proportionate amount of sugar contained in the secretion. But whenever dieting readily lowers the specific gravity, it may be assumed that the quantity of urine passed by the patient is not excessive.

Sometimes a restricted diet altogether fails to control diabetes. There are cases, especially in very young patients, in which all treatment appears to be useless. Other patients, again, are at first benefited by dieting; but after a time it ceases to be useful, and in certain cases the disease appears to advance less instead of more rapidly when all restrictions are withdrawn. Sometimes, again, the appetite fails entirely as soon as the patient is required to give up starch and sugar.

Drugs.—When dieting fails, or as adjuvants to dietetic treatment, various drugs have been recommended, but only one has satisfactory evidence in its favour. Pepsin and rennet have been tested by Parkes, Griesinger, and Roberts, and found useless or injurious. Arsenic, strychnia, belladonna, physostigma, and lactic acid have all been tried and abandoned.

The only drug that has stood the test of experience in diabetes is *opium.* It seems to have been first used by Rollo, its value was known to Prout, and Sir Thomas Watson spoke of it as a treasure. It was also known that patients suffering from this disease could take much larger quantities of opiates than healthy persons. The earlier writers, however, recommended that its dose should as much as possible be kept within moderate limits. Dr Pavy advocates a very different way of administering opium. He gives it in increasing doses, until he finds either that the disease yields or that the patient is unable to take it. We do not know how opium acts in diabetes. Roberts—who says that, without any restriction as to diet, daily doses of six to twenty grains always reduce the quantity of urine by about one half—believes that in patients who are dieted it is useless, except as a sedative. This, however, is inconsistent with Dr Pavy's accurate observations; and from a much smaller scale of observation, the present writer would urge the great benefit of opium or codeia in full doses, particularly in severe cases, and when from poverty or carelessness the patient is unable or unwilling to submit to a serious reduction of diet. In such cases avoidance of sugar, potatoes, and pudding can generally be secured, and opium seems to make up for the bread that is taken.

In some cases opium disagrees, either causing narcotic symptoms, constipation, or, occasionally, diarrhœa. When this is the case, there is often great advantage in the employment of *codeia*, as was first recommended by Dr Pavy. Half a grain three times a day should be given at first, but the daily quantity has afterwards been gradually raised to as much as thirty grains. Such large doses have not appeared to do more good than moderate ones of three or four grains thrice a day. Often codeia may be given without any obvious effect beyond diminishing the sugar excreted. The tongue remains clean and the appetite good. Sometimes, however, codeia causes headache. Morphia may then be used: in fact, from the observations of Dr Mitchell Bruce and Prof. Fraser, it appears that in some cases it is more effectual than codeia ('Pract.,' Jan., 1887, and July, 1888; and 'Brit. Med. Journ.,' 1889, i, p. 118).

Ammonia and alkaline carbonates are sometimes of service. The waters of Vichy have great reputation in France, and those of Carlsbad in Germany. Many pathologists believe that the blood of diabetics is habitu-

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ally less alkaline than normal, and it is said to have been occasionally found acid. Careful experiments by Dr A. E. Wright showed in one case that the sugar steadily diminished under alkaline treatment.

Diabetic patients should be clothed in flannel. Bouchardat maintained that active exercise should be taken, and Trousseau recommends it in the strongest terms,* but few English physicians agree with the advice.

The intolerable *thirst* of diabetes may be relieved by dilute phosphoric acid, or solution of bitartrate of potash. Constipation should be treated with Ol. Ricini or Conf. Sennæ or extract of aloes rather than with saline purgatives.

When *diabetic coma* comes on, intravenous injection of saline solution in order to dilute the thickened blood has been repeatedly tried, unfortunately with even less frequent or more temporary success than in the collapse of cholera (vol. i, p. 245).

In the 'Guy's Hospital Reports' for 1873–74 (vol. xix, p. 173) the author recorded a case of injection in which the pulse was scarcely perceptible, and the body and limbs were cold. Twenty-six ounces of a solution of phosphate of soda and chloride of sodium, of sp. gr. 1020, were injected into the right cephalic vein, with the effect of restoring the patient to consciousness for a time. He sat up, answered questions, took nourishment well, and even asked for it; his pulse was 80. Thirty-two hours later, however, he again became drowsy and died. This patient's condition, before the solution was injected into his veins, was strikingly like that of a man in the collapse of cholera: only a few drops of thick dark blood escaped from the wound in the arm. A few months afterwards Dr Frederick Taylor had a similar case, in which he employed the same treatment, but with scarcely any good result (*ibid.*, vol. xix, p. 521, and xxv, p. 169). Dr Dickinson has since put on record a remarkable case in which very free intravenous injection of saline solution had a decided though only temporary effect ('Clin. Trans.,' 1890, p. 130).

Probably the best treatment of threatening diabetic coma is active purging.

* "Un diabétique, qui chaque jour, fait à pied un exercise violent, peut sans changer rien de son régime, retrouver temporairement la santé qu'il avait perdue," &c. ('Leçons,' tome ii, p. 764). It is curious to note that Celsus wrote, "At, cum urina, super potionum modum etiam sine dolore profluens, maciem et periculum facit, si tenuis est [*i. e.* clear and pale] opus est exercitatione," &c. (lib. iv, cap. xx). Prolonged and severe muscular exertion is prescribed now by several German physicians on theoretical grounds, as tending to oxidation of the glycose. In England it is generally believed to be injurious.

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ADDISON'S DISEASE

WITH OTHER AFFECTIONS OF THE ADRENAL BODIES, AND REMAINING ABDOMINAL VISCERA

"Quid te exempta juvat spinis de pluribus una."
HORACE.

Addison's discovery—The anatomical changes in the adrenal bodies—The symptoms: melanodermia, asthenia, cardiac failure, gastric disturbance—Order of the symptoms—Course, mode of death, and duration of the disease—Controversial points—Origin—Relation to caries and to tubercle——Theory of the symptoms—Prognosis—Other morbid states of the adrenals. Diseases of the pancreas, the spleen, the testes, and the ovaries.

ALTHOUGH many diseases have been named after their first describer, it is seldom that the merit of discovery is not divisible with earlier observers who paved the way, or with later ones who corrected or completed the work. If, however, any disease is to be associated with a single name, it is that which is the subject of the present chapter. There are no rival claims of priority, for no one had suspected either the anatomical change in the curious and unique organs which are affected, or the remarkable symptoms which denote this change during life; nor has subsequent investigation added much to Addison's original discovery, the chief conclusion reached by the inquiries of nearly forty years being that it is not every disease of the suprarenal bodies, but one only, caseous or tuberculous inflammation, which produces the clinical results that he described.

It was while engaged in the investigation of anæmia which led to the recognition of that peculiar form named by its discoverer idiopathic, and by others grave, progressive, or pernicious, that Thomas Addison "stumbled," to use his own expression, upon the connection between the symptoms and the lesion, which belong to what has since been known as morbus Addisonii.

This remarkable disease is rare and apparently incurable, so that its interest is at present pathological. It was naturally hoped that the experiments of disease would throw new light upon the obscure question of the function of these anomalous adrenal bodies (*glandulæ suprarenales, vel atrabiliosæ, renes succenturiati*, as they were once called); but this expectation has not yet been fulfilled.

Melasma suprarenale was the name proposed by Addison in his original work on 'The Constitutional and Local Effects of Diseases of the Suprarenal Capsules,' which appeared in 1858, and has since been republished among Addison's works by the New Sydenham Society, 1868.

The most important later facts on the subject will be found in the ninth volume of the 'Pathological Transactions' (1858), in Dr Wilks's papers in

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the eighth and eleventh volumes of the third series of 'Guy's Hospital Reports' (1862 and 1865), and in Dr Greenhow's Croonian Lectures "on Addison's Disease" (1875).

Dr Wilks has drawn attention to two undoubted cases of the disease, described (but not recognised as dependent on the lesion of the adrenals) by Aran in 1846, and by Bright in 1829, the diseased organs of the latter case being still preserved in our museum, No. 2020[35] ('Guy's Hospital Reports,' third series, xxii, p. 266).

Anatomy.—The exact characters of the lesion of the adrenal bodies vary in different cases, but only within such limits as may reasonably be supposed to belong to the successive periods of a single morbid process. The earliest change seems to be the formation, within the medullary substance, of a firm grey or whitish mass, which is more or less nodulated at its growing edge, and sometimes surrounded by clusters of what appear to be miliary tubercles. In this stage, however, the disease rarely becomes the subject of anatomical investigation. When death has occurred the grey material is generally found to have already extended into and destroyed the cortical substance. The viscus is then much enlarged, hard, and of irregular form; when a section is made, the cut surface shows no trace of the natural structure; it may be of a more or less uniform grey colour, which sometimes acquires a pink hue when exposed to the air. In most cases, however, certain parts of the grey substance have undergone caseation, forming rounded yellow nodules embedded in it; and, at a still later stage, the conversion is complete, so that there is only a single large yellow mass. Softening often takes place at this stage into a purulent or rather a puriform liquid. Finally, a process of absorption begins, and the diseased organ, from being many times larger than natural, shrinks into a small puckered mass, in which irregular nodules of calcareous matter are embedded.

During the early stages of this process the fibrous envelope of the organ becomes swollen until it is from half a line to two lines in thickness. Adhesions to the neighbouring parts are also formed, principally to the liver, kidney, diaphragm or stomach, pancreas, or vena cava.

In some cases nothing is left but a hard, puckered, fibrous knot, sending out bands into the fatty tissue in which it lay embedded, and often containing irregular calcareous masses.

The morbid changes in Addison's disease are probably never limited to a single suprarenal body. One, however, is generally attacked earlier than the other, and on *post-mortem* examination may sometimes be found completely destroyed, while its fellow is still in an early stage of disease.

The new tissue which is formed at the commencement of the morbid process consists of small round cells like lymph-corpuscles lying in the meshes of a delicate, wavy, fibrillated stroma. These cells may subsequently undergo development, and form well-marked fibrous tissue, for some parts, still grey and translucent, have been found to consist entirely of such tissue, with some elongated cells and oval nuclei interspersed between its fibres. It seems, from Dr Wilks's original description in the 'Guy's Hospital Reports' for 1862 and 1865, that the specimens that he examined were generally in this stage. He speaks of a material which he says is "without structure, or sometimes slightly fibrillated, or containing a few abortive nuclei or cells." When caseation has taken place the microscopical appearances are the same as those of any other structure which has undergone this change.

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No one can read the description just given (as written by Dr Fagge for the first edition of this book) without seeing that it corresponds to the "scrofulous," or "strumous," *i. e.* tuberculous inflammation which we have met with in other organs.

Histology.—Addison spoke of the anatomical lesion as "scrofulous," but formerly this name was applied to all caseous processes. Wilks was inclined to deny that the disease was of a tubercular nature; he maintained that cases in which well-marked tubercles were discovered in other viscera were exceptional. Virchow and Rindfleisch, however, decided to call Addison's disease a tuberculosis of the adrenals, and there is now no doubt that they were right. They describe as the earliest stage of the affection one in which nothing but a cluster of grey nodules exist in the medullary substance.*

Schüppell's giant-cells are commonly present—first recognised by Guttman and Goldenbaum (quoted by Eichhorst). Moreover, the bacilli now recognised as characteristic of tubercle are found in the diseased adrenal.

This conclusion is confirmed, as it was first suggested, by the concomitant tuberculous lesions of the bones and lungs which are often met with in this disease.

Symptoms.—The characteristic features during life which enable us to diagnose the above lesion of the adrenals are briefly as follows:—(1) gradually increasing weakness of the heart and other muscles; (2) liability to nausea and vomiting; (3) a peculiar discoloration of the skin.

Melasma.—The discoloration of the skin which occurs in Addison's disease varies considerably in different cases, not only in intensity, but also in tint. It has been variously described as yellowish or greenish brown, dusky, smoky, or as if stained by walnut-juice; the term "bronzed skin" was at one time commonly applied, but is not often very suitable. The colour is unlike jaundice or the sallow tint of lues of malaria or of lead, or the yellow hue of Addison's anæmia; it resembles that of a mulatto or a Moor. As in persons who have long resided in hot countries, the discoloration is deeper on the face and neck and on the backs of the hands than on the covered parts of the body generally; but it is very marked on the genital organs and about the pubes, and also in the axillæ, the navel, the nipples, and their areolæ. In extreme cases it may be universal, so that the patient looks like a half-caste, or like a figure in bronze; but it is never uniformly deep over all parts of the body.

There is no sharp line of demarcation between the affected parts and those unaffected; they shade off into one another. But in most cases there are present, in addition to the general discoloration, a few small black spots resembling pigmented moles, which have more or less defined outlines.

Parts of the body which are habitually rubbed are apt to become the seats of pigment. Thus a brown ring is often seen round each leg where the garters have pressed; or round the waist in women, where the petticoat strings have been tied. A case is quoted of a baker's boy, whose shoulders showed dark stripes corresponding with the bands by which his basket had

* I have observed the same thing, but only in cases of general or at least widely diffused tubercular disease—in which bronzing of the skin was absent or but doubtfully present; it would be very difficult to establish any connection between such cases and those of Addison's disease. Nevertheless, the affection is probably of a tuberculous nature; for whatever morbid changes are found in the lungs or in other parts of the body bear unmistakable marks of a tubercular origin.—C. H. F.

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been slung over his back. So, again, the application of a blister to any part of the skin is followed by the formation of a brown patch; and deep stains are left by eruptions attended with congestion of the skin.

The stains and patches just described are but an exaggeration of pigmentation that occurs in healthy persons, particularly in those of dark complexion, under the stimulus of pressure or irritants; and the diffused discoloration of the face, neck, and hands may be compared with what would naturally be observed in anyone exposed to the heat of a tropical sun. Some years ago, Dr Pavy had under his care a woman who suffered from the characteristic symptoms of Addison's disease; but the only melasma present was in the form of reticulated marks on her legs, such as are seen in persons who habitually sit before the fire, and are known as *ephelis ab igne*. When she died, disease of the adrenals was found at the inspection.

The discoloration of the genital organs, axillæ, and nipples may also be regarded as an intensification of physiological deposits of pigment; and the small black scattered spots are exactly like congenital moles. The increased pigmentation where pressure or inflammation (*i. e.* hyperæmia) has existed is also physiological. The pigment is deposited in the *rete* mucosum just as in health. What is abnormal is its amount. In a negro the disease could scarcely be recognised.

Each lip often shows a bluish-black streak along the line of contact with its fellow; and on the buccal mucous membrane irregular and ill-defined brownish stains may occasionally be seen looking like smears of ink. Dr Greenhow traced the irritation to protruding teeth; but they also occur independently of any such cause. Similar stains are found in the *lining* of the cheeks of Lascars. Another part of the mucous membrane on which stains occur in Addison's disease is the side of the tongue; they are of a purplish or inky hue, and are situated near the free margin.

The *conjunctivæ* always remain pearly white, contrasting with the dark colour of the surrounding parts of the face.

The microscopical appearances of the discoloured parts of the skin bear a close resemblance to those which may be observed in the darker races of mankind. The pigment consists of yellowish-brown granules. Its chief seat is in the deepest layers of the rete mucosum, close to the *papillæ*. Chemically it corresponds to melanin, the black pigment of the choroid and the hair, which possibly contains iron but is not directly related to the *red* series of pigments derived from hæmoglobin, as bilirubin and urobilin, nor to the yellow series which gives colour to the fat, to the corpora lutea and the serum. Dr Greenhow sometimes found traces of pigment in the more superficial scales of the epidermis, and likewise in the cutis. German observers also have detected pigmental connective-tissue cells within the papillary layer; but there is nothing unusual in the presence of colouring matter there, for it occurs in pigmentary moles.

It is still a question whether the internal organs are discoloured in cases of disease of the suprarenal bodies. Addison's work contains drawings of a mesentery, intestine, and omentum, over all of which numerous minute black spots were scattered. They were taken, however, from a doubtful case, and the pigmentation of the peritoneum may have been due to a former attack of peritonitis. The late Dr Carrington once observed the normal pigmentation of the pia mater covering the bulb much intensified in a case of Addison's disease.

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Asthenia, &c.—Next to the discoloration of the skin, progressive muscular weakness is the most striking and important of the symptoms of Addison's disease. The patient becomes more and more languid as it advances. He takes to his bed, and his prostration increases until he becomes unable even to sit up. If he is raised into a sitting posture, faintness and giddiness follow. The loss of flesh is often considerable; but, although the patient becomes thin, a certain amount of fat may still be found after death. Anæmia, though usually present, is not extreme.*

The action of the *heart* is remarkably weak, and the *pulse* very small and compressible. Breathlessness, palpitation after any muscular effort, frequent sighing or yawning, and persistent hiccough, are other symptoms more or less constantly present; and the patient may complain of pain in the loins or in the epigastrium.

The urine is free from albumen, and is not darker than usual.† The blood is normal or only slightly deficient in colour, and the temperature is unaffected except by phthisis or other complications.

Nausea, with retching and vomiting, is very rarely wanting, and sometimes the gastric symptoms are the most prominent of all.

Course.—The development of Addison's disease is gradual; and the order of the symptoms variable. Occasionally the skin becomes dark a long while before the general health begins to fail. Thus a young lady, whose case was recorded by Addison, had become "bronzed" for one year before her death, but appeared ill during only about four months; and a man who was under the care of Dr Robert Harris, of Southport, had been noticed by his wife to be getting darker for two years, but mentioned no other symptoms as present for more than six months. In the great majority of cases, however, the patient suffers from progressive asthenia for a considerable period before pigmentation of the skin becomes noticeable, and if the former should develop itself rapidly in a severe form, death may occur at a time when the latter is still entirely absent.

In a series of 228 cases, collected by Dr Greenhow, there were twenty-nine in which, when the patients died, the skin was either not bronzed at all, or to a scarcely appreciable extent. But in eighteen of these some other disease was likewise present, which probably was immediately concerned in bringing about the fatal issue. In each of the remaining eleven cases in which there was no such complication, the patient's illness had been of comparatively short duration; in one only had it lasted eight months, and in another four months. However, at Guy's Hospital one case has occurred in which there was no discoloration, but in which the patient had been ailing for twelve months before his death. Probably the most precise statement which we can make is that the skin is always dark when the other symptoms of Addison's disease have lasted more than a year.

Although the course of this remarkable malady is progressive, its rate of progress is by no means uniform. Dr Greenhow has pointed out that alternate exacerbations and remissions occur, which can only partially be traced to changes in the conditions under which the patient is placed. It is said that the depth of discoloration of the skin may alternately diminish and increase.

* The fact that asthenia rather than anæmia is the characteristic symptom of the disease seems to be not understood in Germany, judging from an excellent lecture by Professor Nothnagel reported in the 'Medical Press and Circular,' Feb. 12th and 19th, 1890.

† On the urine in Addison's disease, see Rosenstirn's paper ('Virch. Arch.,' lxi, p. 27).

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Death sometimes takes place very gradually. The mind may be clear to the last, or the patient may lie in a drowsy and semi-comatose state, from which, however, he may be roused to give pertinent though slow answers. In such cases the temperature falls considerably below normal. In some cases death is preceded by muttering delirium or coma. One of Dr Greenhow's patients had a convulsive fit, and lay for hours with closed jaws and rigid limbs; whenever he was touched, convulsive twitchings took place. In some cases, again, the fatal termination occurs unexpectedly, the patient having been apparently pretty well a few hours before. Thus, a young man some years ago was under the care of Dr Wilks for eczema which was getting better, when he was attacked with diarrhœa and vomiting; he became exceedingly prostrate, and died in less than twenty-four hours. Adrenal disease had never been suspected, but was found to be the cause of his death. A patient under the care of the present writer, who was in the hospital with Addison's disease, was so much relieved that he was about to go home, when he became suddenly worse and died in a few hours.

The *duration* of Addison's disease appears to be very variable. At least there are great differences in the length of time which elapses between the commencement of the symptoms and the death of the patient; but it must be admitted that the period at which the affection of the suprarenal bodies begins cannot be determined. Dr Wilks some years ago stated that the average duration of the cases which he had collected was eighteen months. One of the most rapidly fatal instances is that of a girl who came under Dr Greenhow's care, and who, although weak and supposed to be sunburnt, attended school until about a week before she died. The longest case was one on which the author made a *post-mortem* examination in 1865. The disease had been diagnosed by Sir Wm. Gull at the time when the patient was first admitted into the hospital in 1860; and he then said that his skin had already been dark for two years. In this instance, therefore, the disease had probably lasted not less than seven years.

Diagnosis—spurious and aberrant cases.—In describing the morbid anatomy of Addison's disease, we have implied that the affection is always of one kind. This, however, was not the opinion of Addison himself, who, in his original work, included four cases of cancer of the suprarenal capsules, and who at that time thought any affection completely destroying the organs would be capable of giving rise to the characteristic symptoms. It was Dr Wilks who first pointed out that in all genuine instances one particular morbid change in the capsules is found. He showed that in Addison's cancerous cases the proper symptoms were not really present. He also first defined the characters of the discoloration of the skin. The error into which Addison fell in regard to this point is much to be regretted, for it has led to much misunderstanding, and even to doubts as to the existence of the disease, which have not even yet been completely dispelled. Its reality, however, is surely proved by the mere number of cases that have been reported, many of which have been diagnosed during life in the most positive manner. In his lectures delivered before the Royal College of Physicians in 1875, Dr Greenhow was able to cite 183 recorded cases, in every one of which the special form of discoloration of the skin, and some at least of the constitutional symptoms, were present.

The cases which have been supposed to lead to conclusions adverse to

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those maintained by Dr Wilks and other modern supporters of Addison's views fall into two groups.

In the first group come those in which the capsules have been diseased *without giving rise to the characteristic symptoms.* We have seen that in some uncomplicated cases of Addison's disease death has occurred before the time at which bronzing of the skin usually develops; and in others a rapidly fatal issue has been due to some other malady, such as phthisis or Bright's disease, by which the constitutional symptoms of Addison's disease were masked. Apart from such cases, Dr Greenhow has been able to state that in every recorded instance of suprarenal disease without symptoms, the affection was really different from that which alone is known to be capable of causing characteristic effects. In most instances it was cancer. The adrenals, indeed, are liable to be the seat of primary malignant growths, as well as of secondary nodules. In one of our cases each of them was three or four times as big as the kidney, so that there was a large tumour observable during the life of the patient. It may seem strange that such an affection should not cause the symptoms of Addison's disease. But, as Dr Moxon remarked, there is an analogous fact that the most extensive cancerous growths in the liver often fail to produce jaundice.

Cases of Addison's disease have, however, been recorded by excellent observers, in which, instead of the usual changes, simple *atrophy of the adrenals* has been found: by Dr Wickham Legg ('St Barth. Hosp. Rep.,' 1874), Mr Davy ('Path. Trans.,' xxxiii, p. 360), Dr Goodhart (*ibid.*, p. 340), and Dr B. Fenwick (*ibid.*, p. 354, with table of cases). See also the same 'Transactions' for 1885, and the 'Clinical Transactions' for 1886.

The second group of cases which have been supposed to justify doubts as to Addison's discovery consists of those in which bronzing of the skin has been said to exist *without the characteristic suprarenal affection.* But it must be borne in mind that discolorations of the skin are from time to time met with, which may be mistaken for that of Addison's disease by an unpractised eye, although they are really altogether different. In one of the most curious of these the skin becomes, as it were, "piebald." Some parts are much darker than natural; others are entirely devoid of pigment, and the hairs upon them white. The white areæ always have very definite convex borders, and thus look as if they are encroaching upon the bronzed parts, which, on the other hand, shade off very gradually into the healthy skin. Thus the absence of pigment in some places is far more obvious than its excess in other places, and the affection is accordingly known by the name of "leucodermia." Addison, however, himself confounded it with what occurs when the suprarenal capsules are diseased; and in one of his cases, which appears to have been genuine, the skin did actually present an affection of this kind. All subsequent experience, however, has tended to show that leucodermia is altogether distinct from the discoloration which occurs in Addison's disease; and no other instance of it seems to have been associated with impairment of the general health. But not long ago a case of this kind was sent up to Guy's Hospital from a distant county as one of bronzed skin.

Old men and women living in poverty and dirt, and often infested with vermin, acquire deep pigmentation of the back, chest, and abdomen, which is increased if not caused by *pruritus senilis;* and this may resemble the melasma of Addison's disease. The roughness of the cuticle in such cases affords a distinction, in addition to the freedom of the face from discolora-

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tion. Dr Greenhow relates two or three cases of chronic phthisis in which pigmentation was present, somewhat resembling that of Addison's disease. The chloasma of women pregnant or affected with uterine disease, the discoloration of skin produced by malarious and tropical fevers, or by hepatic diseases, and even tinea versicolor, have each been mistaken for the bronzed skin of suprarenal disease. But the most extraordinary instance of perversity is that of a writer ('Virchow's Archiv,' 1870) who met with a case of sclerodermia, in which large patches of the skin in succession became deeply pigmented, sometimes in a single night. The patient, an old woman, had Bright's disease, and died of pneumonia. Her suprarenal capsules were healthy; but her physician, instead of seeing that his diagnosis was wrong, proceeded to frame an entirely new theory of Addison's disease, as a functional disturbance of the cerebro-spinal system. The truth is that one cannot accept unreservedly the diagnosis of Addison's disease by a physician who has not already seen other cases, and so made himself thoroughly familiar with its characters. Even in a large hospital the disease is not common enough to come under the observation of every student.

Ætiology.—Addison's disease occurs much more often in *males* than in females. According to Dr Greenhow the proportion is as 119 to 64.

The *age* of the patient is generally between twenty and fifty, but instances have been met with in children of five, eleven, and thirteen years of age.

Dr Greenhow thinks that this disease sometimes starts in a direct strain or blow on the back. He mentions seven such cases: one is that of a woman, who constantly asserted that she had never recovered from a strain in the back which had occurred while she was turning a mangle some years before; another patient had always had ill-health from the time when she fell downstairs; a third dated his illness from a fall through a trap-door.

In none of these instances was there any discoverable affection of the bones or ligaments of the spine. But in many other cases *vertebral disease* has been present. Dr Wilks first drew attention to this fact, and Dr Greenhow mentions eighteen instances. The lower dorsal or upper lumbar vertebræ have generally been the seat of the mischief, and there have always been abscesses in or near the psoas muscles. Very often a sinus has led from the abscess to the neighbourhood of the adrenals; or it has at least been clear that the disease spread continuously from one structure to the other. In some cases the vertebral disease was directly traceable to injury, in others it appeared to be of tubercular origin. But these two causes are far from being mutually exclusive.

Pathology.—What relation can be discovered between the morbid change and the symptoms that characterise the disease? A plausible hypothesis is that the ganglia and branches of the sympathetic nerve, which are so intimately connected with the adrenal bodies, are involved in an inflammatory process, starting from them. Dr Habershon in 1863 dissected out the semilunar ganglia in a typical case, and found that they and their branches of nerve were surrounded on the side on which the suprarenal body was more severely affected by dense fibrous tissue. Similar observations have since been made by other pathologists, both in England and abroad. The semilunar ganglia have been found enlarged and reddened; and under the microscope their cells have appeared opaque and

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granular, and remains of hæmorrhages into their substance have been discovered. The fibres of nerve-trunks embedded in adhesions have also been shown to be in a state of fatty degeneration (Tuckwell, 'Path. Trans.,' 1868). Again, one or two cases have been recorded to prove that the symptoms of Addison's disease may be caused by affections of the semilunar ganglia independently of any primary change in the suprarenal bodies. Sir William Jenner, when President of the Pathological Society, mentioned such an instance as having come under his observation (cf. *infra*, p. 628); and another is reported by Drs Barlow and Coupland ('Path. Trans.,' 1885).

What we know of collapse as a result of a sudden shock to the semilunar ganglia accords with the opinion that the extreme debility of Addison's disease may depend upon chronic changes in these important nervous centres; and the nausea and vomiting are still more readily accounted for.

No such obvious explanation suggests itself of the peculiar pigmentation of the skin. This has been attributed by Jaccoud to irritation of the vaso-motor nerves, but on no sufficient evidence.* The medulla of the adrenals contains dark pigment, and this has been connected with Addison's melasma; but if there were any retained pigment circulating in the blood, it would surely appear in the urine and other excretions. (See Dr Creighton's paper in vol. xiii of the 'Journal of Anat. and Phys.,' and Dr MacMunn's interesting account of the pigments extracted from the adrenals, in the 'Brit. Med. Journ.' for 1888, i, 233.)

It is doubtful whether Addison's disease is of nervous origin at all. The function of the ganglia of the solar plexus is quite unknown, and in many cases chronic inflammatory contraction of the post-peritoneal tissues in that neigbourhood is found without any of the symptoms above mentioned being produced during life. The function of the adrenal bodies is at present an unsolved mystery. They are richly supplied with nerves, and the medulla is darkly pigmented, while the cortex is arranged somewhat like a tubular gland. There can, of course, be no true secretion without a duct; but it might be thought that the adrenals represent a no longer active organ. In the adult, at all events, they seem to be comparatively atrophied, and if their meaning in the economy is ever discovered it will probably be found that, like the thymus, they are connected with the physiology of fœtal life.†

* It is probable that under normal conditions pigmentation, like other organic processes, is kept in check by inhibitory trophic nerves; and if in disease their controlling influence is withdrawn, we might expect pigment to be laid down in excess, or at least in quantity altogether disproportionate to the intensity of its exciting cause. Such an explanation involves more than one hypothesis, and it may appear far-fetched; but it is doubtful whether any simpler one will account for the facts.—C. H. F.

† Recently attempts have been made to connect Addison's with Hodgkin's disease because the adrenals and lymph-glands are both "ductless glands"! And one writer argues that on developmental grounds the adrenals are "ductless glands." This meaningless and misleading term should be given up, and the word gland restricted to secreting organs.

What is taught by embryology and comparative anatomy is that the adrenals are composed of two distinct parts which have coalesced as cortex and medulla, both apparently mesoblastic, but the latter probably nervous and ultimately epiblastic in origin, while the former is possibly hypoblastic and certainly more important in the fœtus than after birth.

It is a singular fact, first observed by Hewson, that in an anencephalous fœtus the adrenals are usually absent or imperfectly formed. See the text-books of Rokitansky, Klebs, and Ziegler, Lomer's paper in the 98th vol. of 'Virchow's Archiv,' also an interesting thesis by Dr K. Biesing (Bonn, 1886) with account of thirteen dissections, in which the sympathetic ganglia and plexus did *not* share the atrophy of the adrenals.

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Many attempts have been made to throw light on the disease by experiments on animals. Extirpation of the adrenals, performed many years ago on rats, failed to lead to death or definite morbid symptoms. But Nothnagel states that by setting up chronic inflammation by the introduction of irritants in rabbits he has succeeded in producing permanent pigmentation of the skin.

In an interesting paper published in the 'Lancet' for February 6th, 1886, Dr Churton, of Leeds, after relating a typical case of Addison's disease in a youth of nineteen, and five others of non-specific lesions of the adrenals without symptoms, discusses the possibility of senile atrophy of these organs producing more or less modified clinical effects. But the patient, aged seventy-two, whose case he describes, may not impossibly have had the specific anatomical change of his adrenals; and, as he rightly states, these organs may be found of normal size and structure in old people.

Prognosis and treatment.—Hitherto no positive proof has been given that recovery from this disease ever takes place. But it is certain that many patients, after having been kept in a hospital for a long time, and perhaps after having been admitted over and over again, have been lost sight of. This might well have happened in the case already mentioned, of a young man in whom Addison's disease was diagnosed in Guy's Hospital five years before the time he died; for the morbid appearances which were found in the adrenals seemed to show that they had been destroyed for a long time. A case of bronzing of the skin in a young woman whose adrenals were found diseased after death was recorded by Dr Kirby, with the history that similar symptoms had appeared four years before her death and had completely disappeared.

The treatment of Addison's disease may unfortunately be summed up in a very few words. The vomiting must be combated by appropriate medicines, but these are too often ineffectual. For the debility and prostration tonics and stimulants are obviously indicated; but they generally fail to do any good. The use of iodine (whether internally or as an application to the lumbar regions) might possibly lead to the subsidence of those inflammatory changes which have been shown to occur in the connective tissue round the semilunar ganglia and branches of sympathetic nerve; and if these changes are the cause of the symptoms such treatment might do good.

Other affections of the adrenals.—These organs may be the seat of miliary tubercles in cases of general tuberculosis; or of hæmorrhage; or of cancer, either by extension from the kidney, lumbar glands or vertebræ, or as a secondary deposit; or of lardaceous transformation; or of embolism; or of secondary pyæmic abscess. In a remarkable case which occurred in a child under the writer's care there was primary sarcoma of each adrenal. Lastly, they may apparently become atrophied without preceding disease. But none of these pathological states are of clinical significance. In a text-book of medicine Addison's disease is the only lesion of the adrenals which demands a separate place.

Diseases of other abdominal viscera.—One of the greatest helps which morbid anatomy has given to practical medicine is the discovery that diseases do not affect organs and tissues indiscriminately. Acute idiopathic inflammation, suppuration, chronic fibroid inflammation with contraction,

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tubercle, primary and secondary cancer—each of these pathological processes is modified by the organ affected, and has a special predilection for certain of the tissues, with more or less complete incapacity of affecting others.

Of this rule we have had frequent examples in the study of diseases of the brain, the lungs, the liver, and the kidneys. In the remaining viscera, the pancreas, spleen, ovary, and testis, there are no special diseases like pneumonia or Bright's disease, acute atrophy of the liver, or morbus Addisonii, peculiar to the organs and associated with more or less constant clinical symptoms. Their morbid lesions belong, therefore, rather to pathological anatomy than to clinical medicine; but a brief notice of them may be conveniently introduced here before leaving the subject of abdominal diseases.

DISEASES OF THE PANCREAS.—Like the salivary glands which open into the mouth, the important abdominal gland (*Bauch-speicheldrüse* of the Germans) which so closely resembles them in structure and function is little liable to ordinary inflammation, and there is not known to be any zymotic affection of the pancreas answering to that of the parotid and submaxillary glands in mumps.

Wilks and Moxon describe suppuration of the pancreas in the later stages of enteric fever, which we may compare with the parotid buboes mentioned before as sequelæ of that disease (vol. i, p. 148).

The gland is indurated in cases of chronic heart disease, like the kidneys and the spleen.

Atrophy of the pancreas has been found after death in cases of diabetes, and some pathologists have supposed that this may be more than a coincidence.

Primary cancer of the pancreas exists, but is in reality a rare disease. In this want of aptitude for malignant growths the pancreas resembles other racemose glands, with the striking exception of the mamma (due perhaps, as Dr Creighton has suggested, to its functional activity being often interrupted), and differs from the testis. What is usually described as "cancer of the head of the pancreas" begins in most cases in the epithelium of the pancreatic duct (or in that of the common bile-duct or duodenum), and gradually involves the glandular structure. Hence it is almost always accompanied by jaundice, to which enlargement of the liver from secondary growths is subsequent. So that clinically, when there is no reason to suppose the primary cancer to be in the stomach or rectum, malignant disease of the liver with early jaundice may be most probably ascribed to primary cancer of the head of the pancreas.

Often the structure of the new growth is cylindroma or duct-cancer, but sometimes it is of the ordinary glandiform type, as in one exceptional case, lately in Guy's Hospital (1887), in which the autopsy showed primary cancer of the splenic end of the pancreas. Like other epithelial structures, the pancreas is very rarely the seat of secondary cancer. Occasionally primary cancer of the body or "tail" of the pancreas occurs, as in the case just cited. The symptoms, if any, are those of post-peritoneal cancer.

When *lymphoma* or lymphosarcoma affects the pancreas, as is occasionally seen, the new growth probably begins in the small lymph-glands, which are found in the pancreas as well as in the parotid. *Tubercle* appears never to affect the salivary glands or the pancreas.

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Calculi have been found obstructing and dilating the duct of Wirsung, like those found in that of Stensen. In one instance numerous calculi were found, of which several are preserved in the museum of Guy's Hospital (1992[50]); and others were analysed by the late Dr Golding Bird, and found to consist of phosphates of lime and magnesia, with oxalate of lime, but without carbonate.

When from any cause obstruction has occurred to the passage of pancreatic secretion into the duodenum we should expect, on physiological grounds, that the digestion of fatty matters would be suspended or at least impaired; and in several cases, notably that of a young woman, a patient of Bright's, whose symptoms during life were "emaciation, diarrhœa, and the passage of fatty stools," the pancreatic and biliary ducts were found after death to be occluded by a cancerous growth. But these cases are exceptional.

In some cases of abscess in the head of the pancreas, the pressure thus caused on the portal vein has led to extreme and rapidly recurrent ascites (see a case reported by Dr Musser in the 'American Journal of Medical Sciences,' April, 1886).

Certain cases of severe and acute symptoms, leading rapidly and sometimes suddenly to a fatal issue, have been recorded by Clässen, Klebs, and other observers, in which *hæmorrhage* into the pancreas has been the only symptom found after death. In some of these, hæmorrhage was due to purpura or could be ascribed to chronic cardiac disease, in others to violence, and in others it was apparently spontaneous (see a case reported in the 'London Medical Record,' 1878, p. 69, from a paper by Dr Hiltz, in the 'Correspondenzblatt f. Schw. Aerzte,' November, 1877). It is supposed that the effused blood (or the injury which caused it) pressed on the solar plexus and produced fatal effects on the heart, as in Sir Astley Cooper's famous case, and in Goltz's 'Klopfversuch.' Pancreatic hæmorrhage is often unaccompanied by clinical symptoms; but cases are recorded of epigastric pain, collapse, and subsequent peritonitis, which simulated acute intestinal obstruction or perforation of the stomach, and were found after death to depend on hæmorrhagic inflammation or suppuration of the pancreas (see an excellent monograph by Dr Fitz, of Harvard, published at Boston in 1889).

As in the kidney, the testes, and the mammary gland, retention-cysts are occasionally found in the pancreas. We have already seen how these may affect more than one organ simultaneously, most often the liver and kidneys (*supra*, p. 505), sometimes the brain as well (*ibid.*, note), and in one case the writer met with multiple pancreatic cysts in a fatal case of simple cyst of the cerebellum ('Path. Trans.,' vol. xxxvi, p. 17). Their coincidence, however, was probably a mere accident.

DISEASES OF THE SPLEEN are remarkable in being all secondary to, or forming parts of, other morbid processes. It is very rarely the seat of acute suppuration, of chronic fibroid inflammation, or of parenchymatous diffuse splenitis.

Rare instances, however, occur of fatal illness, which is found at the autopsy to have been due to the formation in the spleen of a single abscess, for which no cause can be discovered, and which may reach a very considerable size. A case of this kind was recorded by Bright in the 'Guy's Hospital Reports' for 1838; about half of the substance of the spleen was involved, and there was an opening into the colon. In another case, a sloughing

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abscess communicated with the stomach; but it is perhaps doubtful whether the original seat of the suppuration in this instance was the spleen itself. Dr Caton, of Liverpool, has recently recorded a case which ended favourably ('Brit. Med. Journ.,' 1888, i, 586).

The symptoms of splenic abscess are obscure, consisting chiefly of pain in the upper part of the abdomen, frequent vomiting, rigors, pyrexia, and emaciation. A more or less distinct fulness or induration may also be discovered in the left hypochondrium.

During life it would be impossible to distinguish cases of this kind from others in which suppuration occurs outside the spleen, but immediately in contact with it, and which are probably much more frequent. In the 'Guy's Hospital Reports' for 1874 nine or ten such instances were recorded by the author. In some of them there was a clear history of a blow or a fall having been the starting-point of the disease; in others a chronic ulcer of the stomach seemed to have set up inflammation outside that organ. One of the most remarkable among them was the case of a man aged thirty-seven, whose case has been already mentioned in the section on circumscribed abscess in the peritoneal cavity (*supra*, p. 311).

In 1870 the author was consulted by a gentleman aged fifty-six on account of a rounded, tender, and rather painful swelling, occupying the left side of a very protuberant abdomen. It seemed clearly to be an enlarged spleen, and this was confirmed by his pallid appearance, and by the fact that there was a decided excess of leucocytes in the blood. However, the skin over the tumour gradually became reddened, hot, and indurated, and at the end of a month Mr Durham made a puncture with a small trocar, and drew off some pus. For some months afterwards there was a discharge of matter from the opening, but at last it closed, and the swelling disappeared. Still no doubt was entertained that the case had been one of abscess of the spleen. But ten months later a second large tumour formed in the right side of the abdomen. This also was opened, and a great quantity of very fœtid pus escaped. The patient was now exceedingly prostrate and ill, but he again recovered, and in 1876 was in excellent health. Probably the real seat of both abscesses was between the layers of muscles in the walls of the abdomen.

Another interesting case was that of a woman who was admitted into Guy's Hospital eleven days after having crossed the Channel on a very rough day: she had been very sea-sick, and had also been thrown out of her berth upon the cabin floor. A large tumour rapidly formed in the epigastrium, and in the left hypochondrium there was a second mass in the position of the spleen. It appeared not improbable that in the first instance blood was effused over the surface of the stomach, and that the process of suppuration was secondary. In three other cases a sub-diaphragmatic abscess perforated the pleura and set up fatal empyema.

In all fevers the spleen is the seat of congestion, and is found after death soft and more or less swollen. In typhus, acute pyæmia and tuberculosis, scarlatina and pneumonia, it is not obviously enlarged, but in enteric fever it is so, and can be sometimes detected by palpation as well as percussion during life (cf. vol. i, p. 138).

The congested and swollen spleen of anthrax has been described in the first volume. As the result of intermittent fever the spleen becomes much larger, perhaps because of the frequency of the attacks, and at last forms the characteristic ague-cake (vol. i, p. 329). Pigmentation of the spleen

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is part of the result of malaria, which was first observed by Dr Bright (*ibid.*, p. 330).

The passive congestion produced by general systemic venous stasis, particularly in cases of cardiac disease, leads to hardening but not to swelling of the spleen; but that which is due to portal congestion, and particularly to cirrhosis of the liver, leads to its decided enlargement, except when this is mechanically prevented by a capsule thickened from chronic perisplenitis.

Emboli produce the well-known wedge-shaped blocks (*infarctus*), first deep red, then pale with a red border, then completely pale, and lastly shrivelling up into a deep scar. When the emboli, however, are septic, suppuration occurs in the spleen. Hence a swollen and tender spleen with pyrexia and a cardiac bruit is a certain sign of ulcerative endocarditis.

Infective abscesses from general pyæmia are also met with in the spleen, but less frequently than in the liver.

Miliary tubercles are extremely common, especially in cases of general acute tuberculosis in children; and caseous tubercles also occur, but they are probably always secondary.

Lardaceous disease is frequent, either as a uniform infiltration, or affecting the Malpighian bodies only, and producing the well-known "sago-spleen." No local effects follow, and no symptoms result.

Gummata are occasionally found, and have to be carefully distinguished from the fibrous scars of old emboli. Deposits of lymphatic new growths are not infrequent, and will be described in the following chapter under Hodgkin's disease.

Primary malignant disease of the spleen is unknown, and even secondary nodules, whether of sarcoma or true carcinoma, are extremely rare.

Hydatids also are less common in this viscus than in the lung, kidney, or brain; when present, echinococci have often been also found in the liver.

In children the spleen is large and typically healthy. As age advances it atrophies like other lymphatic organs—following the fate of the thymus, the tonsils, Peyer's patches, and the lymph-glands themselves.

Hypertrophy of the spleen is the only condition of this organ which possesses clinical as distinct from pathological interest. It is almost always associated with anæmia, and often with leuchæmia, when it constitutes part of the remarkable disease to be described in the next chapter (p. 623).

A spleen enlarged from ague or any other cause is liable to stretch its attachments and become loose in the abdomen. Such moveable or "floating spleens" are of clinical interest as forming abdominal tumours. They are much more rare than floating kidneys, and usually more readily recognised. They are more common in women than in men (see a short monograph by Dr F. C. Shattuck, Boston, 1878).

The diseases of the abdominal viscera which remain, the testes and the ovaries, are by the conventional arrangement in use assigned to the departments of surgery and obstetrics respectively. The best account of their morbid anatomy is therefore found in treatises on pathology, and of the corresponding clinical disorders in those on practical surgery and diseases of women. But the student must not forget that the divisions of our art have no counterpart in nature, and that the physician must not neglect the diseases of any viscus.

Of the diseases of the OVARIES, the only ones which come under the

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scope of a treatise on general medicine are the cystic tumours of the ovarium and the parovarium, which have been already described, so far as is necessary for their diagnosis from encysted ascites and other abdominal tumours (p. 325); and the functional disturbances of these organs which are sometimes associated with epilepsy, hysteria, and hystero-epilepsy.

The diseases of the TESTIS are acute inflammation from injury, "metastatic" or "sympathetic" swelling from mumps or from renal calculus, epididymitis from gonorrhœa, tubercular (or "strumous") orchitis, syphilitic orchitis with gummata and chronic fibroid atrophy, cystic disease, enchondroma, and medullary cancer.

It is important for the physician to remember that the testes are abdominal organs which have escaped from the general peritoneal cavity, and they should be examined in doubtful cases with the same care as the liver or spleen. The discovery of epididymitis or *fungus testis* will often throw light on the nature of hæmaturia and purulent pyelitis; while the presence of sarcocele may point to syphilis in other organs, or to malignant disease of the lumbar glands.

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DISEASES OF THE BLOOD

ANÆMIA

"The blood the virgin's cheek forsook,
A livid paleness spread o'er all her look."
POPE.

ANÆMIA—*Its general characters—Amount of blood—Number of red corpuscles—Amount of hæmoglobin—Changes in the blood-discs—Anæmic murmur—Dyspnœa—Fatty degeneration of the heart—Other symptoms—Cause and classification—Symptomatic or secondary and idiopathic or essential anæmia.*

LEUCHÆMIA—*History—Anæmia splenica—Definition and characteristic symptoms—The spleen in leuchæmia—Physical signs of an enlarged spleen—The blood in leuchæmia—The other organs—Ætiology—Diagnosis—Pathology, course and treatment.*

HODGKIN'S DISEASE—*Anæmia lymphatica—History—Ætiology—Morbid anatomy and pathology—Course, prognosis, and treatment.*

Primary tuberculosis of the lymph-glands—Syphilitic adenitis—Lymphosarcoma.

CHLOROSIS—*Its relation to menstruation—to imperfect development of the heart and vessels—The blood in chlorosis—Symptoms—Prognosis—Treatment.*

IDIOPATHIC ANÆMIA—*Grave or pernicious anæmia—History—Name—Age and sex—Antecedents—Course and symptoms—Ætiology and diagnosis—Pathology—Prognosis—Treatment.*

THE ancient humoral pathology assigned most diseases, at least in their origin, to *dyscrasia* or ill-tempering of the four humours of the body. When "black bile" was found not to exist, and *pituita* or phlegm was ascertained to be restricted to mucous surfaces, "overflow of the bile" and "impurities of the blood" were still the easy explanations of many obscure symptoms.

Even in recent times, fevers, syphilis, pyæmia, rheumatism, and cancer have been still called blood-diseases. But for this there is no adequate reason. There is no evidence that the blood of a person who is the subject of malignant disease behaves differently to physical, microscopical, or chemical tests from that of one in health; at the utmost it is the chief channel of infection in cases of sarcoma, as the lymph is the chief channel in epithelial carcinoma. Nor are the specific diseases, pyæmia, syphilis and the infectious fevers, specially disorders of the blood. They affect the whole body. The poisons or microphytes which are their material basis circulate no doubt with the blood, but the circulation is only the carrier and not the primitive seat of the contagion. The spirillum of relapsing fever, the bacillus of anthrax, are the essence of the disease, and no doubt

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circulate in the blood; but they are found in the solid organs as well, and ought to be compared to the mineral which is carried by the blood to the tissues in cases of arsenical poisoning, or to the *filariæ* which are transported by the same means to various parts of the body. Uræmia, we saw, may be probably explained as a chemical "intoxication" of the blood with retained urinary constituents or their products, but here again the poisoned blood owes its qualities to a lesion of the kidneys.

The only "diseases of the blood"—maladies, that is, of which we cannot trace the seat further back than to the blood—are the following:

(1) Those in which the red blood-discs are diminished in number. Their diminution leads to the condition long recognised as *Anæmia*.* Their shape, size, and colour may also be altered, as we shall see, and in one remarkable form of disease their scarcity is accompanied by an absolute increase of the colourless corpuscles. The amount of hæmoglobin in each disc as well as in the total blood may also be diminished; and perhaps the amount of albumen and other constituents of the serum may be altered—but the small number of red corpuscles is the constant and demonstrable morbid condition.

Of *plethora*, the assumed opposite condition of too much instead of too little blood, we have no knowledge. Even in anæmia there is no proof that the mass of the blood is diminished; water is rapidly absorbed and makes thin blood, but the vascular system is as full as before. Nor is it ever over-full. If excess of blood is injected into the veins of animals the foreign corpuscles are rapidly destroyed, nor do we find the number of corpuscles or the amount of hæmoglobin increased in any form of disease. What used to be called plethora was only local congestion; and if general plethora exist at all, it is a condition not due to there being too much blood in the vessels, but to the distal vessels being contracted and the ventricles strong, *i. e.* to a condition of high blood-pressure in the arteries.

(2) *Hæmorrhagic diseases*—those in which the blood readily escapes from the vessels, without mechanical cause being demonstrable. Their true pathology is still obscure. More or less anæmia is almost always antecedent as well as consequent to the hæmorrhage; but while in the former group of blood-diseases we shall see that anæmia frequently leads to secondary hæmorrhage, here hæmorrhage is the primary and constant and anæmia the secondary symptom. This group will form the subject of the next chapter, and the present one will be devoted to anæmia.

General account of anæmia.—Under various circumstances the tissues of the human body, which normally have a ruddy hue, from the blood circulating through them, become pale as a result of deficiency or pallor of the blood. In this, as in other cases, there is difficulty in fixing the boundary-line between health and disease. Some persons—nearly all the members of certain families—are naturally pale, and remain so, however favourable the conditions under which they live. Others lose their colour from time to time if they reside in large towns, work at sedentary occupations, or keep late hours; but a rosy complexion returns soon after they go back to the country, under the influence of sunlight, fresh air, and exercise. We may therefore conclude that among those who constantly exhibit pallor of

* Attempts have been made to improve upon this classical word, by substituting *oligæmia* or *spanæmia*. But it is a mere quibble to say that there is not complete absence of blood in anæmia. We say a person is bloodless in English, and the Greek privative is used like corresponding prefixes in other languages.

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countenance, but who are all their lives confined to dark workshops, or cellars, or mines, some, at least, would assume a very different appearance if they could spend their time in light, well-ventilated rooms, or in the open air. Again, there are others who owe their colourless, sallow appearance to habitual excesses, especially to the practice of onanism, or to premature sexual indulgence. Excessive smoking, and smoking at too early an age, are also important causes of anæmia; though whether they act more by disturbing the digestive organs or the heart is uncertain.

Again, the skin becomes pale as the result of many diseases, both acute and chronic, particularly enteric and other fevers, acute rheumatism, Bright's disease, and the cachexia of syphilis, ague, and plumbism.

All these may be classed together as instances of—

(1) Anæmia from deficient formation of hæmoglobin; the extreme type is the pallor of starvation.

(2) Anæmia from loss of blood—as traumatic hæmorrhage, flooding, menorrhagia, bleeding piles; or loss of pus, as in the anæmia which accompanies lardaceous disease; or other losses, of which excessive lactation is the most frequent example.

(3) Anæmia as the result of fevers, rheumatism, Bright's disease, cancer, tubercle, syphilis, and malaria.

(4) Toxic anæmia, from lead, mercury, &c.

(5) Lymphatic and splenic anæmia, including leuchæmia.

(6) Chlorosis.

(7) Idiopathic or essential anæmia, the gravest and most pernicious form.

Distribution.—Anæmia, from whatever cause, is characterised by *pallor*, not only of the skin, but of all visible mucous membranes. The tint varies widely in different cases, and sometimes, instead of being white like marble, it is decidedly yellowish, like wax. The hands, the fingers, the finger-nails, show the change as distinctly as the countenance, or even more so, for in those persons who have the smaller vessels of the cheeks dilated and varicose there may remain a crimson patch on each side of the face, contrasting strangely with the rest of the complexion. Moreover, the gums and *mucous* membrane of the lips do not change colour when the cheeks flush with excitement. The tongue, the fauces, the lachrymal carunculæ, are all more or *less* white and waxy-looking. In the dead body, the amount of blood in the deeper tissues and in the various organs is found to be very deficient; the liver and the kidneys look like wax, while the heart and the great vessels contain only small loose shreds of coagulum and a little thin pink fluid.

Estimate of amount of blood.—The evidence derived from inspections after death seems to show that the total volume of the blood is greatly diminished in the more severe cases of anæmia, just as there is obvious wasting of other tissues; but unfortunately it is difficult to obtain clinical proof of this fact, and almost impossible accurately to estimate the degree of deficiency. The only method seems to be that proposed by Quincke, of first counting (by a process presently to be described) the proportion of red discs contained in the patient's blood, then transfusing into his circulation a known quantity of healthy blood, and soon afterwards again counting the red discs in a fresh specimen taken when the new blood and the old may be supposed to have become thoroughly mixed together. A simple formula gives the total volume of blood which was present in the patient's body before the operation. In two cases of "pernicious anæmia" Quincke estimated by this method that the blood formed only 4 or 5 per cent. of the

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body weight instead of 7 or 8 per cent., which is supposed to be about the normal proportion. Such experiments, however, can rarely be carried out accurately enough to yield trustworthy results.

Amount of corpuscles.—Whatever diminution there may be of the total volume of the blood in anæmia, diminution of the red corpuscles can be estimated with comparative facility.

The "numeration of blood-corpuscles" is one of the most important advances in practical medicine that has been made within the last twenty years.* Originally suggested by Vierordt, it has been simplified by subsequent investigations, and especially by Malassez (1872), Potain, Hayem, and Gowers (1877), until it has now become a very easy matter. The principle is to dilute to a definite extent a measured quantity of blood, and then to count the number of red discs contained in a certain volume of the mixed liquid. The method of employing the *Hæmocytometer* of Gowers, a modification of the *compte-globules* of Malassez, is as follows: (1) A small pipette, holding exactly 995 cubic millimetres, is filled with the diluting solution, which is then poured into a small glass jar or mixing vessel. A good solution, which leaves the corpuscles in a state favourable for observation, consists of sulphate of soda in distilled water, of sp. gr. 1025. (2) The patient's finger is pricked with a lancet, so that a drop of blood escapes without much pressure. Five cubic millimetres of blood are then taken up by a fine capillary tube, graduated for the purpose, and are blown into the diluting fluid in the vessel. If there is a difficulty in getting exactly the right quantity of blood into the tube, the best way is to take up a little more than enough, and then to let the excess escape into a soft cloth. (3) The contents of the mixing-jar are well stirred up with a glass rod. (4) A drop of the mixed liquid is placed in the centre of a cell excavated in a microscopic slide. The cell is exactly one fifth of a millimetre deep, and its floor is ruled in tenth of millimetre squares. The slide rests on a metal slip, to which two springs are attached. (5) A cover-glass is next laid over the cell, in contact with the liquid in it; the springs are brought over the edges of the cover-glass, and keep it in position with a pressure which is always uniform; the slide is placed horizontally on the stage of a microscope, and this is focussed upon the squares in the floor of the cell. (6) In a few minutes the red discs are found to have settled down upon the squares by gravitation. The average number in a square is now counted, and this, multiplied by 10,000, gives the number contained in one cubic millimetre of the blood. The average number in normal blood is believed to be 5,000,000 in males and 4,500,000 in females. It is usual, however, to state the "corpuscular richness" of blood as a decimal fraction of the normal richness, this being taken at 5,000,000 to the cubic millimetre. The decimal figure may be obtained by dividing by 5 the number of corpuscles contained in 10 squares. Thus if the number in 10 squares is 332, the "corpuscular richness" is ·66.

Amount of hæmoglobin.—The deficiency of red discs in proportion to plasma affords, however, an incomplete measure of the diminution in the amount of hæmoglobin in anæmia. For all practical purposes this may be estimated with sufficient accuracy by an apparatus called a *Hæmochromometer* or *hæmoglobinometer*, for which we are again indebted to Malassez ('Arch. de Phys.,' 1877) and Gowers ('Clinical Transactions,' 1879). It consists of two glass cylinders of equal diameter, which are placed side by

* The most complete and elaborate series of measurements yet made have been published by Laage, of Christiania ('Die Anämie,' 1883).

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side upon a small wooden stand. One of them is closed, having been filled with glycerine jelly, coloured by a mixture of carmine and picrocarmine of ammonia, so that its tint is that of blood diluted with water, in the proportion of one part of blood to a hundred of water. The other cylinder is graduated in such a manner that a space equal to two cubic centimetres has 100 divisions. It is open, and a little distilled water is poured into it. Some of the blood, of which the hæmoglobin is to be estimated, is now taken up by a capillary pipette, marked for twenty cubic millimetres; this quantity is carefully measured off, and is conveyed by the pipette into the open cylinder, which is quickly shaken, so as to secure the admixture of the blood with the water before coagulation has had time to occur. Distilled water is then added, drop by drop, by means of a pipette-stopper, until the tint of the diluted blood becomes the same as that of the standard in the closed cylinder. The degree of dilution, when this point is reached, indicates the percentage proportion of hæmoglobin in the blood under examination, as compared with that of normal blood. The best way of observing the tint is to hold the apparatus up between the eye and a window, so that the light passes directly through the cylinder.

The value of hæmoglobin for each red disc may of course be obtained by combining the results yielded by the hæmocytometer and by the hæmochromometer. Thus the blood of an anæmic patient of Dr Gowers contained 60 per cent. of corpuscles, but only 30 per cent. of hæmoglobin; the average amount of hæmoglobin contained by each disc was therefore $\frac{30}{60}$ths, or one half of the normal. Not unfrequently it falls as low as one third.

In such cases the deficiency of colouring matter in the blood of an anæmic patient is obvious to the naked eye when the finger is pricked to allow a drop to be taken for investigation. One sees at a glance that it is pale, thin, and watery, exactly as if it had been diluted.

Corpuscular changes.—More or less marked alterations in the microscopical appearance of the red discs of the blood may be made out in many cases of anæmia. Thus, the average diameter of a normal corpuscle being 7·5 μ (1 μ = 0·001 mm.), the average diameter in anæmia has been found by Hayem and by Eichhorst to be reduced to 7 μ, 6·5 μ, or even 6 μ.* Moreover, red discs are sometimes present which are far smaller than any that exist in health, their diameter being from 6 μ to 2 μ; these have been termed *microcytes*. Contrasting with them, however, there may be others which are larger than normal, having a diameter of 12 μ; they have been called *megalocytes*. Quincke, Eichhorst, Bramwell, and many others have found red discs presenting curious irregular forms, being oval, elongated, curved, or drawn out into pointed processes. Another change, that seems to have been first recognised by Drs Mackern and Davy, then students at Guy's Hospital, is that the hæmoglobin (or zooid) is sometimes separated from the substance of the corpuscle (or œcoid of Brücke), forming a rounded body, which had been mistaken by previous observers for a nucleus. For these conditions the term *pœkilocytosis* has been needlessly coined. They were at first supposed to be peculiar to a special affection, which will be described as idiopathic or pernicious anæmia; but they have since been discovered in cases of secondary anæmia from phthisis, cancer of the stomach, and chronic Bright's disease.

* The thousandth of a millimeter (μ) is often called a micromillimeter, but according to the usage of physical science it is a *micrometer*, and the *millionth* of a millimeter a micromillimeter.

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Murmurs.—Among the effects of anæmia, one which attracted great attention from the earlier auscultators was the production of abnormal bruits in the heart and in the great vessels. When the stethoscope is lightly laid upon the patient's neck just above the clavicle, there is often heard a loud and harsh murmur, which is continuous, but varies in intensity, corresponding with the respiratory movements. This venous humming sound was termed by Bouillaud "bruit de diable," the "diable" being a toy common in Paris in 1835, which made a similar noise. The seat of this venous hum is in the internal jugular vein; it is usually louder on the right side than the left; and though rarely absent in chlorosis, is sometimes not audible in other, and even in extreme, cases of anæmia, particularly in men. According to the theory which refers all murmurs to the formation of a "fluid vein," the *bruit de diable* may be accounted for, if we admit that in anæmia the volume of the blood is diminished. The jugular veins pass through dense cervical fascia. Consequently, when in an anæmic patient the veins in general shrink and adjust themselves to the small quantity of fluid circulating through them, this part remains unaltered in size, and forms a relatively wide space, within which the streams that enter it are thrown into vibration. The explanation is corroborated by the fact that in many healthy persons one can make a *bruit de diable* by pressure with a stethoscope in the neck.

An "anæmic murmur" of another kind is systolic in rhythm, and is heard over the heart and the main arteries. It is loudest at the base, and is usually traceable along the pulmonary artery rather than the aorta. This murmur can be accounted for in the same way as the other. The trunks of the two main arteries are supposed to be dilated compared with the orifices through which the blood enters them.

Whether an anæmic murmur is ever localised at the apex is uncertain. That an apical systolic bruit may be "functional" there is no doubt, but such a functional murmur may be heard without anæmia being present. It probably in most cases depends on temporary mitral incompetence from want of vigorous ventricular contraction (cf. p. 75).

None of these murmurs are necessary for the diagnosis of anæmia; but they are of considerable importance, because they would almost certainly be regarded as signs of organic disease of the heart or of the great vessels if their origin were not understood. This is especially the case with the basic systolic murmur, which often has a rough harsh quality suggestive of anything rather than a functional origin. Indeed, when there are other reasons for suspecting an organic affection of the heart, as, for instance, when the patient has had rheumatic fever, it is often very difficult to determine whether such an affection may not be present, and whether the anæmia may not after all be merely one of its effects instead of being the primary disease.

An anæmic murmur is never diastolic or systolic; it is basal rather than apical, and pulmonary rather than aortic in position; and it is usually accompanied by arterial and venous murmurs, and unaccompanied by symptoms of organic cardiac disease.

Dyspnœa.—Since the red discs of the blood have the function of carrying oxygen to the tissues, imperfect respiration is among the most marked effects of anæmia. In fact, the result is the same whether the oxygen is prevented getting to the blood as in laryngeal and pulmonary diseases, or blood prevented reaching the air as in cardiac disease, or whether, blood

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and air freely meeting, there is not enough hæmoglobin to convey sufficient oxygen to the tissues.

Hence dyspnœa is almost always present in cases of anæmia; even when the patient is at rest the breathing is unduly rapid without his being conscious of it; when he makes any effort he may be seized with the most distressing suffocative paroxysms and palpitation of the heart. In some cases similar paroxysms come on without apparent cause. Probably the nerve-cells of the respiratory centre, which are known to be stimulated to excessive discharge by blood containing a normal quantity of hæmoglobin when this is imperfectly oxygenated, are affected in precisely the same manner by blood in which the hæmoglobin is greatly diminished in amount, notwithstanding that what there is of it may be saturated with oxygen. In either case the oxygen that reaches the nerve-cells is deficient. There is no excess of carbonic acid, for that is conveyed from the tissues to the lungs in solution in the liquor sanguinis. Hence we do not see cyanosis or congestion.

Fatty degeneration.—The reduction in the amount of oxygen supplied to the tissues seems to be the cause of one of the most striking of the morbid appearances which are found in the bodies of those who have died in a *state* of extreme anæmia, namely, a granular or fatty degeneration of the muscular substance of the heart as well as of the lining membrane of the larger vessels, and of the secreting cells of the gastric glands, the liver, and the kidneys. At one time we supposed at Guy's Hospital that such changes were peculiar to the form of idiopathic anæmia which had been described by Addison, otherwise called pernicious anæmia. But in 1873 the characteristic appearance of the heart was observed in a woman who *died of* cancer of the breast; in 1874 in one after hæmatemesis from an ulcer of the stomach; in 1877 in a man who had suffered severely from hæmaturia, and also in a woman who had had a bleeding malignant tumour in the neck. Moreover, Perl has experimentally produced the same fatty degeneration in animals by repeated venesection ('Virch. Arch.,' vol. lix).

The degeneration is not universally distributed, but specially affects the muscular fibres of the columns of the mitral valve and those which lie beneath the endocardium lining the septum and the ventricles generally. It gives rise to the formation of a series of parallel, opaque, yellowish or cream-coloured lines, which run across the direction of the fibres, the so-called "tabby-cat striation" (p. 51). With a microscope the opacity and pallor are seen to be due to the presence of closely aggregated fat-granules and globules, which look black by transmitted light. They are doubtless waste products that have accumulated in consequence of there not being oxygen enough to remove them. The reason why the muscles of the body generally show no similar change is probably that for a long time before death the patient has been resting in bed. At least in some cases, the diaphragm and the intercostal muscles share in the same affection as the heart.

Muscular weakness is one of the most marked effects of anæmia. The patient may be capable of exerting great power in a sudden effort, but he quickly becomes fatigued, and his strength is soon exhausted. He is also incapable of undergoing mental labour; but his nervous centres are often very excitable, being in a condition known as "irritable weakness." Thus there is sometimes exaggerated sensibility to sensory stimuli, such as a bright light or a loud noise. So again, whereas the sexual appetite is, as a rule, diminished or suspended, it occasionally happens that a morbid

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erethism is developed, attended with frequent emissions and with inability to complete the act of coitus. In the female severe anæmia is almost always attended with amenorrhœa, and generally with temporary or permanent sterility; but occasionally menstruation continues, and may even be profuse.

The *pupils* are uniformly dilated, and contract somewhat sluggishly to light, and this *mydriasis* helps the pearly conjunctiva to give a characteristic aspect to the eyes.

The *pulse* in anæmia is small, soft, and feeble, in proportion to the severity of the case; or it may be imperceptible at the wrist. But when the cause is a sudden loss of blood, the pulse is often sharp and jerking.

The *temperature* of the surface of the body is often low in ordinary anæmia, but in the gravest forms we shall see that irregular pyrexia is an important symptom.

Pathology.—The most essential character of the blood in anæmia is deficiency in hæmoglobin; and when the "corpuscular richness" of the blood is diminished this may sometimes be due to the want of an adequate supply of hæmoglobin, rather than to failure of the process by which it is developed. Now it is obvious that there are two ways in which the amount of hæmoglobin may be reduced below the normal standard: (1) it may escape from the vessels by hæmorrhage, or be consumed within the body more rapidly than it can be reproduced; (2) its formation may be defective.

When anæmia results from hæmorrhage, and the patient quickly regains his colour as soon as the bleeding is arrested, all the formative processes, both chemical and histological, are, we may suppose, in a perfectly normal condition. When anæmia seems to arise spontaneously, most pathologists have supposed that it is entirely due to a defect in these processes. But Quincke has recorded some observations which suggest the possibility that even in such cases there may be an undue destruction of red discs and of the hæmoglobin which they contain. He has found that in some instances the amount of iron in the liver is from ten to thirty times as great as under normal circumstances, and that there is also an increase of it, but to a less extent, in the kidneys and in the pancreas. The liver-cells present granules, which are supposed by him to consist of an albuminate of iron; they give to the organ a yellow-brown colour which he seems to think characteristic; the addition of sulphide of ammonium turns the tissue of a greenish-black colour; that of ferrocyanide of potassium turns it blue. However, the significance of this discovery remains doubtful. Quincke himself suggested that it may possibly have resulted from the medicinal administration of iron at some former period of the patient's life. A similar deposit of iron has also been found in diabetes and in enteric fever.*

The striking discovery by Virchow of the condition which he named leuchæmia led to too broad a separation between what was before known as anæmia splenica and other forms of extreme anæmia: it seemed to be forgotten that excess of white corpuscles is always accompanied by a deficiency of red; that leuchæmia is, in fact, only a species of anæmia. Addison's discovery, of equal importance with Virchow's, that anæmia of the severest kind may occur independently of chlorosis and of any other known cause, may go on uninfluenced by treatment, and may end in death;

* Dr Hunter has confirmed and extended Quincke's conclusions. See his papers in the 'Lancet,' September 22nd to October 6th, 1888; and also Dr Mott's (*ibid.*, March 16th, 1889; 'Path. Trans.,' 1890; and 'Practitioner,' August, 1890).

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and that this restricted group of fatal and idiopathic anæmia is marked by definite symptoms and anatomy—was not understood in Germany, where under the name of progressive pernicious anæmia a heterogeneous group of cases were united together, many of them secondary, some uterine, some malarial, and only agreeing in the fact of their fatality.

The *classification* of cases of anæmia is not easy, since many of them are of uncertain origin and nature.

The following is practically the arrangement proposed by the present writer in an article in the 'Guy's Hospital Reports' for 1882:

i. *Symptomatic anæmia.*—(1) The most obvious and direct cause of anæmia is external *hæmorrhage.*

In women the most frequent source of hæmorrhage is menorrhagia, and in men bleeding piles; recurrent epistaxis is a frequent cause in early youth, and it also occurs with the degenerated arteries of old age.

The pallor caused by large losses of blood in a healthy subject is accompanied by muscular weakness, a rapid and irritable pulse, and many of the other signs above noted. In most cases, so soon as the bleeding is stopped, the lost corpuscles are rapidly restored with the help of a healthy appetite, assisted in some cases by wine, and by chalybeate drugs. Sometimes, however, even after traumatic hæmorrhage in a healthy subject, recovery is neither rapid nor complete. In a case which came under the writer's observation a British officer in one of the Chinese ports was waylaid and nearly murdered by assassins. He received several stabs, and lost a large quantity of blood. He ultimately recovered and returned to England, but when seen some years later he was still extremely anæmic. Similar cases of protracted or permanent anæmia not unfrequently follow a single delivery accompanied by severe flooding; and such prolonged pallor is often seen as the result of continued, though small, losses of blood by epistaxis, and above all by bleeding piles.

(2) A second natural group of cases of anæmia consists of those which follow losses, not of blood, but of leucocytes and albumen, which may be considered a kind of modified hæmorrhage. Such is the anæmia of prolonged suppuration with or without lardaceous disease, the anæmia which follows chronic and profuse leucorrhœa, and long-continued diarrhœa or dysentery unaccompanied by hæmorrhage.

(3) Closely allied to the last cases are those in which anæmia depends on excessive expenditure, and is often associated with loss of flesh. Here we may place the anæmia which accompanies too frequently recurring pregnancies, too long-continued lactation, and perhaps that of Bright's disease with prolonged albuminuria. The anæmia of sexual excesses in male subjects may perhaps come under the same head.

(4) Another group is that of cases of anæmia which depend not on hæmorrhage or excessive discharges which impoverish the blood, but on deficient nutrition; in both cases the blood as a tissue suffers in its nutrition, in the one from the drainage of profuse expenditure, in the other from the starvation of diminished supplies. Under this head comes the pallor of inanition from whatever cause, particularly, from a clinical point of view, the starvation produced by stricture of the œsophagus, by the anorexia of gastritis and severe indigestion, and by gastric ulcer in cases which are unaccompanied by hæmorrhage.

We may perhaps include in this kind of anæmia from defective income, that which follows enteric *fever* and other acute diseases, where there is

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neither hæmorrhage nor profuse discharge, but where the anæmia is accompanied by loss of flesh and is soon repaired by the healthy appetite of convalescence. The striking and rapid anæmia of acute rheumatism and of diphtheria may be included in the same group; but there is probably a destructive process going on at the same time.

Closely allied to the last group are cases in which food is taken and apparently digested in sufficient quantity, but either the hæmoglobin is not formed at all or more probably is in most cases destroyed after formation, so that there is deficiency in this remarkable compound of a proteid with iron, notwithstanding good nourishment of the tissues of the body.

In *phthisis* the causes of anæmia are numerous: there is the wasting discharge of pus from the lungs; there is frequently hæmorrhage, and there is always impaired appetite and digestion; but here also the growth and spread of tubercle appears directly to impair the nutrition, since we see pallor and emaciation in children in whom tuberculosis of lymph-glands or serous membranes is unaccompanied by suppuration or by fever.

The pallor of tubercle leads us to the closely allied pallor of *malignant disease*, which also can be most frequently ascribed to hæmorrhage or discharge, or to loss of appetite; but it appears to be partly due to the destruction of red corpuscles with a relative increase of the white, or, to use Virchow's term, anæmia with leucocytosis.

(5) Whether or no we admit a destruction of hæmoglobin in cases of rapidly spreading tubercle, sarcoma, or cancer, we see it unmistakably produced in the cases of *toxic* anæmia which result from the direct action on the blood-corpuscles of mercury of phosphorus or of lead.

(6) Intermediate between these and the preceding cases of tubercular and malignant anæmia may be placed the cachectic anæmia of *syphilis* and of *malaria*. In all the above cases anæmia is secondary, symptomatic, and more or less explainable.

ii. *Primary anæmia.*—We now come to a second broad division of cases of anæmia which own no such causes, which are primary and associated with definite anatomical lesions.

To this group of primary organic anæmia belongs the *anæmia lymphatica*, or anæmia with hypertrophy of lymph-glands and other lymphatic structures of the body, including Hodgkin's disease and Virchow's leucocytosis splenica, and also the primary anæmia which is combined with a considerable excess of leucocytes and a considerable hypertrophy of the spleen—the anæmia splenica of the older writers, which was often confounded with malarial cachexia—the *leuchæmia* of Virchow.

iii. *Chlorosis.*—Another well-marked and familiar clinical kind of anæmia is that which has been known from early times as *chlorosis* or green-sickness, a form characterised by the sex and age of the patients, by the absence of emaciation, and by the striking results of appropriate treatment.

iv. *Idiopathic anæmia.*—Lastly remain the remarkable but decidedly rare cases, first and sufficiently defined by Addison, primary but unassociated with anatomical changes in the spleen and other organs, widely distinct in their clinical aspect and prognosis from cases of chlorosis, idiopathic in the strictest sense of the word; and related rather to leuchæmia and Hodgkin's disease than to any other form of anæmia, by their severity, their proclivity to hæmorrhage and occasional pyrexia (symptoms not present in secondary anæmia nor in chlorosis), their tendency to a fatal issue, and, lastly, in their reaction to arsenic, and their resistance to the

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chalybeate treatment which is so efficacious in chlorosis and most forms of secondary anæmia.

The symptomatic groups of anæmia have already been incidentally mentioned in the chapters on fever, malaria, syphilis, phthisis, cardiac disease, and disorders of the stomach and kidneys. We therefore now proceed to the primary forms as above defined, and begin with the remarkable condition described and named by Virchow.

LEUCHÆMIA.*—In 1845 Dr Hughes Bennett, of Edinburgh, recorded a case of enlargement of the spleen in which, after death, the blood was found to be full of objects which he regarded as pus-cells and attributed to "suppuration of the blood." A month later Virchow published a similar case; but he perceived that the cells in question were identical with the colourless corpuscles now called leucocytes, and proposed to call the affection "leukæmia," *i. e.* a condition of white blood. In 1846 Dr Fuller, at St George's Hospital, and Dr Walshe, at University College, demonstrated the same change in the blood of living patients, and the disease has ever since been universally recognised.

Subsequent research discovered, as one would expect, many previously recorded but misunderstood cases. Thus Dr Craigie, after seeing Bennett's case in 1848, remembered a similar one years previously, in which the pathologist John Reid had recognised in the blood "globules of purulent matter and of lymph," and published it in the 'Edin. Med. Journ.,' October, 1845. Donné also, in 1839, had found what he called "globules muqueuses" in the blood of a patient of Barth. Velpeau, as far back as 1827, had recorded a case in which the spleen was enlarged, and the blood looked as if it were mixed with pus. Other cases are quoted from Bichat and Andral, with descriptions of the blood as "*sanie grisâtre*," and "*sang comme sanieux*." Piorry had named the same condition "*Hémite*," by which he meant to denote "inflammation of the blood." In 1852 Dr Bennett proposed the name "leucocythæmia" for the new disease.†

Definition.—A *slight* excess of leucocytes is not enough to justify the title of leuchæmia. Virchow long ago pointed out that they may be somewhat increased in numbers under various conditions attended with irritation of lymphatic glands, as well as during pregnancy, and in fevers. He proposed to distinguish all such minor degrees of blood-change under the name of "leucocytosis;" and it has been suggested that the line should be drawn at the point where the proportion of white cells to red discs reaches one in twenty.

As a rule, leuchæmia is accompanied by considerable hypertrophy of the spleen, so that "splenic leuchæmia" is almost a synonym. But there are exceptions to this rule.

This was ascertained by Virchow as far back as 1847, and was fully recognised by Bennett, in his work published somewhat later. In the first volume

* *Synonyms.*—Leucocythæmia.—*Fr.* Leucocythémie.—*Germ.* Leukämie.

It matters little whether, with Virchow, we call the blood "white" or "pale," or with Bennett call it "white-celled;" neither term is literally exact. But the former is shorter, is more generally accepted by pathologists, and has the right of priority. For Virchow gave it ("Weisses Blut," 'Froriep's Notizen,' November, 1845; "Leukämie," 'Arch. f. Path. Anat.,' Bd. i, S. 563, 1847) some years before Bennett proposed to amend his first name, "suppuration of the blood," by the new one of "Leucocythæmia" ('Leucocythæmia, or White-celled Blood, in relation to the Physiology and Pathology of the Lymphatic Glandular System,' Edin., 1852).

† On the history of the discovery see further Virchow ('Gesammelte Abhandlungen,' pp. 149—218) and Bennett ('Prin. and Pract. of Medicine,' pp. 857—890).

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of his 'Archiv,' Virchow related an instance in which the principal morbid change was in the lymph-glands, which were enormously enlarged throughout the body. Subsequently he described two forms of leuchæmia—the one "splenic," the other "lymphatic"—distinguished by difference in the size of the leucocytes, these being comparatively large and having sometimes more than one nucleus in the splenic form, but in the lymphatic form being small, and having their scanty protoplasm in close contact with a solitary nucleus. This microscopical distinction is still admitted by Mosler and others. But subsequent experience has shown that to speak of a "lymphatic leuchæmia" as comparable with the splenic affection is practically inconvenient. Virchow himself soon became aware that leuchæmia is usually absent when there is a general enlargement of the lymph-glands, a condition previously described by Hodgkin; and in the 'Krankhaften Geschwülste' he described under the heading of "lympho-sarcoma" cases of this kind, for which other German writers have used the singularly inappropriate name of "Pseudo-leukämie." As we shall presently see, it is impossible to draw a line between cases of anæmia lymphatica or Hodgkin's disease attended with leuchæmia and the larger number in which the proportion of white cells to red discs is nearly or quite normal. Cases now and then occur which might be described as presenting a combination of splenic leuchæmia with lymphatic anæmia or Hodgkin's disease. A marked example of this was recorded by Dr Frederick Taylor ('Path. Trans.,' vol. xxv, p. 246).

Apart from such exceptional instances, leuchæmia is distinguished by the three following characters:—(1) Enlargement of the spleen is present from the commencement, it is considerable, and consists in a simple overgrowth of the splenic tissues, there being no scattered white nodules in the organ; (2) the excess of leucocytes in the blood is great; (3) an affection of lymphatic glands and of various other organs and tissues, if present, begins later than that of the spleen, it is comparatively slight in degree, and does not assume the form of definite tumours.

Physical diagnosis of the enlarged spleen.—First it may be well to enter into some details with regard to the determination of the position and size of the spleen by percussion. It had been pointed out by von Luschka that the long diameter of the organ inclines downwards and forwards parallel with the lower ribs on the left side, and that its normal extent corresponds very closely with the space from the ninth to the eleventh rib. But about one third of its upper and hinder part is covered by the inferior border of the lung, and is therefore inaccessible to percussion. Consequently, as Weil and others have shown, the arch of splenic dulness, as it can be mapped out upon the surface of the chest, forms a figure which is rounded in front and below, whereas above it is flattened; behind it has no definite limit, and merges insensibly into the dulness produced by the kidney and other structures in the loin. Its length (in the line of the long diameter of the spleen) is usually about three inches; its breadth, in a direction at right angles with the length, is two or two and a half inches; the distance of the lower and anterior extremity of the organ from the edge of the costal cartilages is an inch and a half or two inches. The patient, while percussion is being practised, may either be sitting upright or lying towards the right side upon the right shoulder and the right hip, with the left side raised just sufficiently to make the lateral region of the chest accessible. It is also necessary to mention that the splenic dulness is always rather "superficial" or incomplete in consequence of the comparatively small thickness of the organ. Light

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percussion is therefore needed for its determination; and in some cases, especially when there is much subcutaneous fat, when the lung is emphysematous, or when the stomach and the colon are much distended, its extent cannot be accurately mapped out.

When the spleen becomes enlarged, its lower and anterior extremity projects more and more downwards and forwards, and soon can be felt below the costal cartilages. It then constitutes an "abdominal tumour." Guttmann states that he has seen three cases in which a spleen of normal size was displaced so as to lie far below its usual seat. In one instance, that of a man aged thirty, the organ was put back and kept in position by a bandage, whereupon the pain for which he had long been treated immediately disappeared. In another patient, a woman aged forty-eight, the dislocation of the spleen began as the result of a violent physical effort. In the third case the diagnosis was verified by extirpation of the organ; recovery after the operation took place in a fortnight.

In cases of leuchæmia the first thing usually noticed by the patient is that his abdomen is becoming larger, that there is a fulness or a *lump in* the left side, or that he has a dull aching pain there. On examination one generally finds that the spleen is already very large; even at this period its size is seldom less than that which would correspond with an advanced stage, or with an extreme degree, of any other disease. Sometimes it reaches the umbilicus, and in many cases it descends to the level of the iliac crest. As time goes on it occupies a position which could hardly have been anticipated (see the series of diagrams by the author in the 'Guy's Hospital Reports' for 1869). The vessels at its hilus seem to offer a resistance to its expansion in a straight line, and it therefore follows a curved course, its lower end sweeping across the brim of the pelvis, and even turning upwards when it has reached the right iliac fossa. Probably the fold of peritoneum between the splenic flexure of the colon and the parietes, called *sustentaculum* or *trabecula lienis*, may, when well developed, help to direct the spleen forwards.

Ultimately the spleen may so completely fill the abdomen below the navel that in women it has often been mistaken for an ovarian tumour. Its real nature may, however, be distinguished by the sharp edge which crosses the abdomen obliquely from the left lower ribs downwards, and which presents one or more notches. Its surface is almost always smooth and firm. A friction-fremitus can sometimes be felt over it, and with the stethoscope not only a rub, but occasionally a blowing systolic murmur is to be heard, like the placental *souffle*. At an advanced stage of the disease it may be separated from the parietes by a layer of ascitic fluid, through which the fingers dip before they reach it.

After death the spleen is often found to be fixed to the adjacent parts by adhesions. Its capsule may present large white or yellowish opaque patches of thickening. Its cut surface is generally smooth, shining, and homogeneous-looking; but sometimes it is marked with whitish lines and striæ, due to thickenings of the trabeculæ. Its consistence is often very firm, and its colour brownish rather than the natural red. It not infrequently shows a number of wedge-shaped pale nodules, resembling the blocks produced by embolism. Histologically the only change discoverable is overgrowth of a tissue, which is like that of the organ in its normal state; but the stroma often has a peculiarly fibrous character.

The blood.—In splenic leuchæmia this is paler than natural, and may look turbid; indeed, when the excess of leucocytes is very great, it some-

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times has a greyish-red colour, resembling a mixture of pus and blood. Charcot's crystals have sometimes been found in the blood and also in the marrow of the bones. They are of variable size, colourless, elongated, and octohedral, consisting of two very slender four-sided pyramids set base to base. Their chemical nature is uncertain; they dissolve in warm water and in alkalies, but are insoluble in alcohol and ether. They have often been found in the sputum, particularly in cases of asthma (vol. i, p. 1147).

After death the appearance of coagula in the heart, or in the great vessels, is often peculiar; they are grey and opaque, instead of being yellow and translucent, and have been likened to solidified pus.

The proportion of leucocytes to red discs varies widely in different cases, and according to the stage of the disease. From the normal ratio, which is not higher than 1 to 300, it may be increased till it reaches 1 in 20, 1 to 10, 5, or 2. In extreme cases the leucocytes may even be the more numerous, as in a case of Sörensen's, in which they were counted, and found to be as 68 to 47 of red discs.

The number of red discs is always much diminished. But it is remarkable that this anæmia is sometimes unattended with obvious pallor of the countenance; as Wilks long ago pointed out, patients, even at an advanced stage of the disease, have often colour in their cheeks and lips, so that, seeing them in bed, one would hardly imagine them to be very ill; but sometimes the skin is yellowish white, like wax; it was so in the only infant we have had in Guy's Hospital with splenic leuchæmia.

Some years ago Mr Golding Bird examined blood from a case of leuchæmia, upon a warm stage; many of the leucocytes were found to be in active movement, but others remained motionless. In 1880 Dr Cavafy read a paper at the Royal Medical and Chirurgical Society upon a case, already far advanced, in which he repeatedly made observations of this kind: the proportion of leucocytes which showed even slight amœboid movements was at first only 12 per cent., at a later period only 6 per cent. He concluded that the greater number of these were dead or dying, and incapable not only of development, but even of emigrating through the walls of the vessels. Other observers have stated that in some cases of this disease many of the leucocytes are obviously in a state of fatty decay; in fact, these two characters of immobility and granular degeneration go far to justify the old descriptions of "pus in the blood."

The altered condition of the blood, by diminishing the amount of oxygen which can be taken up, helps in producing the *dyspnœa*, which is sometimes the chief complaint of the patient. This may be present only when muscular efforts are being made; in extreme cases even the slightest movement is attended with the utmost distress. Perhaps, therefore, there is nothing surprising in an observation made by Pettenkofer and Voit,* according to which, during rest, the quantity of oxygen absorbed and that of carbonic acid given off seem the same as in health. Another cause of dyspnœa is displacement of the diaphragm upwards by the enlarged spleen.

Hæmorrhage.—It has been supposed that the over-abundant leucocytes adhere to the lining membrane of small vessels, and accumulate so as to obstruct them like minute emboli. Moreover, if it be a fact that many of the cells no longer possess their vital properties, and are undergoing disintegration, nothing is more likely than that they should set up a morbid change in the walls of the capillaries with which they come in contact,

* 'Zeitschft. f. Biol.,' v, 319, 1869. The figures were: O, 790 to 832; CO_2, 265 to 249.

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softening them, and rendering them liable to rupture. In this way the tendency to hæmorrhage has been explained, which is a marked symptom of leuchæmia; but, as we shall see, this is also a symptom of the other grave and primary kinds of anæmia.

Epistaxis is very common; it may recur every day, and it sometimes is the direct cause of death. Bleeding may also take place from the intestine, the stomach, the kidneys, the lungs, or the uterus. Moreover, the hæmorrhage, after a wound or other injury, is apt to be excessive; even the extraction of a tooth has, in at least one case, led to a fatal result. The statement has been made that the amount of fibrin yielded by the blood in this disease is above the normal, but that, instead of coagulating in long elastic filaments, when separated by stirring, it falls in granular fragments. Purpuric spots are frequently seen upon the skin; and after death the surface of the heart may be found ecchymosed. A large quantity of blood is sometimes extravasated among the muscles or behind the peritoneum. Lastly, hæmorrhage into the brain occasionally occurs, with the symptoms of apoplexy.

In the retinæ hæmorrhages are frequently observed, both during life and after death. Dr Gowers, in his article in 'Reynolds' System of Medicine,' says that they are usually small, and most abundant towards the periphery; they often form striæ, following the lines of the nerve-fibres. After a time the blood undergoes conversion into a brownish pigment. The hæmorrhagic patches often have white or yellowish-white centres, and similar spots may be observed without any accompanying extravasations. Dr Gowers has seen the retina affected with diffuse swelling, and its veins distended and tortuous—"leuchæmic retinitis."*

Pyrexia.—The temperature is often raised from time to time, and after a few days follow apyrexial intervals. Sometimes this pyrexia is accompanied with shivering and sweating, and may reach 103°, or higher still; but usually it is of a mild type, and would not be recognised without the thermometer. Leuchæmic patients often complain of weakness, headache, giddiness, noises in the ears, palpitation of the heart.

The lympharia.—Several of the organs and tissues are liable to changes in splenic leuchæmia, but few of these changes can be said to have any clinical significance. In about one of every three cases there is enlargement of *lymph-glands*, especially those of the abdomen and of the chest. The increase in their size is considerable; in our records it is often noted that they were twice as large as normal. They may be firm and fleshy, or soft and medullary in character. They appear not to become fused together, nor does a new growth start from them and penetrate into structures adjacent—characters which distinguish them from lympho-sarcomata. The *follicles* at the base of the tongue and the *tonsils* may be greatly swollen, and here an inflammatory change may also be present, for in one of our cases the tonsil was found after death to be sloughing. There may also be diffused pharyngitis and stomatitis. The gums may become swollen, ulcerated, or gangrenous. The *intestinal follicles*, solitary and agminated, are sometimes greatly enlarged, and a lymphoid growth is said to extend beyond the limits of the glands and to infiltrate the submucous tissue. Even ulcers may form, which are said to have thickened edges, and to resemble tubercular ulcers.

* These changes in question resemble those which occur in Bright's disease. Have observers always been sufficiently careful to exclude the possibility of its presence as a complication?

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Among the most remarkable of all the changes in the lymphoid structures of the body are those which occur in the *medulla* of the bones, and which were first described by Neumann. The cancellous tissue acquires a greenish-yellow appearance, exactly like that which is seen in osteomyelitis; and on pressure a puriform juice exudes. Moreover, the adipose tissue or yellow marrow of the shaft of long bones becomes changed into lymphatic (cytogenic or adenoid) tissue like the red marrow of the cancelli, but of the same pale unhealthy colour which is seen in the cancellous tissue itself. Under the microscope leucocytes fill the greater part of the field, the red discs are often deformed and nucleated, and the normal fat-cells have disappeared, leaving only free oil-drops and fatty granules.

Mosler met with a case in which this leuchæmic affection of the sternum was indicated during life by great tenderness on pressure; and this important symptom has frequently been since observed (see for instance the most recent example, published by Dr Beatty, of Dublin, in the 'Brit Med. Journ.' for April 18, 1891).

It is a curious circumstance that in some instances wedge-shaped infarctus, exactly like those which are commonly seen in pyæmia, are found in the *lungs*. This was the case in a patient whose spleen was excised by Mr Bryant in 1866, and who only survived the operation three hours. The patches which occupied the back parts of the lungs had gangrenous centres and red borders. Dr Gowers speaks of such infarctus as arising from plugging of the pulmonary capillaries by leucocytes; but even if this mechanical theory be adopted, the relation to pyæmia must be admitted to be very close. The lungs in leuchæmia appear not to exhibit definite nodules of a new growth, such as are seen in Hodgkin's disease. In many instances they are quite healthy.

Of the remaining organs, the *liver* is most frequently diseased in splenic leuchæmia. It not infrequently weighs as much as eight or ten pounds. There is not always any obvious change in the appearance of its cut surface, and Virchow has stated that the enlargement may be due simply to overgrowth of the hepatic cells. But in many instances there are masses of lymphoid growth scattered through the organ, especially in the neighbourhood of the vessels. Sometimes these are visible only with the aid of a lens, sometimes they are apparent as minute, greyish-white granules. Moreover, even within the acini, numerous leucocytes are seen between the hepatic cells; some appear to lie within the capillaries, others are outside them, being supported by a nucleated stroma of their own. It does not appear that these changes ever cause jaundice.

The *kidneys* are less often affected; in them the lesion assumes the form of scattered, greyish-white striæ, running through the cortex, and bearing a close resemblance to those which are seen in cases of ascending nephritis (p. 538). The surface is smooth and marked by ecchymoses. On microscopical examination, beside more or less numerous extravasations of red discs, vast numbers of leucocytes are seen between the tubules. The urine is not infrequently albuminous during life, but sometimes, though not always, this is to be accounted for by the presence of other changes in the kidneys, such as occur in ordinary Bright's disease.

Ætiology.—In the majority of cases no cause for splenic leuchæmia can be discovered, but in a certain number it appears to be a remote sequela of *ague*. Some of the writers who first studied the disease thought that it bore no relation to the marsh poison, because it often occurred in persons who had never been exposed to malaria, and because the affection of the

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spleen resulting from ague was then regarded as merely congestive. But whenever numerous cases of leuchæmia have been collected, some of the patients are found to have suffered from intermittent fever. Dr Gowers found it so in thirty among 150 cases which he collected, and at Guy's Hospital the proportion has been considerably higher. There is clearly something more than a mere coincidence in these results, but it is remarkable how long the interval has sometimes been, and how mild the attack of ague to which such serious consequences are traced. In nine out of twenty-one cases, a period of from ten to thirty years passed before leuchæmia showed itself by any symptoms. A patient in Guy's Hospital in 1880, a Pole, had suffered from tertian ague in 1858, during four months in Warsaw, but first noticed an enlargement in the left hypochondrium in 1866.

In women, leuchæmia sometimes is discovered during pregnancy; sometimes it seems to arise out of the weakness resulting from parturition.

Of supposed exciting causes, such as over-fatigue, distress of mind, intestinal catarrh, and injuries, nothing positive is known.

Leuchæmia occurs about twice as often in males as in females. It affects persons of all ages, from infants to men above seventy, but it is most frequent in those who are between twenty and fifty.

Diagnosis.—This offers difficulties only in the early stages, with the exception of certain cases which appear to be transitional between leuchæmia and Hodgkin's disease. Mosler says that it is apt to be taken, in the first instance, for chlorosis; but this should not be, for a splenic tumour and excess of leucocytes in the blood may be discovered before there is pallor.

There may be great enlargement of the spleen with anæmia, but without leuchæmia (*anæmia lienalis v. splenica*). Dr Moxon cites two such instances, one of which occurred to Mr Spencer Wells, the spleen weighing 6 lbs., the other to Mr Squire, the spleen weighing 13 lbs. Griesinger described this affection as *splenic anæmia*. It is certainly rare (cf. p. 626).

Again, as a rare exception, there may be excess of white corpuscles in the blood, while the spleen remains of natural dimensions (*non-splenic leuchæmia*). If the excess is very great, as in a case related by Dr Goodhart, in the 'Clinical Society's Transactions' for 1877, though the spleen weighed only seventeen ounces, one may admit the case as one of splenic leuchæmia.

In other cases the yellow marrow is found changed from adipose into adenoid tissue, as above described (p. 623); we must then use the term *leuchæmia myelogenica*.

The white corpuscles in the blood may be moderately augmented in numbers under various conditions, including not only those enumerated by Virchow, for which he proposed the term "leucocytosis," but also the process of suppuration or the development of malignant new growths. Thus in 1875 a patient died in Guy's Hospital of jaundice from a cancerous tumour in the head of the pancreas, attended with suppuration in the liver. The blood was repeatedly examined during the last fourteen days of his life, and was always found to contain a decided excess of leucocytes—as many as 130 to 150 in a field—which were in active movement.

Pathology.—The theory of splenic leuchæmia is still very imperfect. Many observers, basing their opinions on the admitted doctrine that some at least of the white corpuscles of the blood are normally derived from the spleen, have imagined that they could account for the phenomena of the disease by supposing that the organ, being hypertrophied, produces an excessive number of white corpuscles which pass into the circulation.

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But this hypothesis does not explain the fact that the total corpuscles of both kinds in the blood are actually *fewer* than normal, and that the red discs are greatly diminished in numbers. It seems that there is some change in the leucocytes themselves, which prevents their undergoing conversion into red discs, as they naturally should.

If this change is the starting-point of the whole morbid process, the lesions in the liver, the bones, and other organs might, notwithstanding Dr Cavafy's observations, be plausibly ascribed to the passage of superfluous leucocytes into their interstices; for there is as yet no proof that these bodies constantly, and from the very first, are so paralysed as to be unable to penetrate the wall of a capillary. The overgrowth of the spleen itself might be accounted for on the hypothesis that a large number of white corpuscles are retained in its texture; and the occurrence of splenic enlargement without leuchæmia might be attributed to retention in the organ of all the leucocytes that fail to undergo the normal transformation into red discs; while, conversely, the fact that the blood is sometimes loaded with them, the spleen remaining of normal size, might be supposed to depend on their all escaping.

This, however, is not a satisfactory hypothesis; for why should the leucocytes all lodge in the spleen in most cases, and in the bones or lymph-glands in others? and why should they lodge in the lymphatic organs only, and not in the muscles, brain, lungs, or other viscera?

The fact is that leuchæmia is only a special kind of anæmia. The red blood-discs are not formed or are destroyed, but the white ones are not formed in excess. A diseased organ never does more work, and the enlarged spleen is not a healthy hypertrophy but a morbid swelling.

But the full explanation of the disease must wait a better knowledge of the process of hæmatopœesis and hæmatolysis than we at present possess.

Course and event.—Splenic leuchæmia, as a rule, advances slowly towards a fatal termination. Its duration is commonly from one to three years; in the case of a child it is much shorter. The prognosis in an individual case must be based upon a careful study of the various symptoms; the extent to which the spleen is enlarged is of much less significance than the degree of anæmia and of breathlessness. As regards the state of the blood, it probably matters much less whether the number of leucocytes is greatly increased than whether that of red discs is greatly diminished.

Death often takes place unexpectedly by some complication, such as hæmorrhage, pleurisy with effusion, chronic peritonitis, diarrhœa or phthisis. Sometimes it is almost sudden, as in a case which occurred at Guy's in 1876, when œdema of the larynx was found at the autopsy.

Treatment.—It is possible that at an early stage the disease may sometimes be arrested by treatment. Mosler relates the case of a boy aged ten, whose spleen was considerably enlarged, and whose blood contained leucocytes in the proportion of one to twenty red discs; he took a drachm and a half of sulphate of quinine in the course of four days, then ten grains and afterwards six grains daily; he completely recovered. Dr Goodhart, in 1876, stated to the Clinical Society that in the previous two years he had seen six cases, all in children under two years old, with moderate increase of the spleen, and with about ten times the usual proportion of leucocytes in the blood, and that they all got better under treatment, the drugs used being iodide of iron, or phosphorus, or cod-liver oil. It may, however, be reasonably doubted whether the quinine or these other drugs contributed to the favourable result.

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Other measures which have been recommended are a cold douche directed upon the left hypochondrium, and the application of a galvanic or even of a faradic current to that part of the body, the positive pole being placed over the tenth rib, the negative over the enlarged spleen; it is said that by either plan of treatment the organ may often be greatly reduced in size.

It seems to be well ascertained that steel is in most cases useless, but it is probable that arsenic may be more useful, and it should at least be tried. When a case has already advanced, little or nothing can be done to check its progress. Quinine is then, at least, without any appreciable result. Transfusion of blood is unlikely to be of any avail. Excision of the spleen should be rejected, on account of the danger of hæmorrhage from rupture of adhesions, as well as of peritonitis; moreover, it probably would have no effect on the state of the blood (see, however, Sir Spencer Wells' paper in the 'Brit. Med. Journ.,' July 13th, 1889, p. 55).

Non-splenic leuchæmia.—Beside splenic leuchæmia, rare cases occur of the same condition of the blood, the same clinical features, and the same fatal event, without any enlargement of the spleen, but with hypertrophy of the lymph-glands, the marrow or other lymphatic organs.

The first variety was described by Virchow, and may be named *Leuchæmia lymphatica;* it is extremely rare (cf. p. 629).

The second (*Leuchæmia myelogenica*) was discovered by Neumann and Bizzozero (p. 623), and is also rare alone; but it is far more often associated with the common (splenic) form of leuchæmia, or with Hodgkin's disease (*v. infra*, p. 632). A third variety was observed in an isolated case by Béhier, and named *Leuchémie intestinale*, because it was associated with hypertrophy of Peyer's patches (1868). A similar case is quoted by Virchow as having been observed by Heschl. In neither of these cases, however, were the bones examined.

Anæmia splenica.—If we distinguish by the special name of leuchæmia those cases of anæmia splenica of older authors in which there is excess of leucocytes as well as defect of red corpuscles in the blood, we must, as we have seen, admit the anatomical divisions of leuchæmia splenica, leuchæmia myelogenica, and leuchæmia lymphatica. In the same way when there is no excess of leucocytes we have a corresponding series which may be called anæmia splenica, anæmia lymphatica, and anæmia myelogenica. The last scarcely occurs as an independent malady, but is sometimes present as a complication of anæmia lymphatica (*infra*, p. 632); the second will next be described as Hodgkin's disease. The first is not so common a clinical occurrence. A well-marked case was published by the writer in 1870 as a spleen from a case of fatal anæmia, and another in 1875 of enlarged liver and spleen from overgrowth of adenoid (lymphatic) tissue without leuchæmia ('Path. Trans.,' xxi, p. 390, and xxvi, p. 199).

HODGKIN'S DISEASE.*—In the chapter on tumours it was remarked, under the head of lymphoma (vol. i, p. 114), that at the bedside, and even in the *post-mortem* room, one is obliged to class together under a single name certain cases in which the lymph-glands, the spleen, and sometimes many other organs and tissues, become the seats of growths which in different instances vary; histologically, they may be pure lymphomata, or they may

* *Synonyms.*—Anæmia lymphatica—Multiple lymphadenoma, with anæmia or leucocytosis.—*Fr.* Adénie.—*Germ.* Pseudoleukämie.

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be sarcomata, or they may occupy an intermediate position in the scale, so as to deserve the title of lympho-sarcomata. As a rule, the cases in question present at the bedside a common group of symptoms; namely, a more or less general and sometimes extreme enlargement of the lymphatic glands, a moderate increase in the size of the spleen, with the presence of scattered nodules in it, marked anæmia, and more or less subcutaneous œdema of the face, as well as of other parts, so that the patient's appearance is like that of a person suffering from acute Bright's disease. They want, therefore, a designation which shall leave their precise pathological anatomy an open question, indeterminable as it is during life.

Such a name, and one which has met with very general acceptance, is that of Hodgkin's disease, proposed in 1865 by Dr Wilks, to whom we are mainly indebted for the recognition of the malady in this country, in place of the title "anæmia lymphatica," which he had given it in an earlier paper in the 'Guy's Hosp. Reports' for 1862. He found, in fact, that his own observations had been anticipated by a former lecturer on pathology at his own school, Dr Thomas Hodgkin. In a communication made by that accomplished physician to the Royal Medical and Chirurgical Society in 1832 there are recorded several instances in which the spleen and lymphatic glands were jointly affected; of which two at least, and more probably four, are examples of the disease now to be described. In Germany, Virchow, Wunderlich, and many others have recognised the condition as *pseudoleukämie* or *Hodgkin'sche Krankheit*; and in France Trousseau described it in one of his graphic lectures under the name *Adénie*.

Ætiology.—With regard to the causes of Hodgkin's disease but little is known. A very large proportion of those who are affected by it are children or young adults. Thus, of seventeen cases that have ended fatally in Guy's Hospital between 1856 and 1878, all but two have been in persons between eight years and thirty years old at the time of death; of the exceptions, one was in a woman aged forty-five, the other was in a man aged fifty-six. A series of 100 cases collected from various authors by Dr Gowers contains 30 cases fatal under twenty years of age, and 64 under forty; most of the other patients were between fifty and sixty at death.

Hodgkin's disease is much more common in men and boys than in women. In seventeen cases at Guy's Hospital there were fourteen males to three females. Dr Gowers found the proportion as three to one.

Trousseau thought that there was often a starting-point for the glandular affection in some acute or chronic irritation at the angle of the eye, or in the external ear; and we have had six cases in which there was some evidence of such a local origin. In one boy the swelling began in the neck, and was said to have followed a blow from a cricket ball; a girl had suffered from suppuration of the ear, and indeed died of secondary meningitis; in a young man the development of the disease was preceded by abscess of the cervical glands; in two boys it followed measles, that exanthem having led in one instance to an abscess under the left side of the lower jaw, in the other to a swelling in a similar position which for a time subsided, but afterwards seemed to return. In a young man it began with enlargement of the glands in the left groin, from a chancre acquired six months before. Thus it seems likely that in some cases, at least, hypertrophy of the lymph-glands starts in ordinary irritative swelling.

Symptoms.—As is implied in the preceding paragraph, there have been many cases in which an overgrowth of lymphatic glands in some particular

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region on one side of the body has been present for two or three years before there was any sign of extension of the disease to other parts. Dr Gowers mentions the case of a boy whose axillary glands were excised by Mr Chr. Heath six years after they first became enlarged, and in whom, four years later, the cervical glands on the same side had alone become affected. Sometimes the morbid change has seemed to spread by continuity, as from the cervical glands to the thoracic, or from the inguinal to the lumbar; but in other instances it has apparently developed symmetrically on opposite sides of the body, as in both sets of axillary glands, or it has sprung up simultaneously in the most distant parts. One striking character is that the affected glands, even when they have reached the size of pigeons' eggs, often remain isolated from, and freely moveable upon, one another, and unattached to the skin. They are commonly neither painful nor tender. But after a time they are apt to become fused together, either by a process of periadenitis, or more often by an extension of tumour-growth through their capsules, from one gland to another. They sometimes reach an enormous size; there may be several packets in different regions, each of the size of a child's head. They have been found after death to weigh as much as ten pounds. They are generally firm and elastic to the touch, but they may be very soft. A case of Bonfils' is cited in which an abundant quantity of lymph accumulated in one of the glands, and when a puncture was made, continued to run.

In other instances a general failure of the health precedes the development of external glandular swellings. One cannot help suspecting that the affection has then really begun in some of the deeper glands, which are beyond the reach of manipulation; and this suspicion is confirmed by a case, recorded by Dr Wilks, of a man who died in Guy's Hospital in 1856 in an extremely weak and anæmic state, with an enlarged spleen; for the autopsy showed that the mediastinal and the lumbar glands were much enlarged, although the superficial glands were unaffected. In such cases a doubtful diagnosis might perhaps sometimes be cleared up by recognising signs of pressure; for example, spasmodic cough and dyspnœa, distension of the veins, œdema of an arm or leg, or pain in the course of the lumbar or sacral nerves.

Dr Gowers some years ago made an autopsy in a case of Sir William Jenner's, in which, there being general glandular enlargement, a mass of growth extended from the abdominal glands, and involved the solar plexus and nerves going to the adrenal bodies; here there was a discoloration of the skin having the distribution of Addison's disease, notwithstanding that the adrenals themselves were healthy.

Even glands lying outside the great visceral cavities sometimes interfere seriously with adjacent structures when much enlarged. In the neck they may compress the trachea or the œsophagus, and cause suffocation or a complete inability to swallow food; or, it is said, they may give rise to severe cerebral symptoms by pressing on the great cervical vessels, or to irregularity of the heart's action by involving the pneumogastric nerves. Enlarged glands in the neck may hamper the movements of the lower jaw in mastication. In the armpit they may compress the axillary vessels and nerves, so as to cause much pain and swelling of the arm.

Pallor almost always becomes, sooner or later, a conspicuous symptom in Hodgkin's disease. The cheeks and the lips appear bloodless and waxy-looking; the more so as there is generally considerable subcutaneous

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œdema, the eyelids being swollen, and the aspect that of a patient recovering from scarlet fever. It must be added, however, that albuminuria has sometimes been present as a complication, and that tubal nephritis has been found after death, so that the œdema may be due to the presence of Bright's disease and not to anæmia alone.

The state of *the blood* is that of marked anæmia. When drawn, it is strikingly pale, and has been compared to diluted claret; it coagulates slowly and imperfectly. The red discs in a case that occurred at Guy's Hospital in 1877 were estimated at only 76 per cent. of the normal proportion, and a much greater reduction has been recorded.

The number of leucocytes is, in most cases, normal; but sometimes it is slightly increased, and in rare instances the excess is considerable, approaching that of splenic leuchæmia. In a foot-note to his article in Reynolds' System (vol. iv, p. 335), Dr Gowers cites no fewer than eight observations of extreme leuchæmia with enlarged glands and a spleen of normal size; besides two in which there was overgrowth of the splenic pulp, without any conspicuous growths, and two in which this last character was present. The proportion of leucocytes to red discs was, in all the last four cases, that of one white to four red corpuscles.

Splenic leuchæmia and Hodgkin's disease may thus be associated together in the same patient, as Virchow early remarked. A case of Dr Frederick Taylor's is recorded in the 'Pathological Transactions' for 1873, where the spleen weighed fifty-one ounces, and had the appearance seen in ordinary cases of leuchæmia, but there were also mediastinal and subpleural lymphomata of remarkable size.

The usual symptoms of severe anæmia accompany Hodgkin's disease. There is *dyspnœa*, which on exertion may become extremely distressing, and the respirations are accelerated, being often from 24 to 36 in the minute. *Hæmorrhage* and particularly epistaxis may appear, though less frequently than in splenic leuchæmia. The *temperature* of the body often rises to 100° or higher; in one instance it reached 103·2°. According to Dr Gowers, the pyrexia is sometimes continuous, with slight diurnal fluctuations; sometimes it lasts only for a few days at a time, the febrile periods being separated from one another by intervals of normal temperature; sometimes it is especially characterised by morning remissions, the daily range amounting to 3° or even more. In 1860 a man died in Guy's Hospital nine days after his admission, in whose case no diagnosis was made during life; he lay with his eyes closed, and he was scarcely sensible; his skin was hot, and he had occasional rigors; his spleen could just be felt; there was a mass of enlarged glands in the left side of the neck, and the autopsy showed that it was a case of Hodgkin's disease. A persistent high temperature appears to be a sign which, more than any other, indicates a speedy and fatal termination.*

The *course* taken by cases of Hodgkin's disease is very variable. Sometimes the patient is known to be ill during only a very short period before the occurrence of a fatal termination; and the lymphatic glands may then undergo very rapid enlargement.

Thus in 1867 a man aged thirty came under the author's care whose case was recorded in the 'Guy's Hospital Reports' for 1881. Except that a

* In a case under Professor Bäumler's care, reported from Freiburg-in-B. by Dr Brauneck, there was a temperature of 40·5 C. (105 Fahr.). 'Ueber einen Fall von mutipler Lymphombildung' (Hodgkin'sche Krankheit), 1886.

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month previously he had a slight cough and hæmoptysis (which probably were due to tubercular disease of the lungs, since this was found to be present at the autopsy) he was supposed to be well, and remained at his occupation as a hawker of fish until three weeks before his death. He was then suddenly seized with a dull heavy pain at his chest, and six days later, on March 16th, he became covered with purpuric spots. On the 21st hæmaturia set in. He also expectorated a quantity of blood, which seemed to come from the mouth. He was admitted on March 23rd. The spleen was then much increased in size, its edge being felt about half an inch below the ribs; but no enlargement of lymphatic glands was discovered. During the next few days his temperature ranged from 99·4° to 99·9°. The diagnosis was "purpura hæmorrhagica." On the 28th he had epistaxis. On the morning of the 30th, at about 7 a.m., he noticed, for the first time, that the glands of his neck were enlarged and tender. It was then found that many other glands of the body were likewise swollen, although not so tender. Notwithstanding that he was perspiring freely, his temperature was 103°. Extreme dyspnœa set in two days later, on April 1st; and he died, suffocated by œdema of the larynx, at noon on that day. On *post-mortem* examination the cervical and the axillary glands were seen to be enlarged, so that some of them measured an inch in their long diameter; they were soft, of a pinkish cream colour, and spotted with ecchymoses. The tonsils presented a similar appearance, and were half an inch thick. The thymus formed a large pear-shaped mass. The spleen weighed twenty ounces; it was pale and soft. The kidneys were very pale, and spotted all over with patches which looked as though they were suppurating. Dr Moxon found distinct evidence of leuchæmia: leucocytes were visible in large numbers in the liver between the hepatic cells, and as many as twenty-five were counted in a single short capillary vessel in the substance of the heart. Nevertheless, the blood was examined a day or two before the patient's death, without discovering any excess of white corpuscles. Thus the case tends to show that leuchæmia is no essential feature of the disease even when it is present, but rather an occasional complication.

Two cases, also of unusually rapid course, were recorded by Dr Paterson in the 'Edinburgh Medical Journal' for 1870. The first was that of a young woman aged twenty, who, having previously been plump and well-coloured, became towards the end of a first pregnancy very sallow, with hollow eyes, although she still said she felt well and in good spirits. Her confinement was followed by troublesome hæmorrhage, which, however, readily yielded to ergot. About the sixth day afterwards a marked change for the worse took place. The pulse became rapid, there was considerable heat of skin, the liver and the spleen were found to be enlarged, and the glands in the neck were also slightly increased in size. The blood was now examined, and it proved to be highly leuchæmic. Death occurred only five days later from suffocation, the cervical glands having in the meantime reached a great size, and the dysphagia having been so extreme that she could not swallow even a teaspoonful of water. Dr Paterson's second case occurred in a policeman's wife, who had become languid, pale, and sallow during the latter part of her first pregnancy, and who had considerable hæmorrhage after delivery. Soon afterwards a slight increase in size of the cervical glands was detected; it was also found that there was leuchæmia, and that the liver and the spleen were enlarged. The glands of the throat and neck and upper part of the chest underwent further increase, fever and restlessness

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set in, and she died of asphyxia only fourteen days after parturition. There was no autopsy in either case.

As a rule, however, the progress of the disease towards a fatal termination is slow. Dr Gowers gives a table showing the duration of fifty fatal cases, the length of which could be fixed with some degree of accuracy; thirty-three of them ended within two years. The most common mode of death is gradual exhaustion. But, as we have seen, suffocation sometimes occurs from pressure of the enlarged glands upon the trachea, and sometimes starvation from pressure upon the œsophagus. Epistaxis has occasionally been directly fatal. Coma, delirium, and convulsions, without discoverable anatomical cause, have been observed in some cases by Dr Southey. Pneumonia, œdema of the lungs, and pleurisy are not infrequent complications, and may be the immediate cause of death. Diphtheria of the fauces seems to have occurred more often than can be accounted for on the supposition of a mere coincidence.

Anatomy.—The morbid anatomy of Hodgkin's disease varies in different cases. The affected glands usually appear whitish yellow, waxy, smooth, and firm, both on surface and on section; but sometimes they are opaque, white, soft, medullary, and perhaps spotted with hæmorrhages, and in one case they were of a uniform deep reddish-grey tint. They have remarkably little tendency to caseate,* and only cohere in the later stages of their growth.

The *spleen* is only moderately enlarged, its weight in the cases that have occurred in Guy's Hospital having varied from eight to twenty-eight ounces. On section there are as a rule found scattered through its substance a number of firm whitish-yellow masses, of round or irregular shape, from the size of peas to that of hazel-nuts. Dr Wilks used to compare them to masses of suet in a pudding, or to the almonds in "hardbake." Sometimes the spleen in Hodgkin's disease presents only an indefinite mottling, or its tissue may be uniformly red and homogeneous.

This affection of the spleen in the case of a man aged sixty-seven, who died of cerebral hæmorrhage, was accidentally found at the autopsy, the glands being normal, both in size and in appearance. A second example is recorded by the present writer in the 'Path. Trans.' for 1870, p. 390, in a girl of seventeen; here there was also diphtheritic angina and colitis. Such cases may be distinguished as anæmia splenica (p. 626).

In other organs the growths are very variable in character. The *liver*, which may be greatly increased in size, sometimes contains distinct nodules, but more often it merely shows tracts of lymphoid tissue running along the portal canals, or minute nodules scattered between the lobules in such a way as to be distinctly recognised only with the microscope. Thus the state of the liver is often undistinguishable from that which occurs in splenic leuchæmia.

In a case under the writer's care in 1874 the liver weighed 88 and the spleen 83 oz., without leuchæmia or enlargement of lymph-glands (*anæmia hepatica*) ('Path. Trans.,' xxvi, p. 199).

The *kidneys* present similar combinations of diffuse interstitial growth with more or less sharply defined and rounded tumours.

The solitary *follicles* of the intestine, and also Peyer's patches are sometimes greatly swollen and medullary-looking; and it is worthy of notice that the *tonsils* and the follicles at the root of the tongue may be affected in the same way, since their enlargement can be seen during life (see cases by

* On this point see Mosler's paper ('Virch. Arch.,' lvi, p. 14).

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Dr Moxon, 'Path. Trans.,' xx, p. 369; and Dr Legg, 'St Barth. Hosp. Rep.,' vol. xi). In some cases the "lenticular" lymph-follicles of the stomach are also enlarged (Virchow, 'Krankh. Geschw.,' p. 509).*

Another organ which is accessible to clinical investigation is the *testicle*. In Dr Taylor's case, already referred to, each epididymis was enlarged so as to be two or three times the size of the testicle; and a similar condition was recorded in one of Hodgkin's original cases. Within the thorax there are sometimes enormous masses of growth. In Dr Taylor's case (where there was leuchæmia with a very large spleen) the anterior mediastinum contained a flattened tumour, one inch thick, with the left innominate vein running through its centre. In this instance there were also in each parietal pleura large flat nodulated bands of lymphoid growth of a red colour, running parallel with the ribs. In other cases the pericardium and base of the heart have been invaded, or the disease has spread into the lungs from their roots.

Not infrequently the *thymus* has been greatly enlarged and infiltrated with a soft white growth (Guy's museum, No. 3128).

The medullary tissue of the *bones* does not always escape. In a case examined by the author one tibia contained a soft rounded mass as large as a nut, which, however, had not a lymphoid structure, but was made up of a glistening fibrillated matrix with nuclei. There is sometimes found a diffuse change in the medulla, by which it is converted into a reddish-grey, semi-diffluent substance. The change is similar to, or identical with, that described above in *leuchæmia myelogenica* (p. 626).

Our present knowledge of the histology of the "adenoid tissue of His," and of its (leuco-) cytogenic function, throws clear light upon the close pathological relation of the spleen and lymph-glands with the red marrow of the cancellous tissue of the bones, with the thymus, the "solitary" follicles of the intestine, the "closed" or "lenticular" follicles of the tongue and of the stomach, with Peyer's patches and the tonsils, and with the diffused cytogenic tissue of the liver.

Diagnosis.—The recognition of Hodgkin's disease is generally easy at an advanced stage; but at the commencement, when the only symptom is a mass of glands in the neck, in an armpit, or in one of the groins, they may prove to be "simple lymphoma," or tuberculous, or syphilitic, or secondary to some deep-seated malignant tumour. The fact that degenerative changes, and even suppuration, have occurred in the packet of glands first affected, is not always a proof that the subsequent progress of the case will not be that of Hodgkin's disease. As regards syphilis, the case may be quoted of a man who was admitted into Guy's Hospital in 1867, having in the left side of the neck, reaching from the occiput to the shoulder, a tumour which was believed by Dr Moxon to consist of a gummatous enlargement of the lymphatic glands; after death, however, it was found to have its seat in the other tissues of the neck, the glands being embedded in it, but themselves unaltered. As an instance of secondary sarcoma of lymph-glands, the nature of which was unrecognised during life, may be cited the case of a girl aged ten whose body was examined in 1880, she having died immediately after an operation for the excision of a mass of glands in the left axilla and above the left clavicle. It turned out that there was a primary tumour in the left broad ligament, and that the lumbar glands

* In two cases at Guy's Hospital lardaceous degeneration was found in the viscera as well as in the enlarged glands, but in each case surgical operations had been performed, which led to suppuration.

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were also sarcomatous, as well as one of the mediastinal glands, from which the new growth was extending into the right auricle of the heart.

Such new growths if "innocent" are called adeno-lymphoma, *i. e.* a local tumour without anæmia and its concomitant general symptoms. If malignant, they are called lympho-sarcoma or, better, sarcoma of a lymph-gland (cf. vol. i, p. 87). In the latter case they are of harder feel and more painful. The proof of "malignancy" is reproduction in the lungs or other viscera; the distinction between lymphoma (or lymphadenoma) and lympho-sarcoma (or sarcoma beginning in a lymph-gland) is the histological distinction between lymphatic and sarcomatous growths.

Treatment.—In doubtful cases where only a single mass of glands is found, it is probably well to have the tumour excised as early as possible. Verneuil has recorded one striking case in which an immense mass was removed with perfect success, and the patient was still in good health seven years afterwards.

Of internal medicines, *arsenic* and *phosphorus* have been most recommended (cf. pp. 626 and 648).

The reputation of arsenic began with a case of Billroth's, in which the disease had existed for ten months, the patient being a woman of forty; the cervical, the axillary, and the inguinal glands were greatly enlarged; within a fortnight after the commencement of the treatment they were already reduced in size, and after two months she was discharged with only a single gland of the size of a nut on each side of the neck. No equal success seems to have been since obtained. Injection of liquor arsenicalis into the enlarged glands by a subcutaneous syringe has also been practised at Vienna. The writer once tried it on a patient of his own, but it produced pain and inflammation without apparent benefit. Phosphorus was first given by Verneuil; Dr Gowers in one case found that its administration was followed by a remarkable diminution in the size of the glands, and by improvement of the leucocytosis, but the patient died from intercurrent renal disease. It must be borne in mind that the glands have sometimes become much smaller shortly before death, independently of treatment.

Iron has often been given for the anæmia, but without any benefit; iodide of potassium is equally useless, unless by a happy error in diagnosis the disease is really syphilis; and cod-liver oil is as powerless in these cases as it is efficacious in those of tubercular disease of the lympharia.

Progressive diffused tuberculous disease of the lymphatic glands.—In the chapter on tubercle, affections of the lymph-glands were briefly alluded to (vol. i, p. 320). Instances are not very infrequent of nearly all the glands in the body being simultaneously, or in rapid succession, affected with tubercle, and a fatal illness results. A case in point, which has sometimes been wrongly cited as an example of Hodgkin's disease, was recorded more than a century ago by Morgagni (Epist. lxviii).

No fewer than ten such cases appear in the reports of *post-mortem* examinations at Guy's Hospital during the fifteen years from 1868 to 1882. Two or three of them have already been published. Thus Dr Goodhart related in the 'Guy's Hospital Reports' for 1873 the case of a man who was admitted under Mr Cooper Forster for disease of the right knee-joint, and who also had a large mass of glands in the right posterior triangle of the neck. He died after amputation of the thigh. At the autopsy the glands in Scarpa's triangle on the affected side were found to be enlarged, yellow,

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and of putty-like consistence. The inguinal, the iliac, and the lumbar glands on the right side were all very large, firm and yellow, those on the left side being healthy. Some of the glands in the portal fissure were as big as chestnuts. The bronchial glands on both sides were much enlarged, some being more than an inch long; they were cheesy, and disease seemed to be spreading from them into the lung itself on the left side, in the form of rounded yellow masses. The glands in the right axilla, and on both sides of the neck, were alike affected; and there was miliary tuberculosis of the lungs.

Another case was recorded by the author in vol. xxv of the 'Path. Trans.,' at p. 235. A woman aged thirty-five was admitted with a large suppurating glandular swelling in the left groin, and with another mass of swollen glands in the left side of the neck. She said she had been gradually wasting since her marriage two years before. She died at the end of a week. At the autopsy it was found that along the whole length of the spine there was a continuous mass of enlarged and suppurating glands. The aorta and the vena cava ran through a dense agglomeration of glands, some more than an inch long, many of which contained points of pus, while some were sloughing in the centre. The portal and bronchial glands were similarly affected. The right inguinal and the right iliac glands were in an earlier stage, and looked greyish white and granular on section. The axillary glands were little, if at all, involved. The spleen weighed twenty-four and a half ounces; it contained numerous yellow masses, some as large as walnuts. The lungs showed a few masses of the size of peas, but no ordinary tubercles; nor were there any tubercles in the intestines or the liver.

A third case was that of a man aged twenty-seven, admitted into a surgical ward on account of his having a swelling in one axilla, of the size of a hen's egg, attended with pricking pain; it was growing rapidly, but was moveable, and the skin over it, although red, was not adherent. It was excised, and was found to consist of large caseating and suppurating lymphatic glands. He died subsequently of pyæmia. At the autopsy similarly affected glands extended under the pectoral muscle, and all the mediastinal glands were tubercular; in the spleen there was a single tubercle softening into a cavity.

A somewhat similar case, also from our wards, was published by the writer in the 'Pathological Transactions,' vol. xxvi, p. 202. The patient was a woman of forty-seven, who had been ill for several months with bronchitis and recently with albuminuria, attended with a remarkable enlargement of the cervical, axillary, and inguinal lymph-glands. Some of these suppurated, and the temperature was occasionally raised. There was also a fluctuating swelling on the sternum. The immediate cause of death was dyspnœa from pleuritic effusion. There was double pleurisy, with tuberculous nodules, but no tubercle of the lungs, larynx, or intestine. Some degree of lardaceous degeneration in the spleen and kidneys was obviously secondary to the prolonged suppuration. The case was proved on histological examination to be throughout tubercular, and this conclusion was confirmed by the report of Dr Burdon Sanderson and Dr Green (*loc. cit.*, p. 206).

Of the remaining cases, five were in men, aged respectively eighteen, twenty-four, thirty-five, thirty-seven, and fifty-four years; and one in a woman aged thirty. In every instance, with one exception, it is noted that the spleen contained tubercles.

The duration of this remarkable form of adult tuberculosis was generally from six months to a year. It was attended with fever and with rapid wasting.

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It must be of practical importance to the surgeon to recognise cases of this kind, since he is very apt to be led to excise some prominent mass of glands from the neck or the axilla, a proceeding which is not likely to be successful. To the physician their chief importance lies in the diagnosis from Hodgkin's disease, with which they are generally confounded.

It is interesting to note the great frequency with which tuberculosis of the spleen is met with in association with a similar affection of the lymphatic glands. This fact was long ago pointed out by Bright in vol. iii of the 'Guy's Hospital Reports.'

Syphilitic lymphoma.—The hard bullet-like enlargement of the lympharia in constitutional syphilis must be carefully distinguished from Hodgkin's disease and from tubercular (so-called scrofulous) lymphadenitis (cf. vol. i, p. 283). The enlargement is moderate, the glands are freely moveable, indurated and painless. They are pathologically distinct from the soft suppurating and painful buboes which follow non-specific or inflamed Hunterian chancres; for syphilitic lymph-glands are "indolent," not inflammatory in origin, and are probably of the same character as the induration of the primary sore, the nodules of specific iritis, and the early stage of gummata.

Multiple sarcoma of the lympharia.—This condition, often called lymphosarcoma, is not uncommon, and particularly affects the mediastinal glands. It forms the greater part of what have been described clinically as intrathoracic growths and primary cancer of the chest. Its histology was detailed in the first volume of the present work (p. 80), and its clinical features in the chapter on Aneurysm and Thoracic Tumours (p. 115), for during life it closely resembles some forms of saccular aneurysm of the thoracic aorta.

CHLOROSIS.*—From the time of Hippocrates this name has been applied to an affection characterised by a pale, sallow colour of the skin—a form of anæmia occurring chiefly, if not exclusively, in young women. The peculiar complexion of this as compared with other kinds of anæmia is often overlooked. But although the special tint is not observed in every case, it is, when present, characteristic of chlorosis. The "greenish" olive hue is best seen in those girls who are naturally of a dark complexion; indeed, χλωρός, as descriptive of the pallor of fear or of death, is more applicable to modern Greeks and Spaniards than to the xanthochroic inhabitants of Northern Europe.

Ætiology.—At one time chlorosis was supposed to be constantly caused by disappointment in love. This is not the case; but it may be directly excited by a sudden shock, or by violent emotion, as in cases cited by Trousseau. In many instances it bears a close relation to the development of the menstrual function. Thus the age at which it shows itself is almost always between the fourteenth year and the twenty-fourth. It sometimes arises in a girl who has hitherto been robust, with a fresh complexion, and breasts showing no signs of immaturity; but Immermann remarks that such cases are seldom intractable. The catamenial discharge may have occurred for a certain length of time with more or less regularity, and there may be an interval after its cessation before the first signs of chlorosis are observed.

* *Synonyms.*—Morbus virgineus, Icterus albus, Leucophlegmasia virginum.—*Fr.* Pâles couleurs, Maladie de vierge.—*Germ.* Bleichsucht.

The Greek word used by classical writers is not χλώρωσις, but χλωρότης (from χλωρός, green, pale), of which the vernacular term "green-sickness" is the translation.

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Such cases have been supposed to indicate that amenorrhœa is the cause of chlorosis, but there seems to be no doubt that this is an error. Indeed, those who become the subjects of the disease have often been pale and delicate from childhood. Thus Dr Ashwell, in a paper in the first volume of the 'Guy's Hospital Reports' (1836), declared that chlorosis, when it afterwards assumes an aggravated form, has probably always existed from infancy. In many instances menstruation remains absent, and puberty can hardly be said to occur, the axillæ and the mons veneris continue to be devoid of hair, and the uterus may retain throughout adult life the form which is normally peculiar to childhood. Sometimes the catamenial flow is developed prematurely; and Niemeyer states that he never met with a case in which the menses appeared early while the breasts were still undeveloped, without obstinate chlorosis following.

One can readily understand that a girl in whom the blood-forming organs have never formed enough hæmoglobin to give a healthy tint to the cheeks, will suffer more than others from the establishment of the menstrual function, and why, therefore, chlorotic anæmia should be "a malady most incident to maids." In the same way we may account for the occasional development of a condition analogous to chlorosis in pale and delicate boys when the glandular activity of puberty begins, "a kind of male green-sickness."

Virchow has endeavoured to show that in many if not in all of those who become chlorotic at puberty there is, besides a natural deficiency of blood, a congenital hypoplasia or imperfect development of the heart or of the blood-vessels. He finds that the aorta is much smaller, and that its walls are much thinner than in healthy persons of the same age. Thus he speaks of instances occurring in the bodies of well-developed women, in which the aorta would hardly admit the little finger, whereas it ought to be large enough to receive the thumb; and he cites an observation, made by Rokitansky, of an abdominal aorta that was no bigger than an iliac artery should be or even a carotid. But, on referring to a brief description of some of Virchow's cases, published in 1872 ('Ueber die Chlorose und Anomalien im Gefässapparate'), we find that in several of them there was stenosis of either the mitral or the aortic orifice. Now, in its whole length the aorta has been repeatedly found extremely narrow in persons who were the subjects of obstructive disease of valves on the left side of the heart. But (with a few exceptions perhaps in the case of the mitral orifice) such affections are not congenital, but due to endocarditis occurring in childhood. Thus it seems that the hypoplasia of the aorta, instead of being itself a primary defect, is but a secondary result of the valvular lesion. Nor does it seem clear that when Virchow speaks of some chlorotic patients being dwarfed in stature he was sufficiently careful to exclude the existence of acquired disease of the heart, which is certainly capable of interfering with the natural growth of the body.

Symptoms.—These are generally identical with those of other forms of anæmia (p. 609). Beside breathlessness, feeble muscular power, and a rapid irritable pulse, vascular murmurs are seldom absent, and are more marked and constant than in any other form of anæmia of the same degree of severity. A systolic bruit in the pulmonary area (probably dependent on dilatation of the trunk of the artery of the lungs) is very frequent, usually accompanied by a systolic murmur in the carotid and other arteries. The most constant and characteristic bruit is a continuous *humming sound** in the internal jugular

* *Fr.* Bruit de diable.—*Germ.* Nonnengeräusch.

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vein (p. 613). This is sometimes better heard on the left, more often on the right side; it is increased in loudness during deep inspiration, and has a slight respiratory rhythm even when the patient is breathing quietly; its locality, continuity, and loud harsh quality are very distinctive. The sound is almost exactly imitated by holding a large univalve shell to the ear.* Though rarely absent in cases of chlorosis, it may be heard in other forms of anæmia, and not very infrequently in healthy persons if the head be turned away and the stethoscope pressed firmly down.

A chlorotic patient does not usually grow thin; she may even become fatter than before. There is seldom, if ever, any dropsy beyond slight œdema of the ankles. It does not appear that retinal hæmorrhage has been observed, and certainly no other local bleeding occurs; nor is there menorrhagia, but amenorrhœa.

One symptom of anæmia in general is constant and often extreme in chlorosis—a torpid state of the colon. The *constipation* of chlorotic girls often lasts for many days and sometimes for a fortnight; even if the irritation of scybala occasionally produces what is called diarrhœa, the fæcal accumulation is still unaffected. This symptom, like the amenorrhœa, has been supposed to be the cause of the anæmia by poisoning the blood with retained fæcal products; but the first of the triad is certainly pallor. Leucorrhœa is frequently present.

The blood in chlorosis is pale in proportion to the severity of the case. The discoloration depends chiefly upon deficiency of hæmoglobin in each corpuscle, and the number of the red blood-discs is but little diminished; nor are their size and shape altered, as in the more severe form of anæmia to be next described. This marked *achromatosis*, slight *oligocytosis*, and absence of *pœcilocytosis* can be readily observed by help of the hæmocytometer and hæmoglobinometer above described (p. 611).

Diagnosis.—To distinguish chlorosis from other varieties of anæmia, we depend first on the age and sex of the patient, the peculiar tint, the hæmic murmurs, the absence of loss of flesh, and the presence of constipation and amenorrhœa. Bright's disease, mitral disease, phthisis, lead-poisoning, all produce secondary anæmia, which, when it affects young women, may be mistaken for chlorosis.

Practically we have most often to make sure that the case is not one of gastric ulcer, phthisis, or idiopathic anæmia of Addison.

It has often happened that a chlorotic girl has been unexpectedly attacked by acute and fatal peritonitis; and an autopsy has discovered the cause of both anæmia and death in a perforating ulcer of the stomach.

The early stages of pulmonary disease are frequently overlooked in cases of anæmia, and the only way to avoid the mistake is to take the evening temperature once or twice every week, and to examine the subclavicular spaces from time to time.

In 1861 a girl aged eighteen, who was admitted into Guy's Hospital for chlorosis, gradually sank and died; a large caseous mass, which seems to have produced no marked symptoms, was found in the cerebellum, and there were a few scattered tubercles in the lungs.

One distinction from Addison's idiopathic form of anæmia, to be next

* "Tu quate, sonus abit; tu levia tange labella
Auribus attentis, veteres reminiscitur ædes,
Oceanusque suus quo murmure murmurat illa."—LANDOR.

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described, lies in the diminution of colour of each blood-disc under the microscope being much greater than their diminution of number. Thus in a recent case under the writer's care, the hæmoglobinometer showed the diminution of colour to be 15 per cent., while the number of red corpuscles was about 3,500,000 per cubic millimetre, which is common in women. Another distinction is the absence of pyrexia, and a third the absence of hæmorrhage, especially retinal hæmorrhage. The good effects of iron would confirm the diagnosis. As a matter of fact we do not find that fatal or essential anæmia begins at the age or with the symptoms of chlorosis.

Prognosis.—Chlorosis is never directly fatal, but in exceptional cases it seems to be the starting-point of phthisis; hysteria, chorea, gastric ulcer, and exophthalmic goitre are particularly apt to occur in its course; and its presence aggravates the danger of enteric or other fevers.

Some women have to take steel year after year in order to keep free from anæmia. Indeed, those who have been chlorotic in early womanhood are very liable to recurrence in later life, especially if they become exhausted by child-bearing or by lactation.

Treatment.—Chlorosis resembles secondary anæmia in being amenable to treatment by iron. Splenic and lymphatic anæmia, leuchæmia, and the grave form of idiopathic anæmia to be next described, are seldom benefited, even for a time, by ferruginous medicines. But in most cases of chlorosis sufficient doses of steel act in a "specific" (*i. e.* constant and unexplained) manner. The famous Griffith's mixture (Mist. Ferri Comp.) is deservedly trusted in England, and the scarcely less famous Blaud's pills in Germany.* Mist. Ferri Co. and Pil. Aloes et Myrrhæ go well together. Another excellent combination is sulphate of iron, sulphate of magnesia, and aromatic sulphuric acid. The alkaline preparations are generally most serviceable, but now and then the Tinctura Ferri Perchloridi succeeds better.

The rules of treatment are to exhibit laxatives along with the preparations of steel, to increase the latter until a decided effect is produced, and if it disagrees, to diminish the dose or change the preparation but not to abandon the drug.

The effect of iron on the blood often appears marvellous, especially when the obvious improvement in the patient's appearance and in her breath and strength is confirmed by watching the daily increase of the number and colour of the corpuscles by means of the hæmocytometer and hæmoglobinometer.

The fact that iron is so important a constituent of hæmoglobin suggests that it acts directly as a food; but to make this view tenable one must assume that there has been a deficiency of iron in the dietary of those who are thus benefited; and there is seldom any evidence of this. One must therefore suppose that it stimulates the process by which hæmoglobin is developed and red corpuscles formed.

In some cases in which there is much gastric disorder, it may be advisable to give only bland preparations, such as the ammonio-citrate or the potassio-tartrate or the tasteless solution of dialysed iron. But it often happens that the tincture of the perchloride, or the sulphate, can be borne by patients who have a pale flabby tongue, nausea, vomiting, and even pain after food; and such treatment is generally attended with signal success. Large doses seem to be much more serviceable than small. Probably their

* ℞ Ferri Sulph. gr. iiss, Pot. Carb. gr. iss, Sacch. gr. j, Tragac. gr. ij—iv. Misce, ft. pil. Signetur: Two, three, and at last four or five to be taken thrice daily.

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advantage lies mainly in the fact that only a small part of what is swallowed is absorbed into the blood; the greater portion is converted into a sulphide in the small intestine, and passes away in the blackened fæces.

In the present chapter we have surveyed the various clinical forms of anæmia, and have found that they may be classified in several ways. The most important is that which follows their *ætiology*, and distinguishes the want of blood that is the result of direct hæmorrhage, that which in other ways is clearly secondary and symptomatic, that which is associated with special anatomical lesions of the spleen and other lymphatic organs, and the chlorosis which is associated with the female sex and the development of the menstrual function. None of these varieties are strictly idiopathic, *i. e.* primary, essential, and unexplained by antecedents or circumstances. But there remain certain rare cases which come under none of the preceding heads, and are in the proper sense of the term *idiopathic*. This, therefore, appears to be their most appropriate designation until we learn something of their true cause; and it happens to be also the historically appropriate name, since it was proposed by the physician who first recognised and described this remarkable form of disease in the course of his investigation into the ætiology of anæmia.

Of the *pathology* of anæmia we are too ignorant for it to furnish a satisfactory ground of distinction. But we may more or less probably divide cases on this basis into those which depend on want of power to form blood (*i. e.* to form hæmoglobin) and those which depend on an accelerated process of destruction of red blood-corpuscles. The former would be marked by wasting and other signs of malnutrition, with pale urine. In the latter, anæmia would be unaccompanied with loss of flesh, and the urine would be dark. The former cases would be benefited by good food, good air, and preparations of steel; the latter would not.

An *anatomical* classification, based on the histology of the blood, is not impossible. We should thus recognise cases of moderate diminution of the number of red corpuscles without other change (mostly symptomatic), cases of moderate diminution in number with great deficiency of hæmoglobin (chlorosis), cases of great diminution of red corpuscles in number with increase of leucocytes (leucocytosis and leuchæmia), and, lastly, cases of extreme and progressive diminution of the discs, with microcytosis and pœcilocytosis.

Lastly, we might seek a *clinical* basis for a classification of anæmia, and divide it into slight and recoverable kinds, those which show a more obstinate or an ingravescent character, and those which, however they begin, after a more or less uniformly progressive course show a pernicious or malignant character, and end in death. This was the point of view of Biermer Gusserow, and most German writers who, writing for the most part in South Germany or Switzerland, and sometimes from a gynæcological standpoint, have included cases of chlorosis, of menorrhagia, malaria, gastric ulcer, starvation, and other kinds of secondary anæmia along with Addison's idiopathic cases to form a heterogeneous assemblage which only agree in a fatal issue.

There is, however, another clinical classification which agrees better with pathological facts. We may recognise (*a*) symptomatic or secondary anæmia, moderate in its symptoms and curable if its cause can be removed; (*b*) more severe anæmia of toxic or malarial origin, in which special medi-

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cation is necessary, and is followed by success; (*c*) chlorosis, with more or less grave symptoms, but curable by steel; (*d*) the grave and more or less uniformly *fatal* forms, characterised by *pyrexia* and *hæmorrhage*, in addition to the other signs of anæmia, intractable to steel, and benefited, if at all, by *arsenic*. This last division includes anæmia lymphatica (Hodgkin's disease), leuchæmia, and Addison's anæmia, which, different as they are in anatomy and pathology, form a natural clinical group, and equally deserve the epithet of "pernicious," "progressive," or "grave."

IDIOPATHIC OR PERNICIOUS ANÆMIA.*—The history of the recognition of the last-named member of the great class which forms the subject of this chapter is as follows:—In the year 1855, the late Dr Thomas Addison, in his work on 'Disease of the Suprarenal Capsules,' states that the discovery of that disease had been made by him while seeking in vain to find a cause for a remarkable form of fatal anæmia, cases of which had for a long time occasionally come under his observation. To this affection, he added, he had been accustomed in his clinical lectures to apply the term "idiopathic," by way of distinction from such anæmic states as could be traced to "the usual causes or concomitants," among which he enumerated chlorosis. Ever since this form of anæmia has been recognised by all who have been his colleagues or successors at Guy's Hospital; and allusions to it have been made in successive volumes of our 'Reports' and elsewhere, particularly by Wilks in the 'Reports' for 1857, and in his 'Lectures on Pathological Anatomy' (1859). Dr Frederick Taylor published in the volume of our 'Reports' for 1878 no fewer than twenty-three cases which had been recorded year by year since 1853.

We were therefore not a little surprised to find that when Biermer, of Zürich, described the same conditions in 1868 under the name of "progressive pernicious anæmia," it was thought at first that a new disease was brought to light, and afterwards that it had been lost sight of, even by Addison's pupils, until it was rediscovered by the Swiss observer.

Even before Addison's work appeared, isolated cases had been published by Coombe in 1823, by Piorry and Marshall Hall, and by Dr Barclay in the 'Medical Times' for 1851. Lebert, who was then at Zürich, wrote on what he called "essentielle Anämie" in 1858, without knowledge of Addison's observations, and his cases were not of the same kind. Cases recorded in New England by Channing, as early as 1842, have been discovered by Drs Pepper and Musser ('Philadelphia Med. News,' October, 1882).

The following is Addison's original description of this remarkable disorder, published in 1855:

"For a long period I had from time to time met with a very remarkable form of general anæmia occurring without any discoverable cause whatever, —cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease.

"Accordingly, in speaking of this form in clinical lectures, I, perhaps with little propriety, applied to it the term 'idiopathic,' to distinguish it from cases in which there existed more or less evidence of some of the usual causes or concomitants of the anæmic state. The disease presented in

* *Synonyms.*—Primary, essential, or idiopathic anæmia—Addison's anæmia.—*Fr.* Anémie grave, Anémie essentielle.—*Germ.* Progressive perniciöse Anämie—Anæmatosis (Pepper).

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every instance the same general character, pursued a similar course, and, with scarcely a single exception, was followed, after a variable period, by the same fatal result.

"It occurs in both sexes, generally but not exclusively beyond the middle period of life; and, so far as I at present know, chiefly in persons of a somewhat large and bulky frame, and with a strongly marked tendency to the formation of fat.

"It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to his earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless; the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitation being produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles. The debility becomes extreme, the patient can no longer rise from his bed, the mind occasionally wanders, he falls into a prostrate and half-torpid state, and at length expires. Nevertheless, to the very last, and after a sickness of perhaps several months' duration, the bulkiness of the general frame and the obesity often present a most striking contrast to the failure and exhaustion observable in every other respect.

"With perhaps a single exception, the disease, in my own experience, resisted all remedial efforts, and sooner or later terminated fatally.

"On examining the bodies of such patients after death, I have failed to discover any organic lesion that could properly or reasonably be assigned as an adequate cause of such serious consequences; nevertheless from the disease having uniformly occurred in fat people, I was naturally led to entertain a suspicion that some form of fatty degeneration might have a share, at least, in its production, and I may observe that in the case last examined, the heart had undergone such a change, and that a portion of the semilunar ganglion and solar plexus, on being subjected to microscopic examination, was pronounced by Mr Quekett to have passed into a corresponding condition" (pp. 211, 213, in Addison's collected works, republished by the New Sydenham Society in 1868).

Dr Wilks stated in the 'Guy's Hospital Reports' for 1855 (3rd series, vol. i, p. 363) that "in that class of cases which has specially gained the attention of Dr Addison, and which he has designated idiopathic anæmia," no excess of white corpuscles is found in the blood.*

* Again, in his 'Pathological Anatomy' (1859), Dr Wilks wrote, "We occasionally meet with cases of fatal anæmia where no disease is found in the body, &c.;" and again, "Those cases of simple anæmia which have been called idiopathic." Moreover, in the 'Guy's Hosp. Reports' for 1857 (3rd series, vol. iii, pp. 205—211), and 1859 (p. 108), he put several cases on record, referring them all to the simple or idiopathic form of anæmia described by Addison.

Dr Habershon published a case as "Idiopathic Anæmia" in the 'Lancet' for 1863 (vol. i, p. 518). Trousseau referred to Addison's observations in the first edition of his 'Clinique Médicale,' 1865 (tome iii, p. 533). So also Dr Frederick Taylor, in the 'Guy's Hosp. Reports,' 3rd series, vol. xxiii; Dr Bramwell, in a paper published in the 'Edin. Med. Journ.' for November, 1877; Dr Stephen Mackenzie, in a lecture published in the

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A full discussion of the question of priority will be found in the writer's article in the 'Guy's Reports' for 1882 (vol. xli, p. 236).

Ætiology.—Among the fifteen cases (all in women) described by Biermer in 1871, as also in those previously recorded by Lebert and afterwards by Quincke and by Gusserow, there were many of severe anæmia in women which were not idiopathic, but secondary to pregnancy or parturition or menorrhagia, or were combined with amenorrhœa as extreme forms of chlorosis. Thus Lebert describes his cases as "Puerperal chlorosis," and Gusserow his as "hochgradigste Anämie Schwangerer."

In another group of cases of grave or even fatal anæmia the origin of the disease was in protracted diarrhœa, or gastric ulceration, or (as in some of Immermann's and Quincke's cases from Basle and other parts of Switzerland) privation and poverty.

In other cases again the marrow of the bones has been found after death changed in the way described above (p. 623). It is doubtful whether in some cases this may not be a concomitant effect rather than the cause of anæmia. If the latter view is accepted these cases will fall under anæmia myelogenica (pp. 626, 632). But in many cases of fatal idiopathic anæmia the bones have been carefully examined, and no such change has been found.

Lastly, obscure cases of severe and incurable anæmia have been found after death to be due to the presence of *Sclerostomum duodenale* (p. 294), or, as believed, to that of *Bothriocephalus latus* (p. 285).

Instead of correcting their mistaken diagnosis, some writers have published these and similar cases as "progressive pernicious anæmia" due to medullary degeneration, or to gastric disease, or to parasitic worms, and have even hinted that like causes would be found in all instances if looked for.

But Addison's discovery was that patients may die from want of blood which is not preceded by hæmorrhage or exhausting discharges, which does not originate in malaria or starvation, and is not explained by organic or parasitic disease discovered after death. The anæmia is not accompanied by excess of leucocytes in the blood, nor by anatomical changes in the spleen, the lymph-glands, or the marrow, nor is it associated with amenorrhœa and the other symptoms of chlorosis. It is not uniformly "pernicious," it is not continuously "progressive;" but it is "grave," and, so far as our present knowledge goes "primary," "essential," or "idiopathic."

No doubt these cases have their true cause; no disease is idiopathic in the sense of being spontaneous. But their causes are not those which are known to produce anæmia; they are at present as unknown as those of idiopathic epilepsy or psoriasis or osteo-arthritis. No doubt some rare cases of apparently secondary anæmia or of chlorosis may go on from bad to worse, uninfluenced by treatment, and at last develop the characteristic symptoms of the deepest and gravest anæmia, just as plumbism or cerebral

'Lancet' in 1878, and his three lectures in the 'Brit. Med. Journ.,' Jan., 1891; Dr Coupland, in his 'Gulstonian Lectures' (1881), and the present writer, in a paper published in 'Virchow's Archiv' six years before (Bd. lxv, 1875). Not only Addison's colleagues and pupils at Guy's Hospital, but also Dr Bristowe, Dr Quain, Dr Sutton, and Dr Howard, in Canada, were perfectly aware of the remarkable form of anæmia described by Addison.

Lépine, in France ('Revue Mensuelle,' January, 1877); Gardner and Osler, in Montreal ('Canada Med. and Surg. Journ.,' March, 1877); Osler (Pepper's 'System of Medicine'); and Pepper and Musser, in Philadelphia ('American Journ. of Med. Sc.,' October, 1875, and April, 1877), confirm the same judgment. It is surprising that in Professor Immermann's article on the disease in 'Ziemssen's Handbuch' its recognition is still ascribed to Biermer.

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tumours may produce typical epileptic fits, or as frequently repeated rheumatism or gout or gonorrhœal synovitis may produce at last, more or less completely, the characteristic lesions of osteo-arthritis. But the important clinical fact remains that patients may die of extreme anæmia without any discoverable cause; while in the symptoms during life, in the distribution as to age and sex, and in the appearances after death, these cases resemble one another and form a natural group.*

Idiopathic and fatal anæmia was at one time supposed to be particularly common among peasant women in Switzerland, but that probably depended on compilers including uterine and malarial cases. It is always rare, but may be seen in its typical form in France, Germany, and America, as well as in Great Britain,—in fact, wherever there is a large population and competent observers.

Age and sex.—Among the points in which idiopathic anæmia differs widely from chlorosis is its range with respect to age and sex. Of twenty-eight cases that occurred at Guy's Hospital from 1859 to the end of 1879, sixteen were in males, twelve in females. Of eight more, supplied by the present medical registrar, Dr E. W. Goodall, which ended fatally in the next ten years (1880—1889), five were in males and three in females. Putting the 36 cases from 1859 to the end of 1889 together, we have 21 men and 15 women. Of these 36 patients, 2 only were under twenty, both being boys; 4 occurred between twenty-one and thirty; 10 between thirty-one and forty; 10 between forty-one and fifty; 9 between fifty-one and sixty; and 1 at sixty-eight.

In the table published by the present writer in the 'Guy's Hospital Reports' for 1882 (vol. xli, pp. 293—303), of 102 collected cases of Addison's anæmia, all adequately examined during life, and all verified by autopsy after death, there were 6 between seven and fifteen years of age, 4 between fifteen and twenty, 29 between twenty-one and thirty, 26 between thirty-one and forty, 21 between forty-one and fifty, 13 between fifty-one and sixty, and 4 between sixty and sixty-nine. Thirty-six cases, since collected from American sources by Dr Musser, agree very closely as to the ages of the patients.

In the tabular statements of Heinrich Müller, Eichhorst, and some other observers, there is marked excess in number of female patients over male. Of 44 recorded at Zürich only 9 were men. But this depends on including secondary anæmia, due to parturition, pregnancy, or lactation, cases of chlorosis, and cases which recovered or which were unverified by inspection after death. If from Eichhorst's list of only 30 men to 65 women we exclude all but primary cases, the numbers are reduced to 12 to 11, and this is nearer the truth.

Among 107 cases collected by the writer there were 48 in men and 59 in women. In Dr Coupland's 110 cases (some identical with the last) the figures were 56 to 54, and in Dr Musser's 39 they were 24 to 15. So that there is either no sexual disposition to the disease, or the disposing factors in each sex are counterbalanced.

* Dr Bramwell has recorded two cases of foreign sailors, in each of whom "pernicious" anæmia appeared to arise out of an attack of yellow fever. Dr Stephen Mackenzie, in his valuable lecture in the 'Lancet' (1878), cites three instances in which it followed a severe mental shock; one patient had accidentally poisoned her father instead of giving him his medicine, another had seen a child run over in the street, and the third had been attacked by a sheep in a field, immediately before the anæmia set in. Dr Wilks has quoted similar cases ('Brit. Med. Journ.,' November 28th, 1884). So also Lépine, Coupland, and Musser.

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Symptoms.—The origin of the disease is rarely definite. When a patient comes first under observation his usual story is that for some weeks or months he has been getting paler, weaker, and more breathless.

The anæmia is extreme. But gradually the complexion, instead of being white, becomes of a clear lemon-yellow tint, so that the disease has been mistaken for jaundice. If, however, the conjunctivæ sometimes show a similar colour, this depends on the presence of unusually yellow adipose tissue beneath the mucous membrane.*

As a rule there is no wasting; the face and limbs are plump, and there is no deficiency of subcutaneous fat; only in a few exceptional cases has emaciation been recorded. Most patients have a bad appetite, but some continue to eat well. Dryness of the mouth and of the throat is often complained of; the breath is sometimes fœtid; nausea and vomiting are frequently present, especially in the morning, and there is occasionally pain after food; the bowels have been constipated in some cases, relaxed in others. In 31 cases recorded by Dr Hale in the current number of 'Guy's Hospital Reports' (xlvii, p. 162) more than half the patients suffered from vomiting and more than a third from diarrhœa.

The pulse is rapid and jerking. Pulmonary, arterial, and jugular murmurs are common.

There is always marked dyspnœa on exertion, and often palpitation. One man under treatment at Guy's Hospital said that every muscular effort caused pain at the back of the head, and this occipital headache is, perhaps, more common than that of the vertex.

Many patients have epistaxis again and again; in women sanguineous vaginal discharges frequently occur; in some persons the gums bleed or purpuric spots appear on the legs, but neither of these symptoms is common.

With the ophthalmoscope *retinal hæmorrhages* can often be detected. Biermer seems to have been the first to notice them. They appear either as linear striæ, or as rounded spots or patches, which may have whitish or yellowish centres, sometimes consisting (according to Manz) of accumulations of leucocytes. The optic discs are said to be swollen in some cases, their vessels to be tortuous, and the retinæ generally to have a peculiar smoky appearance. A boy aged ten, who came under Dr Mackenzie's care, had well-marked optic neuritis. As a rule there is no defect of vision; but the reason why one of Immermann's patients went to the hospital was because one eye had suddenly become blind. A patient of the writer's recovered after hæmorrhage had deprived him of nearly half the field of vision of one eye.

The *urine*, as stated by the writer in 1882, in some typical cases of idiopathic anæmia, is of a deep clear colour, one of the characters which point to destruction rather than deficient formation of hæmoglobin. This point has since been taken up by Dr Mott ('Lancet,' March, 1889, and 'Path. Trans.,' vol. xl, p. 127) and Dr Hunter ('Pract.,' 1880). It is non-albuminous; urea is deficient. The ankles commonly become more or less œdematous, and sometimes the face is puffy. In the cases observed in Guy's Hospital there has been no considerable ascites, nor have the pericardium and the pleuræ contained more than a few ounces of serum after death.

* In a case of a clergyman who died at the age of seventy from extreme anæmia, recorded by Dr Douglas, of Newbury, jaundice was actually present as a complication; but as there was no autopsy, there may have been carcinoma or some other organic disease to explain it ('Lancet,' July, 1882, p. 140).

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Another characteristic symptom of Addison's anæmia is irregular *pyrexia*. It was first noticed by Immermann, and it has repeatedly been observed at Guy's Hospital. Its course is uncertain; the thermometer sometimes rises to 104°, or even higher, and after a few days of high temperature there is perhaps a more or less prolonged interval of apyrexia. As a rule there are no subjective symptoms of fever. In some cases the temperature rises at an early period of the disease, but usually not until an advanced stage. Before death the thermometer often falls to 97°, or even to 95°.

The red discs of the blood are very often found altered in size and in form (*pœcilo-cytosis* of Quincke and Eichhorst), or with their hæmoglobin separated from their substance; but such changes are not peculiar to this form of anæmia, as was once supposed. In some cases no abnormal microscopic appearances can be detected in the blood-discs.

The number of corpuscles is often diminished to 1,100,000 per c.c.—very unusual, even in extreme chlorosis, where 1,300,000 is the lowest number the writer has seen. It may reach 750,000, 428,000, or (as in a case of Lépine's) 378,750. In a case under Worm Müllor, of Christiania, reported by Laache, it fell to 360,000.

Some blood-discs are pale and large (8 or 9 instead of 7 or 7·5, or even 14 μ in diameter), others are small and deeply coloured (microcytes), as first noticed by Dr Leared in 1858 ('Path. Trans.'), and these are probably more constantly present; they are not, however, as Eichhorst thought, pathognomonic.

Drs Davy and Mackern observed that hæmoglobin readily leaves the disc ('Lancet,' 1877, ii, p. 642), and Dr Copeman that it easily crystallises ('St Thomas's Hosp. Rep.,' vol. xvi).

The white corpuscles are not increased in numbers, even relatively, to the red, so that they must share in the atrophy of the histological elements of the blood. They maintain the power of amœboid movement on the warm stage.

Diagnosis.—This depends in part upon our definition of the disease and on the rigour with which we apply the terms of our definition. Moreover, it cannot be certain until verified by an autopsy. But when the following characters are present, we may with considerable confidence make the diagnosis of idiopathic or essential anæmia:—(1) Absence of organic disease and of any sufficient cause of anæmia,* particularly of amenorrhœa, menorrhagia, and pregnancy; (2) severe and ingravescent anæmia, with considerable diminution in number (to one half and under) of the red blood-discs, together with the presence of pale large and dark small corpuscles; (3) absence of emaciation; (4) occasional pyrexia; (5) hæmorrhages, and especially retinal hæmorrhage.

The diagnosis of primary pernicious anæmia has been made in cases

* It must not be imagined that to determine the absence of preceding loss of blood is always easy. From motives of delicacy persons will sometimes conceal the fact that there has been draining from hæmorrhoids, or from the uterus. Moreover, serious bleeding may occur *per anum* without the patient's being aware of it. In 1880 I was called to see, with Mr Earle, of Brentwood, a young lady who had fallen into a state of the most alarming prostration and bloodlessness. She had no doubt been ailing for some time, bnt she had been able to play lawn tennis until two days before my visit. She positively assured us that she had noticed no hæmorrhage from any of the mucous surfaces, and some days passed before her mother made the discovery that the evacuations from the bowel contained blood. Ultimately an ulcer was detected in the rectum. I am inclined to think that the suddenness of the girl's illness would almost have justified one in asserting that there must be some such cause for it.—C. H. F.

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which have afterwards proved to be secondary to internal cancer, ulcer of the stomach, phthisis, mitral disease, or morbus Brightii.

The likeness to *morbus Addisonii* remarked by Bristowe, Broadbent, Pepper, and other writers, is less frequent and less close than has been supposed. In the latter disease the patient is pale, but not excessively anæmic, the blood is normal or nearly so, and there is decided emaciation. The bronzed colour of the skin is in strong contrast with the clear yellowish pallor of Addison's anæmia; retinal hæmorrhage and pyrexia are absent, and there is frequent complication of phthisis or caries of the spine.

The distinctions from *chlorosis* have been already pointed out. Apart from the age and sex of the patient, the absence of hæmorrhage and pyrexia and the good effect of martial preparations distinguish all but very exceptional cases of chlorosis.

Experience shows that there is danger of mistaking essential anæmia for *jaundice*. In 1877 a man aged thirty-eight was in the clinical ward of Guy's Hospital for seventeen days with what was believed to be either acute yellow atrophy of the liver or cirrhosis. He became delirious and violent, and had to be placed in a separate room; afterwards he was insensible, but his alarming symptoms ultimately subsided and he was discharged. However, three months later he returned to the hospital, and it was then obvious that his disease was Addison's anæmia. He sank and died, and the liver was found to be healthy. Other instances of the same kind, and scarcely less striking, have occurred.

In the year 1866 a man aged sixty-seven lay for a long time in the clinical ward of Guy's Hospital with what was believed to be the idiopathic anæmia of Addison, but at the autopsy it was found that the lungs were full of miliary tubercles, and that the liver, the spleen, and the kidneys also contained them in smaller numbers. In 1879 a woman died aged fifty-nine, whose skin had been of a lemon-yellow colour, and whose blood had been found to contain red discs of irregular form, so that the diagnosis was supposed to be beyond question. However, the heart weighed twenty ounces, the minute arteries in the pia mater were greatly thickened, and the kidneys, although large, were hard and glistening, and showed an excess of fibrous tissue under the microscope. It was a case of chronic Bright's disease.

When there is a doubt as to the possible presence of the *anchylostomum* in the case of anæmia, it is important to examine the fæces of the patient with a microscope, since the ova of the parasite can be recognised in them without difficulty.

The nearest allies of idiopathic anæmia are the various forms of anæmia lymphatica, particularly that in which the marrow of the bones is alone affected (*anæmia myelogenica*). Indeed, although the distinction between the two is well ascertained, the diagnosis must in some cases depend upon the result of inspection after death.

Morbid anatomy.—The only changes found *post mortem* are the result, not the causes, of the profound anæmia. The blood is pale, thin, and fluid, "like washings of muscle." Hæmorrhages are found in the retina and various internal organs. The fat is of a deep yellow tint, and the muscles of a remarkably dark hue. There is more or less effusion of dark-coloured serum in the great cavities. The heart is in a state of fatty degeneration (p. 51), occasionally the diaphragm and other muscles, and sometimes the liver and kidneys also. The gastric glands have been found atrophied by

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Dr Austin Flint, Ponfick, Dr B. Fenwick ('Lancet,' 1877), and other pathologists. But this condition is not constant; it occurs without marked anæmia, and it would, if an efficient cause of disease, lead, one would suppose, to wasting rather than pallor. The spleen is not enlarged. Degeneration of the semilunar ganglia was described by Quekett in one of Addison's original cases, and has since been observed by Eichhorst, Brigidi, and others; but it is more frequently absent, and is probably of no significance.

Pathology.—A satisfactory theory of this remarkable disease is still to seek. There is, however, every likelihood that it belongs to the group of anæmia from destruction of blood-discs, not to that of anæmia from impaired formation of hæmoglobin. For, as the writer remarked in 1882, "the urine is often of a deep colour.* There is a peculiar yellow tint of the skin which simulates slight jaundice. The fat and muscles are often of remarkably full and saturated tint, contrasting with the pallid œdematous look of the other organs, and in one case the blood gave the hands a yellow stain. Pigmentation of the serous membranes has occasionally been noticed, and it is quite possible that the excess of iron which has been detected in the viscera is derived directly from broken-up hæmoglobin." This last fact, first observed by Quincke in 1876, and confirmed by Dr William Hunter ('Lancet,' Sept. and Oct., 1888) and Dr Stevenson ('Guy's Hosp. Reports,' xlvii, 166), of great accumulation of iron in the liver, and to a less extent in the other organs, even when steel has not been given as a drug, seems to point to increased destruction of hæmoglobin.† Almost every symptom during life, and all the appearances found after death, are the physiological results, not the causes, of severe and long-continued anæmia.

Course and prognosis.—The course of the disease is not uniformly "progressive." On the contrary, there are often decided pauses, and even apparent improvement. The duration is commonly for three months to a year. In 15 cases it is given as from five to eight weeks. In two cases which occurred at Guy's Hospital the disease is said to have run on for three years, and one patient stated that for seven and a half years he had been getting paler and weaker. In ten it lasted between eighteen months and two years.

According to Immermann the fatal termination is sometimes sudden from syncope; but usually it approaches very slowly, there being delirium, apathy, or (*coma vigil*) complete insensibility for two or three days before death, while sometimes a cadaveric odour has been exhaled from the body.

As to the possibility of recovery from the disease, there is good reason to believe that several cases have permanently recovered under, and we may hope as the result of, treatment. Addison himself met with one such case. Other writers have recorded a few, and twenty authentic cases are collected in the 'Guy's Hosp. Reports' (vol. xli, p. 304).

It must be remembered that relapses are common in this as in other forms of anæmia.

A patient was sent to the writer in March, 1880, by Dr Thurston, of Ashford. He was then fifty-three years old, and was well nourished, but for six months had been growing pale and weak. The temperature was

* It, however, must not be forgotten that bilirubin and urobilin contain no iron.

† Dr Hunter has succeeded in imitating the destruction of hæmoglobin and the deposition of iron in the liver by the administration to animals of toluylene-diamine.

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normal, the urine dark, and the fundus oculi pale without retinal hæmorrhage. The case was regarded as either idiopathic anæmia or internal cancer. In October he was paler than ever, with more dyspnœa and œdema of the ankles, and soon after an attack of pyrexia came on. In February, 1881, he came to town again, and a considerable hæmorrhage in one retina was discovered. He took arsenic from October, 1880, till February, 1882, when he had recovered his colour and strength and felt perfectly well. He had also become free from an eczema which had long troubled him. In 1882 he was reported well, and in 1885 came to London again on account of a return of eczema, and was in good health. In the present year (1891) Dr Uhthoff, of Brighton, under whose care he is now for another complaint, writes that he has had no return of anæmic symptoms.

Dr Hale White has since recorded a case of apparently complete recovery from Addison's anæmia in a man aged forty-two, under treatment by arsenic. Three years later he was in good health, and his red corpuscles were fully up to the average in number and colour ('Guy's Hosp. Reports,' vol. xlvii, p. 168).

Treatment.—In idiopathic anæmia the preparations of iron have proved to be altogether ineffectual. Too often no medicine can be found which will check its downward progress. *Arsenic*, however, has sometimes been very successful in cases which appeared to be of this nature. Dr Bramwell first recorded two such instances ('Edin. Med. Journ.,' November, 1877): one is that of a man aged thirty-eight, admitted to hospital for extreme anæmia, with retinal hæmorrhages; he had been ill seven months, and became much worse under the administration of the tincture of iron, which was continued for a fortnight, but he completely recovered in the course of four months, while taking the liquor arsenicalis in doses gradually increased from two to twelve minims. Dr Finny had two successful cases ('Brit. Med. Journ.,' January 3rd, 1880).

In the patient above mentioned recovery took place under full doses of arsenic, and it was so also in Dr White's case.

Phosphorus has sometimes been thought to be useful. Dr Broadbent published a case of apparent recovery under its use in a lad of sixteen ('Practitioner,' Jan., 1875). The writer had under his care several years ago a patient who regained his appetite for a short time under the exhibition of phosphorus in doses of one twentieth of a grain, and became able to get downstairs; but he afterwards relapsed and died. In other cases it has completely failed, and perhaps has sometimes done harm.

It very seldom happens that the *transfusion of blood* can be employed with advantage in cases of anæmia that come under the care of the physician. Human blood only should be employed, and should be defibrinated, by stirring, before it is injected into the patient's circulation, on account of the danger of fatal embolism when it is allowed to retain its power of coagulating. That the red discs retain their structure after having been transferred from one person to another seems to be probable from the fact that no change in the urine is observed after the operation; whereas, when lamb's blood is used, except in very small quantities, an invariable result seems to be the escape, by the kidneys, of hæmoglobin which has been set free by the disintegration of the foreign blood-corpuscles.

A sense of oppression, or of suffocative distress, is sometimes complained of while the operation is in progress; but this may to a great extent be

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obviated if the syringe is used slowly and gently. The largest quantity that should be injected at once is half a pint; in many cases from four to six ounces suffice.

Transfusion in extreme cases of anæmia has proved unsuccessful in the hands of Gusserow, Quincke, Bradbury, and Bramwell. It was practised in a case published by the writer in the 'Guy's Hospital Reports' for 1882–3 (p. 231) with only temporary benefit. But Quincke had five successful cases, including one in which arterial transfusion was practised, beside one of temporary benefit. In all but one of these cases the patients were women. In twenty cases, collected by Dr Coupland in the Gulstonian Lectures already quoted (1881), there were fourteen deaths, the remaining six cases being those just mentioned by Quincke.

It is quite possible that intravenous infusions of milk might be equally useful for the time.

This remarkable operation was repeatedly tried on animals (1665) by Lower and Sir Christopher Wren, and afterwards on human beings (1667) by Denis and Emmerez, at Paris, where the operation had been described nine years previously. The Dutch anatomist, Regner de Graaf, devised apparatus for the purpose. It then fell into disrepute from the many fatal results which followed, but it was revived by Dr Blundell 150 years later ('Med.-Chir. Trans.,' vol. ix; and 'Guy's Hosp. Rep.,' 1837, vol. ii, p. 256), and during the last twenty years has been extensively practised. Its history will be found detailed by Mr C. E. Jennings, together with a description of the form of instrument which appears to be most practically useful, in his book on the subject ('Transfusion of Blood and Saline Fluids,' 3rd edition, 1888). See also the papers by Dr. Aveling ('Trans. Obstet. Soc.,' 1864) and Dr Braxton Hicks ('Guy's Hosp. Rep.,' 1869), and the Report by Professor Schäfer to the Obstetrical Society (vol. xxi, 1880). See also Dr Wm. Hunter's three lectures on 'Transfusion' before the College of Surgeons (August, 1889).

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HÆMORRHAGIC DISEASES

"The mariner, his blood inflamed
With acrid salts, his very heart athirst
To gaze at nature in her green array;
Upon the ship's tall side he stands, possessed
With visions prompted by intense desire;
Fair fields appear below, such as he left
Far distant, such as he would die to find."

COWPER.

SCURVY—*History—Early Symptoms: purpura, spongy gums, hæmorrhagic inflammations—Night-blindness—Course and prognosis—Ætiology—Prophylaxis—Pathology—Diagnosis—Treatment—Infantile scurvy.*

PURPURA—*Symptomatic and primary purpura—Ætiology—Characters and events—Morbid anatomy—Pathology—Diagnosis—Treatment.*

HÆMOPHILIA—*History—Inheritance usually in males through females—Course and symptoms—Prognosis—Diagnosis—Pathology—Treatment.*

THIS group of maladies, like the last, are anæmic as well as hæmorrhagic, but here the anæmia is the consequence, not the cause, of the bleeding.

They resemble primary anæmia in being "diseases of the blood" in the proper sense of the term, *i. e.* they depend, so far as we know, on a primary disorder of the blood, which leads to brittleness and rupture of the capillaries, and perhaps also to want of power to coagulate.

SCURVY.*—*History.*—There is no certain account of this remarkable disease in the classical authors; but Hippocrates writes of a condition marked by foul breath, gums receding from the teeth, bleeding from the nose, and ulcers of the legs, which has been probably referred to scurvy. At the time of the Crusades it made its appearance in unmistakable form as a malady of the camp.

At the siege of Damietta, in 1218, an epidemic of scurvy appeared, and in 1250 the army of Louis IX of France suffered from it before Cairo.

Scurvy became well known towards the end of the fifteenth, and in the sixteenth centuries, among the crews who then first dared long sea voyages to the East round the Cape of Good Hope, and afterwards to America.

Vasco de Gama had 100 men out of his crew of 160 sick with scurvy after he had doubled the Cape. The disease frequently appears in the narratives of the voyages of Dampier, Anson, and Cook.

* *Synonyms.*—Scorbutus.—*Fr.* Le scorbut.—*Germ.* Scharbock or Schormund.—*Dutch.* Scheurbuick. The name is said by Immermann to occur for the first time in the Botanologicon of Euricius Cordus (1534) in the German form "Scharbock," derived from a Danish word, *Skörbeck*, signifying "disease of the mouth." *Scorbutus* is only a Latinised form of the same. According to Professor Skeat, however, *Scheurbuyck* in old Dutch means not "sore mouth," but torn or ruptured belly. He gives the vernacular word "scurvy" an independent origin, from scurf (scab, *schorf*, scrape). Dolæus writes, "Danicè *Scharbuck* indicat oris erosionem et ventris torsionem: inde et Foresto placet *gingibrachium* appellare, cum et brachia interdum afficiat: ab aliis et dicitur *gingipedium*, quod gingivas et pedes infectet" ('*Encycl. Med. Dogm.*,' cap. xii, § 1). Afterwards (§ 3) he describes "*dolor intestinorum totiusque abdominis, lancinans, tensivus et quasi ruptorius, unde fortasse nomen ab Hollandicis accepit, die Scürrbüyk.*"

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It was found that, besides the "sea-scurvy," there was a precisely similar "land-scurvy," which arose from time to time among the inhabitants of besieged towns, in the inmates of prisons and asylums, and among the poorer classes generally, when exposed to privation or famine. Unfortunately the subject was thrown into complete confusion by the publication of a work, 'De Scorbuto,' by Severinus Eugalenus (1658), in which the symptoms and effects of almost every disease were jumbled up together. He seems to have had many followers; and one of the results of their teachings still remains in the popular use of the word "scurvy" as a name for eczematous and other eruptions, and in the traditional practice, in country districts of England, of calling obstinate chronic sores upon the leg "scorbutic ulcers." Indeed, much of the voluminous medical literature of the seventeenth century shows so complete an ignorance of the real characters of the disease that Hirsch doubts whether it can ever have really been common on land.

During the seventeenth century the term *scorbutic* seems to have been used as much as "rheumatic" is at the present time in Germany, "arthritic" in France, and "gouty" in England, to impose an arbitrary pathology on obscure symptoms. Against this use Sydenham, the first physician of modern times who depended on observation rather than authority and on facts rather than words, protested. He asserted that true scurvy, of which in the fifty-ninth chapter of his 'Processus Integri' he gives an admirably terse and accurate description, is comparatively rare in England.* Three years after Sydenham's death was published and dedicated to William III, in 1691, the 'Encyclopædia Medica Dogmatica' of John Dolæus; and in the twelfth chapter of the third book he gives an account of scurvy which presents the exact opposite of Sydenham's masterly sketch: "Prout Beatus Bontekoe non absque ratione omnes fere morbos Scorbuti nomine comprehendit."

In 1753 Dr James Lind, Physician to His Majesty's Royal Hospital at Haslar, gave a full and exact account of scurvy as seen in sailors, and this has been followed by all later writers.

The fullest historical account of this disease, now happily of little more than antiquarian interest, will be found in the late Dr George Budd's article in Tweedie's 'Library of Practical Medicine.' In the fifth volume of 'Reynolds' System of Medicine,' Dr Buzzard gives a valuable description from his own observation of scurvy as it appeared among the allied troops in the Crimea, when there were 23,000 cases in the French army alone. He reviews many other instances of the outbreak of the disease in Scotland, in Ireland, among prisoners of war, in gaols, in Arctic exploration, and among our soldiers in the Punjaub, in Burmah, and in the Cape Colony; and he shows how neither climate nor crowding, squalor nor starvation, bad water nor salt meat can any or all of them produce scurvy (*v. infra*, p. 655).

Onset and early symptoms.—The more definite symptoms of the disease are often preceded by a general failure of health and strength. The face becomes pale and sallow, with a livid discoloration of the lips and cheeks. So characteristic is the patient's appearance, that its cause may often be known at a glance. When several are attacked at the same time, each is struck with the altered aspect of the others. The skin is dry and scurfy, and its hair-follicles are prominent and rough to the touch, as in the condition known as "goose-skin," and in *pityriasis tabescentium*. The muscles

* "Hic enim obiter (sed et libere tamen) dicam, quod, licet nullus dubitem quin scorbutus in his plagis borealibus revera inveniatur, tamen eum morbum non tam frequentem quam fert vulgi opinio occurrere, persuasum mihi habeo."—'*Observ. Med.*,' lib. vi, cap. v, § 9.

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waste and become soft and flabby. The spirits are depressed and gloomy, the mind apathetic and indifferent, while great lassitude, a sense of fatigue, and shortness of breath are felt after exertion. The patient is very sensitive to cold; and pains, described as "rheumatic," are experienced in different parts of the body, especially in the loins and in the calves of the legs. These pains are worse after exertion, and are relieved by rest and sleep.

Hæmorrhage.—After a week or two an eruption appears on the skin, generally first over the lower limbs, but afterwards on the arms and on the trunk; rarely upon the head or face. It consists of reddish or purple spots, chiefly of small size, and presenting the peculiarity that most of them have a hair or a hair-follicle in the centre. The projecting state of the follicles may cause these spots to be slightly raised above the surface. In severe cases there may also be vesicles or bullæ, containing a sanguineous fluid which presently dries up into crusts, while their bases often ulcerate. Much more common are larger subcutaneous extravasations of blood (*vibices*), the edges of which are ill-defined, fading off with varied tints; these sometimes break down, and form large ulcers, with a spongy floor, exuding a thin, bloody, putrid fluid. Some of the nails may be detached by effused blood, and cast off by ulceration.

Another and characteristic symptom is the formation of ill-defined brawny indurations in the connective tissue, especially of the hams, but also behind the ankles, along the back of the thighs, over the recti abdominis, or in the armpits. The skin over them may be free from discoloration, but they nevertheless consist of extravasated clotted blood, mixed perhaps with gelatinous inflammatory exudation. They are sometimes very rapidly developed, and may be hot and painful.

Blood is often poured out into the substance of the muscles, forming more or less obvious swellings, and rendering their contraction painful and difficult; or it may detach the periosteum from the bones over a more or less extensive area, most frequently along the front of the tibia, where an enlargement results which has sometimes been mistaken for a syphilitic node. A similar change may also affect a rib, or the scapula, or one of the jawbones, and may sometimes lead to superficial necrosis and exfoliation. In yet other instances extravasation takes place along the epiphysial lines of growing bones, and causes their separation; or, if there is a recent fracture which has undergone repair, the callus may soften down, and the broken ends may again become loose. Or effusion of blood may take place into the joints, especially the knees and ankles.

The most remarkable symptom, and one which is scarcely ever absent, is that of the gums. Their edges become bluish red, spongy, and detached from the teeth, with which they should be closely in contact. They are also painful and tender, and they bleed at the slightest touch. They may even become black, and so greatly swollen as to rise above the level of the teeth or even to protrude from between the lips. This change is most marked opposite the incisors, but it may also extend round towards the molars. When there is a gap among the teeth the corresponding part of the gum remains healthy. In infants before dentition has begun, as well as in toothless old people, the gingival affection is said to be altogether wanting. M. Fauvel, at the Salpêtrière, had in 1847 a case in an old woman in whom a single remaining tooth was surrounded by a mass of swollen gum; the tooth was extracted, and the gum soon became level and firmer. When severe, a scorbutic state of the gums renders mastication and ingestion of

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solid food impossible. There is a horrible fœtor of the breath. The teeth become loose and may fall out.* Very commonly a grey false membrane forms upon the surface of the affected parts, and the gums may slough, so as to expose the alveolar processes. It is remarkable that the mucous membrane of the rest of the mouth remains perfectly healthy, or at most is slightly livid and puffy, so that the peculiar spongy state of the gums is certainly in some way connected with the presence of the teeth. MM. Lasègue and Legroux, however, speak of having frequently seen ecchymoses on the palate during the epidemic which accompanied the siege of Paris in 1871. The tongue is swollen and marked by the teeth at its edges.

Hæmorrhage from mucous membranes is of frequent occurrence in scurvy. Epistaxis is the most common, but bleeding may also take place from the stomach and intestines. In many epidemics dysenteric symptoms are present, but this appears to be due to a coincidence of the two diseases. Blood seems not to be often expectorated from the lungs, except when gangrenous pneumonia sets in as a complication, as is sometimes the case. More frequently the pericardium, or one of the pleural cavities, or both together, are attacked with inflammation, attended with abundant blood-stained exudation.

The face does not often present purpuric spots in scurvy, but the skin round one or both of the eyes sometimes becomes puffed out into purple swellings, while the conjunctiva covering the eyeball assumes a brilliant red colour and projects above the level of the cornea. Dr Buzzard, in 'Reynolds' System,' says that in many cases seen by him during the Crimean War this condition (which was not inflammatory, being attended with neither pain nor discharge) constituted the chief symptom of the disease; they were usually severe cases, and often ended fatally. Sometimes hæmorrhage takes place in the globe of the eye, especially into the anterior chamber, and may lead to iritis; or there may be hæmorrhagic choroiditis, or panophthalmitis with sloughing of the cornea.

Other symptoms.—The most curious effect of scurvy is what is known as nyctalopia or "night-blindness."† The patient can see well during the day, and at night he can distinguish objects near a candle and even read. But when he has not the assistance of artificial light, he becomes so blind, even though the moon may be shining, that he has to be led about. The pupils may be dilated in such cases, but there are no ophthalmoscopic changes. Not infrequently a sudden night-blindness has been the earliest indication of the disease. In vol. ii of the 'Ophthalmic Hospital Reports' (1859), papers on this subject will be found by Dr Bryson and others, founded upon observations made during the Crimean War and in Her Majesty's ships in different parts of the world.

The order in which the various symptoms of scurvy develop themselves is very different in different cases. Sometimes the gingival affection is the first to appear; it may even constitute the sole manifestation of the disease; but sometimes it follows the purpura by a considerable interval.

* "Upon examination we found their teeth loose, and that many of them had every other symptom of an inveterate sea-scurvy."—Cook, 'Second Voyage,' book i, chap. iv.

† Amblyopia nocturna. Hemeralopia or Hemeropia would better express the modern meaning of the word, for *νυκταλωπίασις* or *νυκτάλωψ* (an irregular compound of *νύξ* and *ὤψ*) is used by Aristotle and Galen to signify *day-blindness*, "a defect of sight incident to children with black eyes," only able to see by night (*v.* Littré *sub voce*). Nyctalopia is the term, however, used by Heberden in a remarkable case he reports of night-blindness in a man who had suffered from ague and saturnine palsy, and who probably died of chronic Bright's disease ('Commentaries: Oculorum morbi'). "Night-blindness" is, at all events, an unambiguous name.

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There may have been no sign of the patient's being otherwise than well until some part of the skin, which has received a trifling blow, becomes the seat of extensive extravasation of blood, or an ordinary purgative dose may unexpectedly be followed by profuse intestinal hæmorrhage, or an old chronic ulcer of the leg may be found to be spongy and its discharge sanious, *i. e.* thin and stained with blood.

Course and event.—The course of scurvy is slow and protracted. There is no fever, except perhaps when there are inflammatory complications. The appetite is bad, though there is sometimes a longing for vegetables and fruit; and there is often much thirst. The pains in the limbs become so severe as to interfere with sleep. Anæmia and emaciation advance rapidly, and subcutaneous œdema is present in many cases. The urine is sometimes albuminous, even though the kidneys may be subsequently found to be healthy. The pulse becomes extremely small and weak, and the heart's impulse may be imperceptible. The muscular weakness is frequently so great that the patient faints if he attempts to sit up in bed. Indeed, syncope is sometimes the cause of a fatal termination at a comparatively early stage of the disease, which otherwise seldom destroys life, even in bad cases, until after the lapse of several weeks. At the "Dreadnought" hospital ship it used to be a rule for all patients admitted with scurvy to be hoisted up the ship's side in a recumbent position. It is rare for hæmorrhages of mucous membranes to destroy life. Death is more often due to gradual exhaustion and prostration, and then the mind is usually clear to the last. In other cases it is the result of some complication, such as dysentery or acute pneumonia (which may or may not pass into gangrene) or ulcerative endocarditis; or, again, it follows extravasation of blood into the cerebral membranes, or the hæmorrhagic form of pleurisy or pericarditis already mentioned.

But in the great majority of cases—at least when the patient comes under medical observation—recovery takes place. The improvement produced by proper treatment is often immediate and striking; but nevertheless many weeks or even months pass before the health is completely restored. The purpuric spots undergo changes of colour like those which are seen in bruises, and gradually disappear; the smaller ones round the hair-follicles merely turn brown. The brawny indurations slowly subside, but they not unfrequently leave behind them thickenings and fibrous bands which may cause permanent contractions of parts of the limbs, especially at the knee or the ankle, with atrophy of the corresponding muscles. Even the joints themselves may become permanently stiff.

The late Dr Carrington's experience was, that at present these protracted cases are not seen at the Seamen's Hospital at Greenwich, and that during six years no permanent injury had followed scurvy. In the worst cases the rations had been short and the water bad.

Cause and prevention.—Our knowledge of the ætiology of scurvy is now so far perfect that we can predict its occurrence, and are able to prevent and to cure it.

So satisfactory a result of scientific medicine should lead us to follow carefully the path which led to it. It has been that of patient and thorough investigation, without speculation, or idle, vague, and perfunctory explanations. It was first necessary to define what was meant by scorbutus, which in the mouth of Eugalenus and Dolæus meant anything or nothing, as scrofula did until our own day. This was done by Sydenham. The next step was to give up the "philosophical" point of view expressed by the great

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Dr Arbuthnot that "the scurvy is a distemper of the inhabitants of low countries, and amongst those who inhabit marshy, fat, low, moist soils near stagnating water, fresh or salt; invading, chiefly in the winter, such as are sedentary or live upon salted or smoked flesh and fish, or quantities of unfermented farinaceous vegetables, and drink bad water." It was necessary to ascertain not whether the commonplaces of ætiology there assigned *might be*, but which of them, if any, *was* the true cause of the disease: not the exciting, predisposing, or co-operant, but the efficient cause, without which the effect is not. One by one the other supposed causes have been disproved by experience, and by the middle of last century scurvy was proved, though not universally believed, to be caused by something wrong in diet. The inquiry has since been pushed further, and of all the constituents of healthy diet, the one of which the lack causes scurvy is now proved to be a due supply of *fresh vegetable food*. The question whether scurvy ever arises when a proper quantity of such food has been taken may be answered in the negative. Immermann, indeed, cites in 'Ziemssen's Handbuch' a few instances in which he says that the disease prevailed notwithstanding that there was an abundant supply of vegetables. But on looking up the first of them, an epidemic which occurred in the barracks of Rastatt, in the winter of 1851–2, we find that Opitz, who recorded it nine years later in the 'Prager Vierteljahrschrift,' says expressly that the poor lived almost entirely on soup and beef and dumplings, because vegetables were very scarce and dear. And even had they been cheap it would have proved nothing, unless those persons who were actually taken ill could be shown to have partaken freely of such food. So, again, it is not sufficient to assert that rations have been duly served out to a body of men; we want to know that they have eaten them. It does not appear that any case is on record in regard to which these conditions are satisfied.

Potatoes have a high antiscorbutic value; the disease was exceedingly prevalent in Ireland after the failure of the potato crop in 1846, and at Millbank Penitentiary in 1832 an outbreak occurred which was directly traced to the introduction, a few months previously, of a new dietary from which potatoes were omitted. Peas and beans are incapable of preventing scurvy, and the same is true of rice and other cereals, but watercress and all green vegetables are useful. The modern methods of preserving vegetables in a succulent condition leave them with their antiscorbutic properties almost intact; but complete desiccation seems to destroy their usefulness. All fruits, including apples, tend to ward off the disease; cider is said to have no such power, but the acid wines of France are believed to be antiscorbutic.

In the absence of ordinary fruit and vegetables many antiscorbutic plants have been discovered. Thus, Captain Cook, with wise forethought, provided *sauer-kraut* for his seamen, and other antiscorbutic remedies detailed in the account of his second voyage. Scurvy-grass has been discovered and eaten, and in the Crimea the soldiers found by experience that dandelion leaves were an excellent antiscorbutic.

It is, however, undoubtedly true that scurvy does not always show itself in those who fail to get a proper share of vegetable food. We have therefore to ask, in the second place, whether the disease requires accessory causes for its production; or, if not, what circumstances are capable of counteracting its chief cause. Some conditions which affect the frequency of scurvy, and are therefore mentioned by writers on its ætiology, have only

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an indirect effect; thus it is more common in cold than in hot climates, and in winter than in summer, only because a low temperature is unfavourable to the growth of plants. It is possible, however, that the development of the complaint is favoured by want of sunlight, residence in narrow, cold, dark, damp dwellings (such as cellars or the casemates of a fort), over-fatigue, excessive drinking. It is also said to be particularly apt to occur in persons who are convalescent from ague or dysentery, probably because soldiers in aguish districts were not properly fed; for neither dysentery nor ague is followed by scurvy in India.

The absence of fresh vegetables from the dietary can be neutralised in several different ways. Thus *fresh meat*, if eaten in large quantities, is an efficient antiscorbutic, especially when raw or but slightly cooked. Indeed, one of the earlier views with regard to the ætiology of scurvy was that it was a direct effect of the salt pork which constitutes so large a part of the diet of sailors; but it has often occurred when no salted provisions of any kind had been taken.

The antiscorbutic value of *milk* was discussed in a valuable essay by the late Dr Parkes in the 'Med.-Chir. Review' for 1848. The conclusion seems to be that the ingestion of milk in quantities of a pint or a pint and a half every day does not always supply the omission of vegetables in preventing scurvy; but children and others who live mainly on milk undoubtedly remain free from the disease. On the other hand, infants suckled by scorbutic mothers have often been attacked; and it is very likely that the milk of cows fed almost entirely upon hay may fail to possess the same properties as that of animals which have had plenty of grass.

The most important of all antiscorbutic agents, however, in the absence of fresh vegetables, are the juices of certain *fruits*, especially the orange and lemon. Their value was recognised as early as the sixteenth century by Albertus Salomon.* In 'The Surgeon's Mate,' by John Woodall, published in 1617, we read (p. 184), "The Lemmons, Limes, Tamarinds, Oranges, and other choyce of good helpes in the Indies which you shall find there, do far exceed any that can be carried thither from England. The use of the juice of Lemons is a precious Medicine and well tried."

But it is only since 1795 that *lime-juice* has been regularly furnished to ships in the Royal Navy, with the result that scurvy, which used to commit the most fearful ravages among our sailors, is now scarcely ever seen. In 1780 the number of cases of scurvy received into Haslar Hospital was 1457, in 1806 one, and in 1807 one.†

Were it not for the systematic use of this agent, every long voyage, when vegetables are no longer to be had, would probably be an experiment demonstrating the real cause of the disease. The usual plan is to serve out an ounce of the juice daily to each man.

In the spring of 1876 an outbreak of scurvy took place among the men of the sledging parties sent out from the ships "Discovery" and "Alert" engaged in the Arctic Expedition. These men had no supply of lime-juice with them, and received only very small quantities of potatoes.‡ Nevertheless it was afterwards argued that the defects in their dietary were not the cause of their falling ill, chiefly on the ground that cases arose from ten

* 'Alberti Salomonis Scorbuti historia: Vitæbergæ, 1574,' vol. ii, p. 784.

† Sir Thomas Watson assigns the merit of the Admiralty order in 1795 to Dr Blair and Sir Gilbert Blain.

‡ They had been given extra rations of lime-juice for some time before starting, "in order to saturate their systems."

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to twenty-seven days after the commencement of the sledging operations, and must consequently have had their origin in the unfavourable conditions under which the men had laboured during the previous long winter. But the Admiralty Committee appointed to inquire into the matter reported that the disease was really due to the absence of lime-juice. In future, it would, under similar circumstances, be well to concentrate the juice so as to render it more portable; and glycerine might be added to prevent freezing.

No case of scurvy from the Royal Navy has been admitted into the Seamen's Hospital since 1879. Most of the patients now treated there are Norwegian sailors.

Theory of scurvy.—Why the absence of vegetables from the food should cause scurvy, and how lime-juice is able to take their place and to prevent the development of the disease, are questions upon which there has been much speculation, but hitherto with no positive result. In the last century dilute sulphuric acid and vinegar were largely made use of, in the vain hope that they too might prove to be antiscorbutics; and crystallised tartaric and citric acids have since been tried, but have generally failed.

Dr Garrod, in 1848, propounded the theory that the essential cause of the disease was the absence of a due supply of potass in the food. He showed that there was a great deficiency of the salts of this alkali in dietaries which were known to be liable to give rise to scurvy, and that it was present in abundance in all the substances which possessed antiscorbutic properties. It is curious that whereas his views have never been widely accepted in this country, they are with a slight modification upheld in Germany by the most recent writers at the present time. This modification consists in regarding as valueless those potass salts that pass out of the body unchanged, and in attaching importance to those alone which undergo conversion into carbonates and may be supposed to enter into the composition of red blood-discs or of muscle. It has in fact been demonstrated that nitrate of potass is incapable of preventing scurvy; but it has not yet been shown that the pure citrate or the tartrate of potass possess antiscorbutic value at all comparable with that of lime-juice or fresh vegetables.

Histology.—There can be little doubt that the immediate cause of the purpura and of the other hæmorrhagic symptoms of scurvy is a morbid condition of the walls of the smaller vessels. But hitherto no visible change in them has been detected with the microscope. In 1871 Lasègue and Legroux examined the capillaries in seven fatal cases which had occurred during the epidemic in Paris at the time of the siege; they could discover nothing but some scattered fat-granules. Nor have we as yet any certain knowledge as to the alteration in the composition of the blood which must be supposed to form an intervening link between the dietetic cause of the disease and its varied phenomena. After death the blood within the body has sometimes been found to be coagulated; sometimes it has been liquid. It has not seldom been pale and watery; but this is only equivalent to saying that anæmia is a symptom of scurvy. For the same reason it is doubtful whether much importance can be attached to observations proving that the red discs are deficient in number. Such oligocythæmia necessarily involves a deficiency of potass salts and of iron, which has in fact been shown to occur by several chemists. Laboulbène (1861) made out a slight degree of leucocytosis; but this again may probably have been merely relative, depending upon the scarcity of red discs.

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Dr Garrod, Dr Ralfe, and others have carefully investigated the state of the urine, in the hope of indirectly throwing light upon the constitution of the blood in scurvy. Dr Ralfe's conclusions are that the uric acid is increased but that the acidity of the urine is diminished, and that there is a great reduction in the amount of alkaline phosphates. He has propounded the theory that the primary change in the blood is a diminution in its alkalinity, citing in support of this view certain experiments upon animals by Hoffmann and others, in which it has been shown that food yielding only an acid ash produces, after a time, effects like those of scurvy, namely, dissolution of the corpuscles, ecchymoses, and external hæmorrhage.

Diagnosis.—The diagnosis of scurvy is chiefly determined by our knowledge of its ætiology. Most observers are of opinion that, if it is certain that a patient has eaten an adequate amount of fresh vegetable food, one is justified, on that ground alone, in denying the scorbutic origin of his symptoms, whatever they may be.

As a rule, the gingival affection is a safe criterion between scurvy and other purpuric affections, including the "morbus maculosus," to be presently described. But in splenic leuchæmia Mosler and others have sometimes found the gums swollen and ulcerated, as well as inclined to bleed.

Whenever there is a doubt as to the diagnosis, it may be quickly settled by the administration of lime-juice; and the same may be said of the exceptional cases of scurvy in which the gums remain in a normal state, as well as of those rare instances in which there are no symptoms except the spongy state of the gums.

There is some danger of overlooking the slight forms of scurvy which occur sporadically among the poor of London and other cities. Dr Buzzard remarks that such persons, who probably never brush the teeth, attach no importance to an unhealthy condition of the gums; nor do they notice a slight petechial eruption upon the legs. What they are likely to seek relief for is a supposed "rheumatic" complaint attended with muscular weakness. It is important, therefore, to be on the look-out for the other symptoms of scurvy, including the peculiar sallow complexion, when advice is asked for vague pains in the limbs. In out-patients' practice we are thus able to detect the disease in men who have been living without vegetable food, and find our diagnosis confirmed by the presence of petechiæ and spongy gums.

Treatment.—In the treatment of persons already ill with scurvy, the administration of lime-juice is almost as important as in its prevention, and the presence of diarrhœa does not contra-indicate its use.

If fresh meat, potatoes, cabbage, and salad can be eaten, so much the better; but when the gums and teeth are very tender, the diet has often to be limited to milk, beef-tea, and eggs beaten up with wine. On the Continent the yeast of beer is given.

Dr Buzzard says that a daily application of solid nitrate of silver to the gums affords great relief when they are sloughing and bleeding. Washes of chlorine-water, permanganate or chlorate of potash, alum or decoction of oak-bark may also be freely employed locally, and chlorate of potash is also useful internally to subdue the stomatitis.* For the hard swellings

* "I have been induced to try the oxygenated muriate of potash (*i. e.* Potassic chlorate), and have found it efficacious in the true scurvy, cases of which sometimes occur among the poor in consequence of improper diet." 'Medical Notes and Reflexions,' by John Ferriar, of Manchester, 1810, vol. iii, p. 250.

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in the legs, friction with soap-suds is said to have been used with success in the Turkish hospitals during the Crimean War. Iodide of potassium is recommended when effusion occurs under the periosteum, and scorbutic ulcers should be dressed with lint soaked in lemon-juice, or with the bruised substance of succulent herbs, such as the house-leek.

Infantile scurvy.—This is an occasional consequence of bad feeding, as is now established by the observations of Dr Cheadle and Dr Thomas Barlow. It often occurs with rickets, and when thus complicated has been described by German writers under the title of "acute rickets," by Mr Thomas Smith as hæmorrhagic periostitis, and by Dr Gee as "osteal or periosteal cachexia." The fact that young children are often given fruit and potatoes, with other improper food, probably saves many of them from scurvy. Besides the other symptoms, effusion of blood among the muscles of the thighs, under the periosteum, or at the junction of the epiphyses with the shafts of the long bones is particularly common (see Dr Barlow's paper 'Med.-Chir. Trans.,' 1883). This condition has often been mistaken for infantile syphilis. The gums are seldom spongy, perhaps from the absence or fewness of teeth, for the child is usually under eighteen months old.

Dr Eustace Smith recommends the administration of orange-juice and the liquor of raw meat together with free ventilation of the nursery and taking the child out in the open air whenever the weather is suitable. Cod-liver oil is also useful.

PURPURA.*—This affection, "the purples," consists in the formation of spots of hæmorrhage in and beneath the skin, and occurs in many different diseases, viz.:—1. In scorbutus. 2. In hæmophilia. 3. With rapidly diffused sarcoma (vol. i, p. 85). 4. In Hodgkin's disease, splenic leuchæmia, and grave anæmia. 5. With cirrhosis of the kidneys, acute rheumatism, and ulcerative endocarditis. 6. As a complication of "erythema multiforme." 7. In measles and variola (vol. i, pp. 181, 209).

In all these conditions the cutaneous hæmorrhages are secondary, nor are they by any means universally present. But there is another form of purpura of which they are the fundamental and essential symptom, and for which at present no cause can be discovered. We are, therefore, justified in making the primary or idiopathic symptoms into a disease.

In Germany this malady is commonly spoken of as the "Morbus maculosus Werlhofii." It was described in the last century by Werlhof, a physician who held a court appointment to George III in Hanover, and died in 1767. In his 'Opera Medica,' collected by Wichmann, a well-marked case is recorded; the patient was a girl previously healthy, who also had epistaxis, hæmatemesis, attacks of syncope, and a small and very rapid pulse; she ultimately recovered.

Of course it cannot be pretended that Werlhof would have exactly limited his morbus maculosus to what we now regard as primary or essential purpura. Nor have we even now any certain warrant for assuming that this may not hereafter be still further broken up into separate affections, due to different causes. There is, indeed, already a subdivision, accepted by most writers into "P. simplex," confined to the skin, and "P. hæmorrhagica," attended with bleeding from various mucous membranes and with ecchymosis of the deeper structures of the body. But this distinction is artificial and unneces-

* *Synonyms.*—Purpura simplex et hæmorrhagica—Idiopathic purpura—Morbus maculosus Werlhofii.—*Germ.* Blutfleckenkrankheit.

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sary, for the less severe "simple" cases of purpura are very apt after a few days to pass into the "hæmorrhagic" form.

Ætiology.—The morbus maculosus seems to be more common about the age of puberty than at any other period of life; females are said to be more subject to it than males, and this applies particularly to children.

The patient is often well nourished and fresh coloured, having been well fed and apparently in good health up to the time when the cutaneous spots are observed, or when hæmorrhage begins from some mucous surface. Sometimes he is anæmic and weakly, or he may have recently recovered from an acute disease, such as enteric fever. Immermann (in 'Ziemssen's Handbuch') remarks that in these cases the purpura usually shows itself when the appetite is returning and when the first attempts to stand are being made. Now and then, when there has been no antecedent malady, there is a short prodromal stage of malaise, anorexia, and headache, lasting two or three days or as long as a week.

In some cases the occurrence of purpura is directly attributable to the medicinal administration of iodide of potassium, although the corresponding sodium-salt is generally incapable of producing a similar effect. This toxic or "iodic purpura" appears to be in all respects identical with the ordinary form of the disease. Descriptions of it, with copious references, are given in papers by Dr Stephen Mackenzie (in the 'Medical Times and Gazette' for 1879) and by Dr Duffy (in the 'Dublin Med. Journ.' for 1880). In one case—that of a syphilitic infant, five months old—it is said to have directly followed a single dose of two and a half grains of iodide of potassium; but it is generally not seen until the salt has been taken for some days or for several weeks. The spots may sometimes subside, notwithstanding that the patient goes on with his medicine; but in most cases, should he have discontinued it, a fresh crop appears as soon as he attempts to resume it.

Symptoms.—Not infrequently the legs are alone affected by purpura, or the spots may appear there earlier than anywhere else, the forearms being the next parts to be attacked, and the face suffering last, or not at all. But sometimes ecchymoses come out simultaneously over nearly the whole of the cutaneous surface in untold numbers. They present differences of colour, which chiefly depend (as Dr Hyde Salter pointed out in the 'Medical Times and Gazette' for 1856) on the depth in the skin at which the blood is extravasated. Thus the more superficial ecchymoses which are seen through only a thin layer of tissue, appear bright red and sharply defined; the deeper ones are of a purple hue and fade off gradually at their edges; they are also generally larger, because the meshes of the tissue in which they lie are more open. Occasionally the cuticle is raised into a dark bleb by blood poured out upon the surface of the rete mucosum. The spots bear no definite relation to the hair-sacs, such as has been described in scorbutus. Nor is there so marked a tendency to the formation of large subcutaneous vibices; but the eyelids are sometimes surrounded by broad black rings, and extensive effusions of blood may sometimes be seen in other regions, especially when there have been blows or other injuries. Occasionally retinal hæmorrhage occurs; Dr Goodhart describes a case in a child four years old.

Among the mucous membranes, that of the nose is perhaps more apt than any other to bleed in cases of morbus maculosus; and epistaxis is often

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the earliest symptom. Hæmorrhage also frequently occurs from the stomach, the intestines, the urinary passages, or the female genital organs; and occasionally from the bronchial tubes. Blood may ooze from the gums, and collect round the bases of the teeth in dark red or black crusts; but when these crusts are removed, the gingival tissues are never found to be swollen, spongy, nor of a purple-red colour, as in scorbutus; they are either perfectly normal in appearance, or more or less anæmic.

In the more severe cases of purpura, when there has been a considerable loss of blood, the patient may rapidly pass into a condition of extreme anæmia, with waxy pallor of the skin, a rapid feeble pulse, and liability to faint on the slightest exertion, even on attempting to sit up. Under such circumstances fever may be present, as in all other forms of extreme anæmia. Immermann believes that the temperature may sometimes rise as the result of reabsorption of the extravasated blood, or in consequence of local inflammatory changes set up by its irritant action upon the tissues among which it lies. But it seems still doubtful whether fever is ever present as an initial symptom, or at least whether the occurrence of a high temperature at the beginning of an attack of purpura does not show that the case is of a peculiar kind.

With regard to the condition of the blood within the vessels in purpura there have been different statements. It has been said to be deficient in coagulating power, but this seems to be a mistake. There is always diminution of red corpuscles. Immermann estimated the proportion of leucocytes in a severe case at Basle; during the first few days it was normal, but afterwards there was a slight excess, as is usual after all kinds of hæmorrhage. Laache, however, found leucocytosis from the first.

Event.—As a rule, purpura ends in recovery. In some cases fresh spots may cease to come out after a few days, the old ones fade and disappear, the mucous membranes cease to bleed, the anæmia is quickly repaired, and within three or four weeks the patient is as well as ever. If he should get up too early it often happens that a new crop of spots may be seen upon his legs within an hour or two of the time when his feet are first allowed to touch the ground; but these soon undergo absorption in their turn. Sometimes, however, the disease runs on for several weeks, or it may recur again and again, with intervals of many months, during which the health appears to be perfectly good.

Beside the ordinary cases of idiopathic purpura which are confined to the legs, and readily cured by laxatives and arsenic or steel, we occasionally see more severe cases in previously healthy young patients. Two of these have been lately under the writer's care in Guy's Hospital. One was a healthy lad aged fourteen, whose case is published in the 'Pathological Transactions' for 1884. The disease came on without known cause, and ended fatally in about ten days. While lying almost unconscious the day before his death, a house-fly settled on his face, and before it could be brushed off had left a bleeding mark. Here micrococci were found in venous thrombi after death. The other case occurred in a robust young man aged twenty-two. The onset was unexpected, and the symptoms well marked. Severe epistaxis was effectually stopped by the inflated bag (vol. i, p. 1134), and active treatment checked the hæmorrhage from the bowels, stomach, and kidneys. But all the ill symptoms returned, and he died on the fourteenth day. Here micro-organisms were looked for by the writer and by Mr

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Cheyne, who had seen the former case, but none were found; nor were they in Dr Wickham Legg's two cases ('St Barth. Hosp. Rep.,' 1884; and 'Path. Trans.,' 1885).

Anatomy.—After death the lining of the stomach, intestine, uterus, kidneys, and bladder are generally found spotted with ecchymoses, as are also the pleura, the pericardium, the arachnoid, the peritoneum, and even the substance of the lungs and the medulla of the bones. In some cases an effusion of blood upon the surface of the brain or into the ventricles is the immediate cause of death. Thus a man aged thirty-four was admitted into Guy's Hospital for purpura, and appeared to be doing well, when he became insensible and paralysed on the right side. Afterwards there was loss of power in the left limbs also, and he died on about the twentieth day of his illness. A quantity of blood was found extravasated on the left hemisphere of the brain beneath the pia mater, and also within the ventricular cavities. Another case, which occured in 1871, was that of a woman aged twenty, who, while in the hospital for phthisis, was attacked with a severe form of purpura, and with epistaxis. At the end of about a week she became delirious for some hours on two successive days. Then the bleeding ceased, and the spots disappeared, but she sank gradually a fortnight later with diarrhœa. The dura mater over each hemisphere was found lined with a uniform layer of blood, which was yellowish in tint, and almost membranous.

Occasionally some of the more delicate structures of the body seem to slough as the result of the infiltration of blood into their tissues. Thus a girl nine years old was admitted into Guy's Hospital for gangrene of the external genitalia and purpura. So far as could be learned she had not been affected with any one of the contagious exanthemata. There was a fœtid discharge, and she died in four or five days. The bladder, the vagina, the uterus, and the Fallopian tubes were all found intensely inflamed and covered with spots of hæmorrhage. Some years later a man aged twenty-three died after an illness of eight days' duration, which began with a purpuric affection of the right thigh. The lower end of the ileum, for about one foot of its length, was of a purple colour, its coats thickened and infiltrated with exudation, its serous surface coated with lymph, and its mucous membrane slightly excoriated. Zimmermann has related in the 'Arch. f. Heilkunde' for 1874 a case in which several intestinal ulcers formed and sloughed through into the peritoneum, setting up a fatal peritonitis. (See also Dr Legg's second case above referred to.)

In a few instances pigment has been found staining the deeper organs after death. A striking example of this interesting result was recorded by Dr Hindenlang, from Prof. Bäumler's wards. An apparently healthy postman, who had gone safely through the campaign of 1870, was suddenly attacked with subcutaneous hæmorrhage in several parts of the body, and died two months afterwards with all the symptoms of purpura. The lymph-glands and the liver showed deep brown pigmentation, and also the pancreas and kidneys. The amount of iron in these organs was much increased, and there can be no doubt that the pigment was directly derived from extravasated blood, though no crystals of hæmatoidin were discovered ('Virchow's Archiv,' Bd. lxxix, 1880). Dr Hindenlang refers to three somewhat similar cases recorded by Orth, Tillmanns, and Wm. Müller. The pathological relation of this pigmentation to the deposit of iron in the liver observed in severe idiopathic anæmia (p. 646) is important, and shows the intimate connection between hæmatolysis and purpura.

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Pathology.—There are good analogies in support of the view that the morbus maculosus depends primarily npon a peculiar alteration in the blood; but it seems clear that, before the hæmorrhages occur this must have led to a morbid state of the walls of the capillaries, perhaps by impairing their nutrition. Immermann suggests that the purpura which occurs during convalescence from fever is possibly due to the circumstance that the recovery of the minute vessels is sometimes retarded beyond the time at which the volume of the blood is restored and the heart regains its vigour. In a case recorded by Dr Wilson Fox, in the 'Med.-Chir. Rev.' for 1865, the arterioles and capillaries of the skin in the neighbourhood of purpuric patches were found to be obviously altered in appearance; they were brittle, had a glistening waxy look, and assumed a most intense reddish-brown colour with iodine. The patient, a man aged thirty-three, had been affected for about a month with a syphilitic eruption, which followed an indurated chancre at five months' interval. He had taken iodide of potassium for some time, but not continuously. It seems doubtful whether any of the viscera were lardaceous, except perhaps the adrenal bodies and the intestinal mucous membrane. Many of the muscles were in parts pale and waxy looking; their fibres had lost their striation, and became deeply stained by iodine, while their blood-vessels showed changes similar to those observed in the affected parts of the skin. Dr Thin ('Med.-Chir. Trans.,' lxii) has described the minute blood-vessels as obviously altered and disorganised within the area of a bulla caused by the administration of iodide of potassium; and he supposes that iodic purpura is due to a more extreme change.

In a case of the writer's, in 1883, referred to above, Mr Watson Cheyne found strings of micrococci in the tissues. In a case of Dr Russell's, of Carlisle, he had previously found bacteria ('Path. Trans.,' vol. xxxv, p. 408). In other instances no microphytes have been found, and their presence is probably accidental.

Diagnosis.—The diagnosis of the morbus maculosus rests upon the exclusion of the various diseases above mentioned which may give rise to symptomatic purpura. The possibility of the sporadic occurrence of scurvy must not be forgotten, but even mild scorbutic cases are generally distinguished by positive characters—the debility and anæmia that precede the cutaneous affection, the swollen and spongy state of the gums, the brawny induration in the hams, and the formation of each of the spots round the mouth of a hair-sac. In cases of purpura, hæmorrhages from the mucous surfaces are generally much more profuse than they are in scorbutus. Malignant sarcomatous growths must always be carefully sought for, the heart and the urine must be examined, and the state of the spleen and lymphatic glands must be investigated. In fact, the diagnosis of idiopathic purpura, like that of idiopathic anæmia, can only be certainly established after a *post-mortem* examination.

Treatment.—There is reason to believe that certain medicines are capable of preventing the formation of fresh spots of purpura and of averting the hæmorrhages from mucous membranes which constitute the most serious part of the disease. Of these *arsenic* appears to be the most valuable. Dr Habershon recommended it in the 'Guy's Hospital Reports' for 1857; it has since been commonly employed in our wards, and in one case it succeeded at once, when many other drugs had failed. Turpentine is strongly recommended by Dr Gee, and seems to be the most efficient hæmostatic in some

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instances; while ergot, acetate of lead, or gallic acid succeed better in others. Immermann says that it is important not to treat the consecutive anæmia by ferruginous preparations for some time, and that their administration has sometimes led to a relapse. Chlorate of potash is often of service, but is not so certain a remedy as in cases of local hæmorrhagic stomatitis (p. 133). The patient should be kept in bed in a cool room; he should have a light milk diet, and his bowels should be kept open by laxatives. Stimulants are often needed. Dr Eustace Smith recommends in previously healthy children oil of turpentine in castor-oil, which acts as a drastic purgative.

HÆMOPHILIA.*—From the commencement of the present century it has been known that in certain families the males during successive generations are liable to protracted and sometimes fatal hæmorrhage after injuries of no great severity: such persons are called "bleeders." Attention was drawn to the subject in 1803 by an American physician, Dr J. C. Otto, who gave an account in the 'Medical Repository of New York' (1803), of a family in which this morbid tendency had existed for seventy or eighty years. In 1784 Sir William Fordyce recorded the case of a Northamptonshire family affected in a similar way. A monograph on the disease was published by Dr Wickham Legg in 1872.

Ætiology.—The isolated occurrence of hæmophilia is exceedingly rare. When a "bleeder" is born of healthy parents, some or all of their subsequent children are generally also affected; and probably the disease had already existed in a grandfather or some other ancestor.

The preponderance of males among those who suffer from hæmophilia is nearly as thirteen to one. Moreover, when it does affect women, hæmophilia is much less severe, and scarcely ever fatal: its signs are often limited to the occurrence of cutaneous ecchymoses, spontaneously or after slight injuries—or, in other cases, to epistaxis, menorrhagia, or excessive post-partum hæmorrhage. Immermann, however, quotes an exceptional instance, recorded by Reinert, of a family of sons and daughters, in which the daughters alone were bleeders, while the sons were all free.

It is remarkable that the inheritance of the complaint takes place mainly through the female line. The sons in a hæmorrhagic family do not all invariably suffer; and if any escape, their children are almost always exempt. Even those sons who are affected, if they live to beget offspring, may have some boys who are healthy. But the daughters of such a family, though they have not themselves shown the slightest indication of hæmophilia, are almost certain to transmit it to their male children. The same curious law of heredity is seen in the case of gout.

Obviously these facts have an important bearing on the propriety of marriage on the part of members of a hæmorrhagic race. At Tenna, in the Grisons, there were once two families, not known to be related to one another, in which the disease had been known to exist for a century. In 1855 the females of these families determined to renounce marriage, and in 1879 Immermann was able to state, on the authority of Dr Hörsli, of Tusis, that there was no longer a well-marked example of hæmophilia in the village.

A curious circumstance is that persons with an inherited tendency to the

* *Synonyms.*—Hæmorrhaphilia (Schönlein), a barbarous word, intended to mean love of bleeding (αἱμορραγία, φιλία)—Hæmorrhagic diathesis.—*Fr.* Hémophilie.—*Germ.* Bluterkrankheit.

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disease seem to be unusually prolific, the average number of children to each union being as high as nine.

Hæmophilia occurs indifferently in those who are well nourished or thin, and along with dark, ruddy, or light complexion. Its victims are, however, said to have generally a thin, delicate, and transparent skin, with full subcutaneous veins.

The disease has repeatedly been observed in Jews, and a large proportion of recorded cases have occurred in nations of Teutonic as compared with those of Latin origin; but the reason probably is that it is only in certain countries that much attention has yet been given to the subject.

Course and symptoms.—Hæmophilia does not usually manifest itself at the time of birth. The detachment of the navel-string has but rarely been attended with bleeding in children who were afterwards to suffer from it. But about the end of the first year, or at least before the close of the second, definite symptoms generally appear. The latest recorded age at which hæmophilia commenced and assumed a serious form appears to be the twenty-second year.

The hæmorrhages which characterise the disease are commonly divided into those which are traumatic and those which are spontaneous, but the distinction is only partially applicable, since slight injuries are very apt to be forgotten, and yet in "bleeders" may give rise to considerable extravasations. Thus spots and patches of effused blood in and beneath the skin may sometimes be traced to the pressure of the clothes. But there is no doubt that they sometimes arise independently of any such cause, especially where they are so small as to be termed petechiæ. Indeed, successive crops of cutaneous purpura may be observed in this disease, exactly as in the "morbus maculosus" and in many other morbid states. The spontaneity of hæmorrhage from mucous surfaces is said to be sometimes shown by its being preceded by well-marked signs of "fluxion"—throbbing of the heart and of the arteries, redness and heat of the cheeks, ears, and lips, headache, giddiness, restlessness, and irritability of sight and hearing. Epistaxis is the most common form of mucous hæmorrhage, especially in children; according to Grandidier it is four times as frequent as any other. Next in order of occurrence comes bleeding from the gums and mouth; this, however, may be in part traumatic, for Dr Legg mentions that some patients cannot use an ordinary tooth-brush without drawing blood. Again, there may be hæmorrhage from the stomach, the intestines, the lungs, the urinary passages, the female genitalia, or even the lachrymal caruncle. Blood does not often escape from the unbroken skin, but cases are on record in which it has oozed from the finger-tips or the ears.

In marked contrast with these spontaneous or quasi-spontaneous forms of hæmorrhage are those which result directly from blows or cuts. Even slight superficial scratches, such as would scarcely be noticed in a healthy subject, may bleed so as to endanger life. There are, however, considerable differences as to the amount of injury that can be borne, not only in different hæmophilic patients, but in the same patient at different periods. One cut may cause but slight loss of blood, whereas there may be the greatest difficulty in checking the oozing from another precisely similar injury on a later occasion. The operation of ritual circumcision has several times proved fatal. So have venesection, the application of leeches or cupping-glasses, and, above all, the extraction of a tooth. Indeed, although Dr

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Legg says he has seen a tooth drawn without there being any remarkable amount of hæmorrhage, both he and all other writers are agreed that extraction is a hazardous operation in those who are "bleeders." The slight punctures made for vaccination have scarcely ever given rise to much bleeding.

It must be remembered that the risk of hæmorrhage from a wound continues until it is completely healed; for a thin cicatrix has been known to give way after having formed.

The deeper structures may also be the seat of extravasations of blood, which reach an enormous size, and are almost peculiar to hæmophilia. A subcutaneous tumour may be formed as large as an apple, or even as a child's head. It sometimes seems to rise spontaneously, but very often it is due to some slight injury. Thus, in a case of Sir William Jenner's, cited by Dr Legg, the fall of an india-rubber air-ball upon the thigh filled the connective tissue with blood from the knee to the trochanters. The thigh is, indeed, one of the favourite seats of such swellings, and Immermann says that they are also frequently seen under the false ribs and upon the back. They are generally of a black or dark-blue colour, surrounded by a zone of red. They are sometimes very hard, sometimes soft or fluctuating. They may be hot, and painful and tender to the touch; and in some cases they suppurate, discharging a mass of altered blood with shreds of broken-down tissue, after which hæmorrhage is apt to go on for a considerable time. If they are punctured by the surgeon, dangerous bleeding commonly follows; but if left alone they slowly subside, and at last disappear.

In hæmophilia the effusion of blood into any one of the large serous cavities seems to be rare. Immermann cites two cases in which the peritoneum was the seat of hæmorrhage, and four in which it occurred in the cerebral membranes; but in at least three of these latter there had been a fall or a blow upon the head. He says that extravasation into the pleural sac or into the pericardium have not yet been observed. It may therefore be well to note that in a case of hæmophilia Dr Goodhart found the right pleura closed by adhesions which were in part stained of a deep orange colour.

Articular affection.—One of the most remarkable features of hæmophilia has still to be mentioned, namely, the occurrence of swelling in one or more of the joints, especially the knees. This affection usually begins between the seventh and the fourteenth year. It is sometimes the direct result of a blow, and sometimes it immediately follows a long walk; but Dr Legg says that the most common cause is exposure to cold or the occurrence of damp and chilly weather, and that it is most frequently seen at the beginning of spring or at the end of autumn. The enlargement often occurs rapidly, and appears precisely like that due to rheumatism, or to synovitis from injury. Many writers have therefore supposed that it often follows serous effusion into the articular cavity. But all the pathological evidence which at present exists seems to point to the conclusion that the primary lesion is extravasation of blood. It is true that when a joint has been affected for a length of time the cartilages show signs of chronic inflammation, and that the projecting folds of synovial membrane in its interior are thickened and swollen. This was markedly the case in the knee of a boy who was under Mr Bryant's care at Guy's Hospital in 1880, and in whom the joint had been more or less swollen for three years. But in the same patient there were other joints in which after death all the structures were found healthy, except

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that they were stained by orange-coloured pigment, and covered with stringy masses of ochre-brown fibrin. It might perhaps be supposed that if a great quantity of blood were poured out into a knee-joint a discolouration must be visible through the skin, but this is negatived by observation.

The usual course of the articular affections of hæmophilia is slowly to subside under treatment, but to return again and again at intervals of months or years.

Prognosis.—In some exceptional cases of hæmophilia the hæmorrhagic tendency is said to cease during childhood or youth, and never to return. But by far the larger number of those who are affected die before they are eight years old. Grandidier (quoted by Eustace Smith) found that, of 150 boys who were bleeders, more than half died under seven years of age, and only nineteen reached twenty-one. When adult life has been reached, the danger is less, but it is by no means at an end, for fatal bleeding has been known to occur as late as fifty or sixty years of age.

The habitual condition of bleeders, even when they have long suffered from the disease and are perhaps still troubled with the articular complication, is not generally one of permanent anæmia; they often have as much colour in the face and lips as other people. But they may of course be blanched by a profuse loss of blood. The quantity poured out is sometimes enormous. One case is related in which, after the extraction of a tooth, half a gallon was lost in less than twenty-four hours. It often happens that oozing goes on at the rate of three or four pints in the twenty-four hours for several days together. When the source of the hæmorrhage is visible, it seems to come, not from a single vessel, but from the whole surface, as from a saturated sponge. Sir William Jenner has remarked that it has generally appeared to him to be venous rather than arterial. As the bleeding goes on, the patient becomes pallid, pulseless, delirious and unconscious, and death is often preceded by convulsions. But sometimes, when his vital powers are reduced to the lowest ebb, the oozing, which may have resisted all treatment, ceases spontaneously; he remains apparently on the brink of dissolution for several days, and then slowly revives. Writers generally say that the blood undergoes restitution in such cases more rapidly than might be expected, but Dr Legg remarks that the anæmia remains for at least four or six months.

Diagnosis.—The diagnosis of hæmophilia is not difficult in confirmed cases. But one must keep in mind the possibility that an effusion into the joint may be due to this cause, even when it occurs in a person who may not be anæmic, who exhibits no purpuric spots, and who does not mention that he is a bleeder. The real nature of less marked examples of the disease, such as occur in the female sex, could probably never be positively determined without reference to the history of family predisposition Many women have a tendency to bruise very readily, and others are subject to the recurrence of spontaneous hæmorrhages, especially "hæmatidrosis," in which blood oozes from the mouth of the hair-sacs or sweat-glands.

In the 'Medical Times and Gazette' for 1871 Dr Legg recorded two cases, in women, of a "hæmorrhagic diathesis," in which the resemblance to hæmophilia was nearly complete, there being a great liability to hæmorrhage from slight wounds, and also to epistaxis, menorrhagia, and purpura. In each instance the abnormal state had been present for some years, but in neither of them did it exist before puberty. This last circumstance, how-

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ever, could hardly be held to exclude hæmophilia, because many of the slight forms which are seen in women seem not to be recognisable during childhood. But what appeared to be conclusive was that each patient had borne male children who were not bleeders, and that no family history of hæmophilia could be elicited.

Pathology.—With regard to the nature of hæmophilia, very little can as yet be said. Some of the older writers supposed that it depended upon a defective coagulability of the blood. But it is now known that this was a mistake; there is no proof of any abnormal condition of the blood, except as the result of the continuance of hæmorrhage. So, again, the fatty change sometimes found in the substance of the heart is clearly consecutive to the anæmia which commonly precedes death (p. 64). Several observers have noticed a peculiar thinness and transparency of the arteries—not only of the aorta and pulmonary artery, but also of such branches as the temporals and radials. Others, however, have failed to discover anything of the kind; and the most that can be said is, that although not itself the cause of the symptoms of the disease, it may possibly point to the existence of a similar abnormality of the capillaries, and that this may really be the cause.*

Dr Legg suggests that hæmophilia may depend upon a backwardness of growth or imperfect development of the vascular system generally. Immermann also speaks of a "hypoplasia" of the vessels; but he thinks that the essential cause of hæmophilia is a disproportion between the capacity of the circulatory apparatus and the volume of the blood. He imagines that in hæmophilic males there is an actual overgrowth of the blood itself, and he ingeniously endeavours to account for the transmission of the disease by females who themselves are not bleeders by supposing that they have the thin vessels, without the excess of blood. Sir William Jenner's authority may be quoted ('Brit. Med. Journ.,' 1876, ii) for the view that there is in hæmophilia "a tendency to plethora of the smaller vessels." He remarks that it is when the patient is looking his best, that injuries have the worst effect and that spontaneous hæmorrhage is most likely to occur.

Treatment.—These views are not without bearing upon the management of bleeders. Their diet should be light. Jenner recommends "a considerable proportion of white meats." An aperient dose of sulphate of soda may be given every week, and a mercurial purge every third week. Dr Legg has found that after taking iron, patients have been less liable to spontaneous bleeding and have lost less blood after wounds. A warm, dry climate is desirable.

When actual hæmorrhage is going on, the tincture of iron, or ergot, or gallic acid may be used internally. Locally there appears to be no styptic so valuable as the perchloride of iron; a strong solution may be applied to the mucous membrane of the rectum, if that is the seat of hæmorrhage; and when the socket of a tooth bleeds after extraction it may be checked by the introduction of solid crystals of the salt.

* See a paper by Dr Percy Kidd ('Med.-Chir. Trans.,' vol. lxi, p. 243), and Dr Legg's account of four other cases ('Path. Trans.,' xxxvi, p. 490); also Dr Theodore Acland's description of the state of the thymus (ibid., p. 491), and Dr Laache's observation (loc. cit., p. 43).

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GENERAL DISEASES AFFECTING THE JOINTS

GOUT*

Ὦ στυγνὸν οὔνομ', ὦ θεοῖς στυγούμενον,
Ποδάγρα, πολυστένακτε, Κωκυτοῦ τέκνον.

Πόδα, γόνυ, κοτύλην, ἀστραγάλους, ἰσχία, μηρούς
Χεῖρας, ὠμοπλάτας, βραχίονας, κορώνα, καρπούς,
Ἐσθίει, νέμεται, φλέγει, κρατεῖ, πυροῖ, μαλάσσει.

Κωλυσοδρόμα, βασαναστραγάλα
Σφυροκρησιπύρα, μογισαψεδάφα
Γονυκαμψεπίκυρτε Ποδάγρα.

LUCIAN, *Podagrotragœdia.*

History—Onset and symptoms of a first attack—Subsequent course—The affected joints—lithate of soda—tophi—Pathology of gout—Ætiology—hereditary gout—age and sex—diet—climate—saturnine gout—Diagnosis—Prognosis—Gouty nephritis—other complications—Treatment by drugs, diet, and baths.

THIS remarkable disease was well known to the ancients. The Hippocratic writings show that it was common in Greece, and it is frequently alluded to by Cicero, Horace, Juvenal, and Martial. Celsus, Galen, and Aretæus describe the disease very much as we see it now. Alexander, of Trales, in the 6th century, recommends hermodactyl, and this drug was praised by the Byzantine and Arabian physicians; it was probably the corm of a species of colchicum. Lucian wrote a burlesque tragedy which describes the power of the goddess *Podagra*, and the uselessness of remedies.

In modern times, the medical literature of gout dates from Sydenham (1683), who was very competent to write about this disease, having himself suffered from it for thirty-four years. His masterly description was copied by Cullen, and has formed the basis of almost all that has since been written upon the subject.

At the beginning of the last century Sir Richard Blackmore, the court physician, whose poems live in Dryden's satire, wrote a worthless discourse on the Gout (1726), and Dr George Cheyne an essay on the same subject (1722), better written, but with no other merit. Heberden's chapter on the subject is one of his best (1782). Early in the present century Sir Charles Scudamore published a treatise on Gout and Gravel, which went rapidly through many editions (1817 to 1823); and here, for the first time, we find the modern methods of collection of facts, anatomical observations, and

* *Gr.* Ποδάγρα. The terms χειράγρα, ὠμάγρα, and similar compounds were also used.—*Lat.* Podagra: in Mediæval Latin Gutta, whence *Gout*, *Goutte*, and *Gicht*. "Arthritis," ἀρθρῖτις, sc. νόσος, is sometimes used as a synonym by classical and by modern writers.

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statistics. A great advance was made by the publication of Sir Alfred Garrod's well known and original researches (1859), and recently, Sir Dyce Duckworth's Treatise (1889) is an excellent summary of both the clinical and the pathological aspects of the disease.

Abroad, gout has always been comparatively rare, but Trousseau and Charcot in France, and recently Professor Ebstein in Germany, have made valuable contributions, clinical and pathological, to its study.

The fit of gout.—In a large proportion of cases, when gout occurs for the first time in a young subject, its onset is sudden and severe. The patient is asleep in bed, when he is awakened, about two o'clock, or between two and five, by a pain in one of his feet, generally in the ball of the great toe, but sometimes in the heel, ankle, or instep. The parts feel as if tepid water were poured over them. Then follow chills and rigors and a little fever. The pain which was at first moderate becomes more intense. It is characterised as a grinding, crushing, wrenching pain like that of dislocation of a joint; or as a burning, such as would be caused by a hot iron pressed into the joint; or as if a dog were gnawing it. The patient keeps changing the position of his foot, in the vain hope of finding a place in which to lie in comfort. He cannot bear the bedclothes to touch it. The least vibration of the floor causes him extreme distress, so that those about him have to tread the room with the lightest possible step, and the passage of a waggon along the street below sends him almost mad with rage. For, as Sydenham puts it, "a fit of gout is a fit of bad temper."

Towards morning the patient has a sudden and slight respite, which he imputes to having found a comfortable position. He perspires gently and falls asleep. He wakes freer from pain, and then finds that the part is swollen. Till then the only visible swelling had been that of the veins round the painful joints. For the next two or three days the pain becomes worse towards evening, and abates in the morning. Then, however, not infrequently, the other foot begins to swell, and the whole tragedy, as Sydenham calls it, is acted over again.

A joint affected with gout, besides being painful and swollen, is also of a deep red colour, tense, and shining. As soon as the patient can bear the pressure of the finger, one finds that the skin over it pits, from the presence of œdema. Afterwards the cuticle peels off; and then the part begins to itch.

The amount of febrile disturbance is proportionate to that of the local inflammation. This constitutes one of the distinctions between gout and acute rheumatism; for in the latter disease there may be high fever, with scarcely any affection of the joints.

In robust patients, who have not had more than a few previous attacks, the duration of a fit of the gout is about a fortnight. But in persons of advanced age and broken-down health, it may last two months or even longer. In that case, however, the length of the attack is rather apparent than real; for it is in fact made up of a series of irregular minor fits, which gradually become milder.

It is generally said that before a first seizure of gout the patient appears to be in his usual health. Certainly he seldom has any idea of what is about to befall him. Sydenham, however, observed that indigestion and flatulence precede the attack; and Sir Alfred Garrod says that the premonitory symptoms are sometimes very distressing. Trousseau remarks that in addition to symptoms of hepatic disorder the patient often has an irregular and

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capricious appetite, preferring acids and meats strongly spiced, as if he felt the necessity of stimulating his torpid organs of digestion.

After an attack of gout, on the other hand, a man often feels much better than for some time before. He is more active, and free from many uncomfortable feelings that had before troubled him. Sooner, or later, however, these return, and are the prelude to a second seizure. Sometimes this does not occur for two or three years after the first; but often the interval is not more than a twelvemonth, and it may be less. The second attack is in its turn succeeded by others, and always at shorter intervals; until, at last, the disease becomes chronic and the patient is scarcely ever free from it.

Probably in consequence of Sydenham's graphic and accurate description of a first attack of acute gout, it has been too often assumed that such is its usual or constant mode of onset. But in many cases, perhaps in the majority, the first attack is far less acute, severe, and dramatic: the tragedy is more slowly played out. And in not a few patients gout begins in no acute or sudden form, but stealthily and almost imperceptibly. A man about fifty complains of his boots being tight or sprains his ankle while shooting, or feels a twinge in one knee as if it were strained. There is no pain at night and no redness or swelling to be seen, but a moderate walk is more and more apt to be followed by a "sprain," and at last the great toe or instep or ankle is found to be decidedly swollen, tender, and slightly reddened, so that the suspicion, which the patient had long silenced, becomes certainty,—he has the gout.

Locality.—It has already been stated that the ball of the great toe is the joint most frequently first attacked by gout. Sir Charles Scudamore found that this joint was affected on one side or the other in 373 out of 512 first seizures. When any other part shows the earliest manifestation of the disease, it is often found that this particular joint has in some way been injured. Thus, according to Garrod, the knee may be attacked first before any other joint, if the patient has previously hurt it by a fall; and a considerable time may pass after the accident before the result follows. Next to the great toe, the joint most obnoxious to gout is probably the metacarpo-phalangeal articulation of the index finger; certainly not that of the thumb. Both the great toe and the root of the index are more exposed to pressure and strain than the other joints of the foot or hand.

Whatever may be the part first affected, other joints afterwards suffer, and even in the first seizure two or three different articulations may be attacked. These are generally some of the small joints, such as those of the toes or fingers, the ankles or wrists. Even in the most advanced and intractable cases, the largest joints of all—the shoulders and hip-joints—are usually spared.

Anatomy.—When a patient has suffered from repeated attacks of gout the affected joints become greatly deformed. This is more marked in the fingers than in the toes, no doubt because their natural movements are more free. The fingers become bent irregularly in all directions; sometimes inwards, sometimes outwards. A very common state is for the metacarpo-phalangeal and the second phalangeal joints of a finger to be stiffly flexed, while between them the first phalangeal joint is over-extended, and its knuckle represented by a deep hollow.

Sometimes all the phalangeal joints are uniformly enlarged. Sydenham

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compares their appearance to that of a bunch of parsnips. But in many cases they are very little altered in form.

Lithate of soda.—In long-standing cases of gout there are found in the tissues round the joints masses of a white material having the consistence of putty or mortar, or hard and dry like chalk. These masses are called *tophi** or "chalk-stones." They do not, however, consist of carbonate of lime, but mainly of urate of soda. This salt is also found deposited in the articular cartilages of the affected joints. It there looks as if it were upon the surface of the cartilages, covering them more or less completely, and appearing just as if it had been laid on by a brush. However, on making a section of the cartilage, one finds that the deposit is really in its substance. Examined by the microscope, it proves to consist entirely of crystals. There are bundles of very fine needles, which (according to Cornil and Ranvier) commonly occupy the centre of the cartilage-cells. It is in the superficial part of the cartilage that the crystals are most densely crowded; they often make it opaque to transmitted light. Towards the articular end of the bone they are more thinly scattered; here they often traverse the whole thickness of a cartilage-cell, which is, as it were, impaled on them.

The synovial membrane may contain similar deposits of the urate; but they are much more apt to occur in the fibrous structures outside the joints, particularly the surrounding ligaments and tendons. There are some discrepancies in the statements of writers as to the deposition of lithate of soda in the *bones*. Dr Garrod says that he has never been able to find it there But Cornil and Ranvier teach that it is not uncommon in the cancellous tissue. They mention one case in which they observed it, and in which the bones forming the metatarso-phalangeal joint of the great toe had entirely lost their cartilages and were ankylosed together, and say that absorption of the articular cartilages is a common remote effect of urate of soda in a joint. This, however, is not in accordance with our experience in England. Bony ankylosis is unknown, and it is very rare to find the cartilage gone in a gouty joint.

The same salt is also deposited in many cases, even at a distance from the joints. The writer remembers seeing a man who had a number of small ulcers, some of them in the middle of his thighs and legs, from which masses of urate of soda were discharged. In 1886 and 1887 a patient was in Philip Ward, who had tophi of lithate of soda in the skin of his legs and arms, as well as in the joints and ears. The integuments of the limbs, however, very rarely present such deposits, except over articulations. But there is one particular region where they are very often to be found. This is in the external ear, generally in the helix. Garrod remarks, with his wonted accuracy, that these deposits in the pinna of the ear are at first fluid, the skin over them forming a vesicle of a milk-white appearance. He says that some months elapse before they become the white, hard, bead-like masses, which are commonly seen. Is any fluid present when the deposition of the urate of soda first occurs in the articular cartilage?

The bursæ are very liable to receive deposits of urate of soda in gout. This is particularly the case with those over the olecranon, which may become enlarged until they are almost as big as oranges.

* *Tophus* or *tofus*, the Greek τόφος, seems to have been applied to rough crumbling rock, the disintegrated volcanic *tufa*. Virgil associates it with chalk: *Et tophus scaber, et nigris exesa chelydris Creta* ('Georg.,' ii, 214).

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A man used to attend our out-patient room with numerous tophi in the skin of the scrotum; a second much earlier case is figured in the Guy's Museum; and a third is cited by Duckworth from Mr Butlin's practice at St Bartholomew's Hospital.

Sometimes when these tophi are large, the skin wears through. Nodules in the ears may thus be cast off, and the patient freed from their presence. Sir Thomas Watson tells the story of a gentleman who had exposed tophi on his fingers, and who used, when playing at cards, to score the game upon the table with his gouty knuckles.

Tophi may cause suppuration in the tissues round them. Although a joint affected with acute gout often looks so inflamed that one might imagine it ready to point, yet as a matter of fact this never happens. But abscesses round the extra-articular deposits in chronic gout are very common, and the lithate of soda is freely discharged from them, mixed with pus. Garrod speaks of as many as five or six abscesses of this kind being open at one time on each hand, and others on the feet, and he remarks that they give rise to very little constitutional disturbance.

It was once supposed that deposits of urate of soda were to be found only in advanced cases of gout, and, indeed, that their presence was somewhat exceptional. Sir Charles Scudamore asserted that they did not occur in one gouty case in ten. This, however, is true only so far as concerns masses of the urate deposited outside the articulations, and large enough to be seen and felt. There is reason to believe that within every joint, affected by the disease, urate of soda is always present in greater or less quantity. Garrod mentions two patients, of whom one had only a single attack of gout thirteen years before his death, while the other had had two attacks within the last two years of his life. In each case a small quantity of urate of soda was found as a white deposit upon the surface of the joints, which had been affected by the disease.

Pathology.—In considering the theory of gout we must start from the fact that the blood in this disease contains an excess of uric acid, in the form of urates of soda and potass.

Garrod has not only shown that excess of uric acid can be detected in the blood in gout by an elaborate chemical analysis, but also that for clinical purposes a simple method of determining its presence may be employed. About two drachms of the serum of blood are put into a flat glass dish, somewhat larger than a watch glass, and acetic acid is added so as to give a slight acid reaction. A fibre from a piece of linen cloth is then placed in the fluid, and the dish is set aside until its contents have acquired a gelatinous consistence by evaporation. If the blood contains uric acid in excessive quantity, it is deposited upon the fibre, and this becomes studded with crystals, the characteristic appearance of which can readily be identified with a pocket lens. The serum from a blister will also yield crystals of uric acid when examined in this way, but only when the blister is placed at a distance from a joint affected at the time with gouty inflammation; and hence Garrod infers that gouty inflammation causes a local destruction of uric acid. However this may be, it appears to be certain that an attack of gout has in some way the effect of clearing the blood from its impregnation with the acid. In patients who had partially recovered from an acute seizure Garrod found a marked decrease in its quantity, and, indeed, in the intervals between the early attacks of gout he failed to detect any appreciable amount of lithate

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in the blood. This tendency of gouty inflammation to free the blood from accumulated lithic acid accounts for a fact which appears to be well established, that an attack of acute gout often leads to the rapid disappearance of certain other symptoms. These are commonly described as "irregular" gout, the ordinary affection of the joints being "regular" gout. Other epithets are "atonic," "latent," "lurking," "masked," and "misplaced." Most of the symptoms have already been described as effects of lithæmia (p. 333).

Garrod has demonstrated by direct experiment the fact that excess of uric acid is present in the blood "in some cases where symptoms of irregular gout were present without any accompanying joint disease." He shows that lithate of soda is invariably present in a gouty joint; and he supposes that the salt is deposited not as the result of the inflammation, but before it occurs—that, in fact, the presence of urate of soda in a joint is a condition antecedent to an attack of gout, and very probably its cause.

In the pinna of the ear gouty concretions commonly form without any indications of previous inflammatory action, and they are seldom followed by local inflammation. In some cases the patient experiences sensations of heat and pricking and the part is tender, but more often he is quite unconscious that such concretions in the pinna are present. Judging from the observations, which were made by Dr Moxon and the author in the deadhouse, one would infer that in the interior of joints also the deposition of urate of soda sometimes takes place no less slowly and with the same absence of symptoms. At any rate, we have found the articular cartilages of the great toe encrusted with the salt in many cases in which no mention of gout had been made during life. So that, perhaps, the occurrence of a definite gouty seizure is often more or less of an accident in the course of an essentially chronic process.

This, however, is not exactly Garrod's view. He states that among a large number of autopsies on persons who were not known to have gout, there were only two in which even a slight trace of urate of soda was found in the great toe joints. He supposes that the deposition of the salt in a joint is always the immediate precursor of a gouty seizure.

There are two conditions which may be supposed to cause a rapid increase in the lithate of soda in the blood, and so lead to its deposition in the joints and provoke an attack of gout: namely, (1) the ingestion at a particular time of such food as "disorders the liver" and produces lithæmia, and (2) the failure of the kidneys to excrete lithic acid.

The first of these conditions is well known to be a frequent exciting cause of a gouty seizure. Scudamore mentions the case of a gentleman, free from hereditary tendency and with no reason to suspect that he would be attacked, who was seized with gout for the first time after three or four days of excessive conviviality. Another striking case is that of a gentleman who had never had gout in the summer and who, persuaded of his security, drank six or seven glasses of champagne; in twelve hours he had a fit of the gout. In three instances patients had sat down to dinner with scarcely the sensation of gout, but when rising to leave the table found themselves completely disabled.

Garrod, however, believes that in many cases the other condition (that of the kidneys) is really operative. He found that in severe cases of acute gout the amount of uric acid contained in the urine was on an average less than four grains daily—the normal amount being eight or ten grains. It is true that in such cases the urine as it cools often deposits lithates, which are

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of a bright pink or red colour such as has generally been supposed to be characteristic of lithæmia, and to indicate an excessive secretion of these salts. But this conclusion is fallacious; for, first, the quantity of urine passed in the twenty-four hours is diminished, and secondly, its acidity is much increased, so that the whole of the lithates are deposited. As gout becomes more and more chronic, uric acid is excreted by the kidneys in diminished amount even in the intervals between the attacks, and in advanced stages of the disease it may be entirely absent from the urine. But the *blood* in chronic gout always contains excess of uric acid in the form of urate of soda.

Sir Wm. Roberts has recently ascertained that the condition in which lithic acid normally circulates in the blood is that of sodic quadrurate (as in the urine); but in the gouty state, whether from renal inadequacy or some other reason, the quadrurate lingers too long in the blood and so becomes there gradually transformed into sodic biurate, a far less soluble salt. This process can be imitated out of the body by mixing a solution of the biurate of soda, with one of bicarbonate of the same base. When the accumulation of the biurate has reached a certain point of saturation, it is suddenly precipitated as a crystalline deposit in the joints or elsewhere; and it is believed that the tissues are less alkaline than the blood, and the cartilages the most nearly neutral of all, and that blood, synovia and cartilage are all less alkaline in gout than in health.

The question still remains why one part of the body more than another is subject to the deposition of the crystalline lithate of soda. Garrod's hypothesis is that the crystals are formed especially in those parts which are not vascular or in which the circulation is sluggish. This applies both to the pinna of the ear and to the articular cartilages, and in reference to the former he says that persons with cold ears are most apt to have gouty concretions in them. In the knee-joints, too, the parts which are in contact with the vascular fringes remain free.

The reason why the metatarso-phalangeal joint of the great toe should be attacked by gout in preference to all other joints is supposed by Garrod to be the fact that it is subjected to so much pressure from the weight of the body, and also to sudden shocks. When any other joint has been previously injured it is frequently made the first victim of a gouty seizure.

Accepting the strictly chemical or (it used to be called) humoral origin of gout in excess of uric acid in the blood, we naturally suppose that this depends on some error in the metabolism of the proteid elements of the food. To explain this faulty metabolism the ready influence of the nervous system has been invoked, or direct trophic nerves which determine the arthritis have been assumed, and even articular centres in the cerebrum have been invented to complete the nervous theory of gout.

For some reason it has become common to ascribe bronchitis, dyspepsia, gastralgia, iritis, cystitis and urethritis, phlebitis, eczema, and even psoriasis to a gouty diathesis.* But the evidence is very slight, and the "gout" to which such evidence applies, is the distillation of morbid humours which belongs to a bygone pathology, not deposit of urate of soda in the tissues. There is no reason to believe that gout ever "flies to the stomach," but over-indulgence at the table may produce acute dyspepsia as well as inflammation

* Crabbe satirises this weakness of our art as follows:

"One to the gout contracts all human pain,
He views it raging in the frantick brain,
Finds it in fevers all his efforts mar,
And sees it lurking in the cold catarrh."

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of the great toe; elderly people are liable to gravel, gout, stone, and cough; and while lead and drink may lead to gout and chronic Bright's disease, cirrhotic kidneys favour an attack of gout.

It is doubtless possible that the deposition of lithate of soda causes inflammation in other tissues besides the joints; as neuritis of the sciatic nerve, or inflammation of a varicose vein, of the conjunctiva or the bronchia. The proof, however, must be given in each case.

Ætiology.—In the first place, gout is without question an *hereditary* disease. Garrod found that more than half of all his gouty patients could prove an inherited disposition to this disease; and he says that among persons belonging to the upper classes the proportion is considerably greater. Even among hospital patients the writer found 21 out of 61 cases with a clear hereditary history.

Sir Charles Scudamore found among 522 patients suffering from gout, 190 in whom no hereditary taint could be traced. Of the remaining 332 patients, 181 inherited it from the father, 59 from the mother, and 24 from both parents, while the other 68 had grandparents (44 cases), or uncles (21) or aunts (3) who had suffered from the same disease.*

The popular idea that the disease often misses a generation probably only applies to the case of transmission from a grandfather through his daughter to his grandson. Mr Hutchinson ('Med. Times and Gaz.,' 1876) believes that the younger children in a family are more likely than the elder to suffer, and to suffer severely.

The *age* at which a first attack of gout is most apt to occur is between twenty-five and forty. In persons less than twenty years old the disease is very rare. In early cases there is always a strong hereditary predisposition, and often the habits of the individual are also such as favour its development. Garrod has seen it in the great toe of youths of sixteen. Scudamore mentions two cases in boys of eight and twelve, but neither was seen by him and they were possibly not genuine: his own youngest patient was fifteen. Sydenham said, "I have not hitherto found children or very young persons affected with the true gout." The aphorism of Hippocrates is still true: *παῖς οὐ ποδαγριᾷ πρὸ τοῦ ἀφροδισιάσμοῦ* (vi, 30).

Beyond the age of sixty-five a first seizure seldom occurs: Scudamore had never seen a case, but Garrod records one at eighty and another in a woman nearly ninety. Among sixty-one cases collected by the writer, the first attack occurred once at seventy, and one of the worst cases of (hereditary) gout was in an abstemious young woman of twenty-one, who was under his care when her father, who was an intemperate man, was also a patient with gout.

The disease is much more common *in men* than in women, perhaps as much as eight or nine to one. This doubtless depends mainly upon the fact that their habits more frequently tend to develop the disease. It was said that the intemperance of the Roman ladies under the Empire led to gout becoming common among them. The occurrence of the catamenia during a large part of female life perhaps helps in warding off gout, for it seldom

* The hereditary character of gout seems to have been unknown to the classical writers, but it is referred to by Aëtius in the sixth century. It is clearly stated in Pirkheimer's 'Apologia seu Podagræ laus,' published at Nürnberg in 1522, and afterwards translated into German (1577) and into English ('Praise of the Gout,' translated by W. Est, Lond., 1617); and it was made part of the definition of true gout by Cullen.

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attacks women until after the cessation of the menstrual function.* Among seven female patients with gout at Guy's Hospital, in three it was hereditary and in three it was caused by drink.

High living, with excessive indulgence in *animal food*, is generally supposed to be a cause of gout.

As regards the power of *intoxicating drinks* to produce gout, there is no doubt whatever; but all liquor does not act equally to this effect. The fact that malt liquors are more apt than ardent spirits to produce gout is well shown by comparing the working men of London with those of Edinburgh and Glasgow; the former drink beer and porter and are very liable to the disease; the latter drink little but whisky, and although they are by no means sparing of this, they scarcely ever have gout. The rarity of gout in many of the cities on the Continent, where distilled spirits form the chief intoxicating beverage, is another proof that they have little tendency to produce it. It also shows that Bavarian and Strassburg beer has not the same effect as London porter. Perhaps the quantity drunk, and its powerful diuretic effect, may account for this as much as its weaker alcoholic strength. With regard to the different kinds of wine, gouty patients well know the danger of indulgence in port, champagne, Burgundy, or Madeira. Garrod gives a caution against sherry also—however dry and pure—as by no means so innocent as is imagined, and thinks that cider, when sweet and partially fermented, is apt to cause gout, although rough cider is comparatively harmless. With regard to the causes which render one kind of alcoholic drink more injurious than another, no positive conclusion can as yet be drawn. Their comparative effects in causing dyspepsia, and on the other hand in leading to free diuresis, are probably important.

A fact which Sydenham long ago observed, in his description of gout, is that the first attack generally occurs in the *winter season;* he says towards the end of January or the beginning of February. Why this should be the case is not very clear. In many patients the disease returns for two or three years in the spring only; after a time, a second attack occurs in the autumn; and at length the seizures occur quite independently of the season.

The influence of cold in the development of gout is shown by the effects of change of *climate.* Garrod says that a gouty man may often escape his accustomed winter attacks by going to Malta or Egypt. In hot countries, this disease appears to be very rare, or unknown. Even in the south of Europe it is much less frequent than in England; but we must remember that the habits of the people of different countries are very different.

Certainly the disease seems to have been more common in ancient Greece and Rome than in modern Italy, Spain, and France. Pliny says that gout was not so common before his time in Italy, and to prove that it was no native of Italy argues that it had no Latin name (lib. vi, cap. x). To the same effect Sir Wm. Temple writes: "Among all the diseases to which the intemperance of this age disposes it, at least in these northern climates, I have observed none increase so quickly within the compass of my memory and recollection as the gout." Among sixty-four autopsies at Vienna, the writer, when studying there under Rokitansky in 1865, only once saw a case of gout, and the deposit in the joints was not recognised by anyone but Rokitansky himself. It is believed to be more common in Paris

* Here again the aphorism of Hippocrates holds: *Γυνὴ οὐ ποδαριᾷ ἢν μὴ τὰ καταμήνια αἰτίη ἐκλίπῃ*, vi, 29.

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and Berlin than it used to be, and also in the United States. It is said to be rare in Russia and Scandinavia, notwithstanding cold and alcohol; and to be more frequent in Holland and Flanders. It is undoubtedly less common in Scotland and Ireland than in England. Pagenstecher speaks of gout with respectful envy as the heritage of "die reichen Söhne des gesegneten Albions."

A remarkable fact in the causation of gout has been established by Garrod, namely, chronic *poisoning by lead.** He found that about 30 per cent. of the gouty patients in his hospital practice had been exposed to the influence of lead. Some of them were painters and plumbers, others workers in lead mills, and others "composite-doll" makers. A careful inquiry into the habits of the men failed to show that they had been less temperate in their habits than other men of the same class. That this is a true cause is sufficiently established by everyday hospital experience in London, but the way in which lead produces gout is at present doubtful. Probably the explanation of this fact involves that of another which Garrod believes he has established: that persons who are already gouty are more susceptible than others to be affected by lead. He says that in several instances he has found those patients to be of gouty habit, or to have already had severe attacks of gout, in whom the medicinal administration of the preparations of lead has produced colic or a blue line upon the gums with unusual rapidity. Possibly the excretion of lead in the urine leads to irritation of the kidneys, and thus to chronic Bright's disease, and retention of uric acid. Saturnine gout is said to be rare in the north of England (Dr Thomas Oliver, Goulstonian lectures, 'Brit. Med. Journ.,' March, 1891). Dr Lorimer, of Buxton, has published an account of 107 cases—most of them occurring in visitors (*ibid.*, July 24th, 1886). He finds that it usually befalls patients earlier than hereditary or than alcoholic gout, that the fits are less severe but more lingering, and that albuminuria is almost constantly present.

An occasional exciting cause of gout is *mental fatigue or anxiety.* Sir Charles Scudamore mentions the cases of two female patients, in each of whom a severe first attack of gout was brought on by sitting up for several nights in succession, nursing a sick relation. Severe intellectual labour, watching, sorrow, and exhaustion from sexual excesses, may each be the occasion of a fit of the gout.†

Diagnosis of gout is in some cases very easy, in others exceedingly difficult.

(*a*) When *arthritis* is actually present and we can exclude primary local disease, the diagnosis is limited to gout, rheumatism, gonorrhœal synovitis, pyæmia, and osteo-arthritis.

A difficulty has been supposed, from the existence of a form of inflammation, intermediate between gout and rheumatism; or from the existence of both together. But there is in reality no *tertium quid.* Either the affected joint contains urate of soda or it does not: in the former case the disease is gout, in the latter it is not. Doubts never arise in the dead-

* See also Wilks in the 'Guy's Hosp. Rep.' for 1870, vol. xv, p. 40.

† The ancients laid stress on the last exciting cause:

"Λυσιμελοῦς Βάκχου καὶ λυσιμελοῦς Ἀφροδίτης
Γεννᾶται θυγάτηρ, λυσιμελὴς ποδάγρα."

May not there have been some confusion between gout and gonorrhœal synovitis, or syphilitic periostitis?

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house as to the gouty nature of disease in a joint. As for the simultaneous presence of gout and rheumatism in the same patient, its theoretical possibility must be admitted, but practically it does not occur oftener than coincidence of acute rheumatism and enteric fever, or of scabies and scarlatina, or of syphilis and psoriasis. The term "rheumatic gout" is either a bad name for osteo-arthritis, or is a mere excuse for shirking a difficult diagnosis.

The following characters suggest an affirmative answer to the question whether a case is one of gout. (1) That the small joints are affected, and particularly the great toe-joint; (2) that the attack began suddenly in the night, especially if it be a first attack; (3) that the skin over the affected joint is tense, shining, and red, with distended veins around it, after a day or two œdematous, and finally desquamating; (4) that the febrile disturbance is moderate, and in proportion to the local inflammation. The sex, age, and family history of the patient have also to be taken into account; and the state of the heart and kidneys must be investigated. All those parts which are apt to be the seat of tophi should be carefully examined; for if a single deposit of urate of soda can be found, it settles the question. But care must be taken not to mistake other kinds of enlargement of the finger-joints for those caused by gout, and it is needful to distinguish from tophi in the pinna of the ear comedones of the sebaceous glands, and that occasional nodule in the edge of the helix which Darwin described.

In exceptional cases other forms of multiple arthritis beside rheumatism may be mistaken for gout. Garrod mentions a case in which the great toe was swollen, tense, red, and hot; in which, in fact, the joint looked exactly as though it were affected with severe gouty inflammation; but the disease turned out to be *pyæmia*. The diagnosis from gonorrhœal synovitis and osteo-arthritis will be best considered in the following chapters.

(*b*) In cases where *no articular inflammation* is present the recognition of gout is a matter of far greater difficulty, and often it is impossible. If the patient has suffered from unmistakeable gouty arthritis before, or if he comes of a family with strongly marked "disposition to gout," *i. e.* with many members frequently and severely affected with genuine articular gout, we may then reasonably ascribe wandering pains, headache, lumbago, or sciatica (cf. vol. i, p. 392), to the presence of excess of lithic acid in the blood, and may anticipate an attack of arthritis.* But in the absence of such evidence the assertion that dyspepsia, bronchitis, cutaneous diseases, cardiac palpitation, asthma, gonorrhœa, iritis, or any other disease is of a gouty origin, cannot be called anything but arbitrary, and it has the fatal objection to all such easy "explanations," that they can be neither proved nor disproved; they only check investigation. An "arthritic diathesis" is often made to play the same part as "an inactive liver," "nervous debility," a syphilitic taint, a strumous cachexia, or a "scorbutic habit of body," or an "unhealthy state of the system generally;" the more or less discredited commonplaces of bygone pathology which, when abandoned by the physician, still linger among the laity. As the existence of non-articular gout or a gouty diathesis cannot be proved, so it cannot be refuted; for if the patient dies and no lithate of soda is found in his joints,

* The late Professor Hebra used to relate how a patient, who had gone the round of "baths" and "cures" for *Gicht*, came for his advice, and was found when the feet were examined to suffer from nothing but corns. He did not, however, anticipate that corns themselves would one day be regarded as a proof of the gouty diathesis.

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"one must not be bound by too narrow and mechanical a theory of the disease;" if an inflamed joint can be clearly traced to a traumatic origin, it is argued that the gout was only thus determined to a particular spot; if the patient has lived freely, the diagnosis is clear; if he is abstemious, he suffers from the excesses of his forefathers; if the urine deposits lithates on a cold morning, the existence of lithiasis, of lithæmia, and of a gouty disposition are easy steps in reasoning; if the urine is albuminous, he has a gouty kidney; if it is pale and free from deposit, that is a proof that uric acid is not properly eliminated, and must be accumulating in the blood. Diabetes is confessedly gouty, so is dyspepsia, gravel, bronchitis, and asthma, and "skin diseases" in general. If any doubts remain they must yield before the voluminous testimony which is laid before us every year, of the sovereign effects of the fashionable waters of the Continent where cures are still wrought.*

We must learn to explain less and to investigate more; and by keeping to the narrow path of terms which admits of strict definition, of ætiology which is logically demonstrable, and of diagnosis which can be verified or refuted, slower but surer progress will be made, and our successors will not regard our pathology as we do that of Eugalenus.

Course and prognosis.—Acute gouty arthritis appears never to be directly fatal; the prognosis relates to the ultimate effect on the health of the patient.

It was once deemed rather an honour than a misfortune to have the gout; it showed that not only the man himself, but perhaps his father and grandfather before him, had been able to afford good living. Sydenham himself, after suffering for thirty-four years, speaks of it as a comfort that gout, unlike any other disease, kills more rich men than poor, more wise men than simple. "Great kings," he says, "emperors, generals, admirals and philosophers have all died of gout."

At the present day any consolatory reflections of this kind may be checked by the consideration, that all insurance offices charge an additional premium to anyone who has had even a single attack of gout. Their actuaries know well that the disease tends to shorten life. However slight it may have been, a seizure of gout is always an admonition that the patient's habits of life are incompatible with the preservation of sound health. Sir Thomas Watson says that "in not a few instances men of good sense, and masters of themselves, having been warned by one visitation of the gout, have thenceforward resolutely abstained from rich living and from wine and strong drinks of all kinds; and they have been rewarded by complete immunity from any return of the disease; or at any rate, its future assaults

* The following is extracted from an advertisement of a certain mineral water:—"I am very much pleased to be able to inform you that, according to the advice of my friend, ——, M.D., medical officer of the staff, I used your mineral water against the arthritic affection, from which I have been suffering the last ten years, with such success that I think it my duty and in the interest of suffering humanity to relate to you the history of my illness. Already ten years ago I had an attack of an acute rheumatic fever, which developed itself to such a severe gout that I had to keep in bed, and was suffering from the most awful pains for weeks every spring. I tried different cures and visited watering-places, but without any effect. In the month of August last year I commenced to drink, and after having consumed 150 bottles I was delivered from all sufferings, and was able to get out of bed. The inflammatory alterations in the joints and the chronic sediments of uric acid disappeared, and I can say to be totally cured. S— promotes the secretion of urine, and acts also very favourably on digestion, and is a very pleasant refreshing drink. 28th May, 1887. F— B—, curate in the Austrian Army."

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upon them have been few and feeble." "I am sure," he adds "it is worth any young man's while, who has had the gout, to become a teetotaler." For the earlier the age at which a first seizure occurs the worst is the prognosis; and particularly if there be an inherited predisposition. Garrod says that he has known thirty-five years elapse between a regular attack of gout in the great toe and the patient's death, which latter event took place when he was seventy years old. He has also seen several cases in which the disease, after having recurred periodically for many years, gradually declined in intensity and duration, and at last altogether disappeared.

The repetition of attacks of gout is a serious matter, if only on account of the crippled state of limbs which it induces, preventing the patient from taking exercise, and destroying his enjoyment of life.

But the main risk connected with the gouty condition is its liability to induce *chronic interstitial nephritis*, or renal cirrhosis, so that Dr Todd called the "small, red contracted kidney" the "gouty" kidney. When the disease is caused by gout, white streaks are often seen, running in the course of the straight tubes in the pyramids; these white streaks consist of prismatic crystals of urate of soda and of amorphous masses, blocking up the tubes, and also embedded in their walls.

The signs by which this affection of the kidneys may be detected have been fully discussed already (pp. 493, 498). A prolonged examination of the urine, both chemical and microscopical, is often necessary. But merely looking at the secretion may be enough to excite suspicion. Sydenham remarked that in cases of long-standing gout the urine, "no longer high coloured, is pale and copious, like the urine of diabetes." He did not understand the significance of this, but we now know that it points to the fact that the kidneys are diseased. We also know that hypertrophy of the heart is an almost constant attendant upon this form of renal affection, and that apoplexy frequently results. The slightest indication of cerebral mischief must, therefore, be watched very carefully in such cases, and it is often right to warn the patient, or his friends, that he should not be left alone at night, nor while out of doors.

Evidence of Bright's disease was found in a third of the writer's sixty-one cases of gout. In many of them atheroma was present, and it also has been called gouty. The calcareous deposit, however, contains no urate of soda. The atheroma, like the renal cirrhosis and the dyspepsia of gout, is probably not the result of excess of uric acid, but, like them, follows over-indulgence in eating and drinking.

In ten consecutive cases of death in patients, who suffered from repeated attacks of gout, two were due to cancer, and in the other eight the writer found granular kidneys. The immediate causes of death were renal pericarditis, pleurisy or pneumonia, uræmic coma, and cerebral hæmorrhage or softening with hypertrophied left ventral and diseased arteries.

Of diseases of the skin the only one which has any probable relation to gout is *eczema*. This is so common, that few cases of eczema are gouty, but those affected with chronic gout are more subject to eczema than other persons of the same age, and the disease is apt to be particularly irritable and is more readily curable by alkalies and colchicum.

Bronchitis in gout may be arthritic or renal or senile; not infrequently it proves the immediate cause of death in long-standing cases.

It has been said of gout, as of cancer, that it is incompatible with *phthisis*. This, however, is not the case. The fact that they affect different periods

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of life, so that few have gout who die under thirty, explains their seldom being met with together, but in the small number of sixty-one cases above quoted the ordinary symptoms of phthisis occurred three times in patients with well-marked gout. In a fourth case of gout tubercles were found after death in the lungs, and at the present time (February, 1888) a patient in Philip Ward with unmistakeable tophi is also suffering from tuberculous disease of the larynx and lungs.

Treatment.—The pain of a fit of the gout is so great that every effort should be made to relieve it. The great Dr Harvey was one of the eminent men subject to this complaint, and he used to cut short a fit by plunging his feet into cold water. This and like attempts to stop the local inflammation were always condemned on the supposition that they "drove in the peccant humour," and would lead to gout of the stomach or the brain.* But no recent and carefully observed cases of such treatment seem to be on record, and the judicious Heberden remarks: "I do not recommend Harvey's example (of which I have been told by some of his relations) as proper to be imitated, though it is known he lived to a good old age; but I am not warranted by any experience to condemn the practice of endeavouring by exercising the limb, to prevent the gout from settling there." There are numerous instances of persons subject to gout who, on the first intimations of a fresh attack, have begun walking, riding, or using prolonged and violent exercise, and have thus escaped the threatened invasion.

Hot fomentations sometimes give ease; more often evaporating lotions of chloroform, ether, or alcohol. Lead and opium, infusion of poppy-heads, or laudanum on lint lightly wrapped in cotton-wool, may each have a good effect in different cases. Local applications of colchicum are useless, blistering and iodine are hurtful, and leeches do not seem to relieve the inflamed joint as one would have hoped.

A solution of atropine and hydrochlorate of morphia may be used as a sedative, in the proportion of one grain of the former and eight grains of the latter to the fluid ounce, applied on a small piece of lint beneath oil-silk. A lotion containing a drachm of the spiritus ætheris sulphurici to six ounces of water has sometimes proved serviceable. Scudamore recommended a lotion composed of one part of alcohol and three of camphor-water, made agreeably lukewarm and applied on thick linen compresses.

Garrod recommends that oil-silk or gutta-percha sheeting be carefully applied, so as to keep in the moisture exhaled from the skin, and form a kind of vapour bath. He has seen irremediable injury from the application of leeches to joints affected with gout, and two cases in which the patients completely lost the use of both knees. Hot poultices are said by Scudamore to be very injurious.

Happily the *internal treatment* is more efficient, and gout is one of the

* Sir Thomas Watson mentions that Dr Parry, of Bath, had at one time two patients under his care, each of whom had attempted to cut short or to ease a paroxysm of gout by plunging the affected part into cold water. This gave instant relief to the pain, and the inflammation presently abated, but in each case hemiplegia occurred a few hours afterwards. Trousseau relates a case that occurred to Dr Demarquay of a gentleman who applied cold water compresses to his foot, which was affected with very severe gout. The pain was almost immediately relieved, but a few hours later the patient fell into a state of apoplectic semi-stupor, which disappeared under the use of sinapisms to the foot, and consequent return of the articular inflammation. These cases certainly resemble one another so closely that it is difficult to believe that the cerebral disturbance was a mere accident.

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few diseases for which modern medicine has found a specific remedy. When Sydenham wrote, he spoke of the possibility of such a discovery, which he said would delight him above all other physicians; but he knew of no specific for gout. Cullen, a century later, advised patience and flannel alone.*

The ancient writers recommended hellebore and hermodactyl. The latter plant has been identified with *Iris tuberosa;* it was probably a species of colchicum, but not that which is now officinal. The Portland powder was a famous nostrum for gout in the last century, until it was eclipsed by the *eau médicinale de Husson.* Its success was unmistakeable, and it was at length discovered that it was prepared from meadow saffron (*Colchicum autumnale*). There has been much discussion as to the use of colchicum in gout, and, perhaps, it bore a taint from its introduction as a quack nostrum, for many writers have depreciated its value. Some have asserted that when it acts beneficially it is only by purging; others, that even when it removes the local symptoms it leaves "the disposition to the disease much stronger in the system." This was the opinion of Scudamore, and Trousseau advised his hearers to cross their arms and look on, doing absolutely nothing to subdue an attack of acute gout.

Very different from this is the teaching of Watson and of Garrod. The former prescribed forty minims or a drachm of the colchicum wine in a saline draught at bedtime, and half-a-drachm more in a warm draught the next morning, repeating the sequence if the disease continued. The latter gives about twenty minims of the wine every six hours. Both say that the effect is almost magical; the pain is calmed, and the swelling reduced often within a few hours. The tincture, made from the seeds instead of the corm, is supposed to be more powerful, and to have a greater purgative action.

It is certain that the curative action of colchicum is not dependent upon its purgative operation; for it is often effectual when it does not act upon the bowels at all. Sometimes, however, this remedy produces an effect which may enable a patient accustomed to its use to tell that he is taking it. The stools become green, and if a loose motion is passed it is said to resemble green pea soup.

In the severity of a gouty attack it is well to add twenty drops of laudanum to forty of colchicum wine; but the freedom of the kidneys from disease must first be ascertained. It is common to give a blue pill or colocynth and calomel purge at the same time, and probably with reason.

Many French physicians advise the administration of salicylate of soda in acute gout, but experience in England shows this to be inferior to colchicum in immediate effect, and the results are sometimes negative, if not injurious. It is, however, right to add that Dr Haig has found the output of lithic acid increased under the influence of salicylates.

* Sir William Temple gives an admirable description of how he cured his first fit of the gout when he was ambassador at the Hague, and forty-seven years of age, in February, 1674, by applying a certain kind of moss that grew in the East Indies, and is called a moxa, to his foot and setting it alight. This kind of actual cautery was afterwards much employed in sciatica and other painful disorders, but it is now almost forgotten. According to Temple it more than once proved effectual in his case, and also in one of toothache.

He narrates of Prince Maurice of Nassau, that though often attacked with gout he laughed at it, and was sure he could always cure it with one remedy, which was: to boil in water a good quantity of horse-dung from a stone-horse of the Hermelinne colour, and set his leg in a pailful of it as hot as he could bear it. Temple adds that "He even had a set of such Hermelinne horses in his coach, which he told me was on purpose that he might never want this remedy." He mentions other treatment, including carbonate of ammonia internally, and shampooing and urtication locally, and concludes that exercise and temperance are better than them all ('Works,' vol. iii, pp. 238–265).

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It is probable, as Sir Thomas Watson observes, that the striking efficacy of colchicum in attacks of gout has often led men to disregard those precautions of regimen and diet by which alone the disease can be kept at bay. Having what they deemed a specific, they have cast aside all restraint, and before long they have had a fresh seizure.

The *diet* in an attack of acute gout should for the first few days consist of milk, arrowroot, sago, tapioca, and the like, with water or toast and water. A little brandy may be given if the previous habits of the patient render it necessary, but not otherwise. It must, however, be added that even a first seizure of gout sometimes occurs in a patient so broken down in constitution as to require as much nourishment as the stomach can readily dispose of, such as beef-tea, soup, and eggs.*

The *treatment of chronic gout* must be in some respects different from that of an acute attack. But it is a great mistake to suppose that colchicum is useless, even when the joints are deformed and crippled.

Alkalies are decidedly useful in gout, and they may often be advantageously given with colchicum; the traditional way is to add ten or fifteen grains of carbonate of magnesia to fifteen or twenty of colchicum wine in a draught, taken thrice daily; another way in which alkali can be given is in the form of salts of citric, tartaric, or acetic acid; and in the hope that the base may combine with uric acid, and lead to its elimination by the kidneys, it is best to prescribe potass rather than soda; for the urate of potass is much more soluble than that of soda, and potass also possesses great power as a diurétic. The salts of lithia were introduced by Garrod as possessing some advantage over those of potass in the still greater solubility of the urate of that base; perhaps this gain does not outweigh the disadvantage of their higher price. The carbonate of lithia may be given in doses of five to ten grains dissolved in aërated water, or the citrate in doses of eight to twelve grains or more. Whatever alkaline salt is prescribed, it should be largely diluted with water, and taken at least an hour before meals, when the stomach is empty.

Dr Haig has shown by strict observations that alkalies increase the excretion of uric acid, while acids decidedly diminish it.

Sarcosin, which diminishes the excretion of uric acid, has been recommended on theoretical grounds as a remedy for gout by Schultzen.

Other remedies which are useful in certain cases of chronic gout are guaiacum and iodide of potassium. Garrod says that he has given the former of these medicines extensively, especially for the asthenic gout of old subjects; a patient may go on taking it for a whole year at a time. The latter is particularly useful when the pains are increased at night by the heat of the bed; and also when the joints contain fluid of which the absorption is slow. Bark and quinine are also useful in some cases of what is called atonic gout, *i.e.* when the attacks have become more frequent, short and slight, while the patient's digestive powers, his strength, and his appetite fail. In these cases opiates are often more useful than colchicum during one of the subacute attacks; and stimulants, particularly old port wine and even good champagne, are not only admissible but therapeutically valuable.

In other cases a patient subject to gout should live an abstemious life, taking no stimulants, and meat only once a day, eating freely of vegetables and fruit and taking occasional saline laxatives. Alkaline and diuretic

* "Potus theæ et caffeæ inter reliqua remedia calculosis et podagricis excellunt."—Baglivi: Prax. Med., lib. i, p. 117.

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waters are no doubt often valuable aids to treatment and prophylaxis. Even more desirable are active habits and habitual exercise in the open air (cf. pp. 164, 332, 336).

Certain *baths and mineral springs* have so great a reputation for the relief of gout that there are few patients, among the richer classes, who do not sooner or later visit one of them. Of such resorts, Vichy is at present the most in vogue; the main ingredient of its springs is carbonate of soda in the proportion of about forty grains to the pint. Another very similar water is that of Vals. Many persons who are robust and of full habit derive much benefit from these springs; but to feeble patients they often do harm. Garrod says that in very chronic cases a tendency to the formation of concretions about the joints has appeared in his experience to be hastened by the use of Vichy water. Trousseau advised that as a general rule alkaline water should not be taken for more than ten or twelve days at a time, and only in small quantities, for not a year passed in which he did not see evil consequences from their prolonged use. Mineral waters should never be taken when an acute attack is present or threatening; nor by patients who have organic diseases of the heart or kidneys. It is worthy of remark that, according to Heberden, the Bath was valued during the eighteenth century for the efficacy of its waters in *bringing out* an attack of gout.

For patients with symptoms of hepatic disorder, the waters of Carlsbad and the other springs mentioned under lithæmia (p. 335) are often very useful. When the circulation is sluggish or the secretions deficient, Wiesbaden is indicated, the water of which contains a large amount of chloride of sodium; or Aix-la-Chapelle. Old or infirm persons may be sent to Bath or Buxton, where the waters are hot and the active saline ingredients small in quantity; or Teplitz or Gastein may be chosen, if more distant places are preferred.

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RHEUMATISM*

"Joint-racking rheums."—MILTON.

"The throttling Quinsey 'tis my Star appoints,
And Rheumatisms descend to rack my joints."—DRYDEN.

History and Definition—Onset, course, and symptoms—Anatomy of the inflamed joints—The pyrexia, sweating, &c.—erythema—cardiac complications—pleurisy, tonsillitis, nodules—Ætiology and pathology of rheumatism—Prognosis: fatal cases—Rheumatic hyperpyrexia—Diagnosis, particularly from pyæmia—Rheumatism in children.

Treatment by older methods—Observation of the natural course of the disease—Treatment by salicin and salicylates—Treatment of rheumatic hyperpyrexia.

Chronic articular rheumatism—Muscular rheumatism.

The name of rheumatism, like that of gout, carries with it the impression of the system of humoral pathology, the words *rheuma* and *catarrh* are used by the Greek writers, from Hippocrates downwards, as having similar meaning, and their etymology is also alike, for the one term was derived from ῥέω, and the other from καταρρέω. The notion was that of an acrid humour, generated in the brain and distributed over the body. In the course of time, diseases of the mucous membranes became known as catarrhs, while the name of rheumatism was applied to painful affections of the joints. Baillou or Ballonius,† is said by Bright and Addison to have given it this meaning, and he also distinguished it from gout or *gutta*, another name witnessing to the humoral doctrines of Galen. Holland in his translation of 'Pliny's Natural History,' speaks of the "sharp and eager flux of phlegm," which the Greeks call rheumatism (lib. xxii, cap. 18 and 25). *Destillatio* was a term applied in common to catarrh, rheuma and gutta. The conception of rheumatism became modified in some countries so as to include the notion of its production by external cold. All sorts of complaints have accordingly been termed rheumatic; some of which the cause was unknown because they were painful; others, although painless, because cold was the supposed cause.‡

But there is a very common, definite and important acute disease, which was first accurately described by Sydenham in 1670, and for which we have

* *Synonyms.*—Rheumatic fever—Acute and subacute Articular Rheumatism—Arthritis vaga—Polyarthritis rheumatica acuta—Rheumarthritis.

† He died in 1616 and his posthumous work was only published in 1642.

‡ This confusion is still common with good German writers, so that it is often as difficult to know what is meant by "rheumatic" as to tell the meaning of "typhus."

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no other name than rheumatism or rheumatic fever. The pyrexia, the multiple but transient synovitis, and the frequent implication of the membranes of the heart sufficiently distinguish it. We will include under the same name genuine cases, though their course may be "subacute," for they have no claim to a separate designation.

With regard to the so-called "chronic rheumatism" there is more difficulty. We shall describe separately the complaint which is known as *osteo-arthritis* or *arthritis deformans*, and which probably bears no relation whatever to acute rheumatism. But there are other cases in which a chronic joint-affection has clearly arisen from, and can only be described as a sequel of, the acute disease; and it is impossible to deny that a similar affection may, as a rare exception, be chronic from the first, and yet be of the same nature.

The use of the adjective "rheumatic" ought, of course, to be limited in precisely the same manner as that of the substantive; but in practice it is often employed loosely for various affections of the muscles and of other parts, of which the only common character is that they are caused by cold. Nevertheless, it is much better to speak of "myalgia," than of "muscular rheumatism;" for the common vague use of the term implies a connection, of which there is no proof, between "rheumatic" affections and true rheumatism.

Onset and course.—Most attacks of rheumatism begin gradually. There are no rigors nor sudden initial symptoms. For a day or two the patient feels uncomfortable, and, perhaps, complains of sore throat or of pains in the limbs. There is seldom headache, and still more rarely rigors or vomiting. Pyrexia gradually sets in, and has commonly reached its height on the second day. Presently one of the joints, generally a large joint, is found to be swollen as well as painful. In many cases inflammation of the joint is the first thing noticed.*

This affection, which is characteristic of rheumatism, is synovitis, acute or subacute, serous, comparable to pleurisy, but not leading to adhesions, nor ending in suppuration or destruction of the tissues.

It is not easy to determine clinically the fact that fluid is effused into the interior of the shoulder-joint, the elbow, or the ankle; for there may be a more or less elastic swelling at those points where the synovial membrane is most able to yield, and one may hesitate to assert absolutely that it cannot be due to exudation into the sheaths of tendons or into bursæ. But in the knee very small quantities of fluid may be detected with certainty. One has only to grasp it gently between the two hands, pressing the sides of the synovial cavity upwards, so that the fluid may accumulate beneath the patella; a slight tap upon this bone with one forefinger will then bring it down upon the condyles, giving a sensation which is unmistakeable. In those exceptional cases which prove fatal all the joints which have been affected are found at the autopsy to show distinct signs of inflammation. The synovial membrane is not, indeed, always injected, but there can be no doubt that in

* Many years ago Dr Frederick Chambers, of St George's Hospital, and Dr Francis Hawkins endeavoured to draw a distinction between two varieties of acute rheumatism, one of which they termed "fibrous," the other "synovial," and their views were adopted in Sir Thomas Watson's 'Lectures.' They were, however, altogether mistaken in supposing that the disease ever attacks the parts outside a joint rather than the articular cavity itself, and the clinical distinction they drew, so far as it is real, corresponds to that between true rheumatism (fibrous) and osteo-arthritis (synovial).

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this, as in so many other tissues, redness may subside after death; and a considerable quantity of fluid is almost always still present. This is often cloudy, with floating shreds of fibrin. Not infrequently a separable layer of lymph lines the synovial membrane, which is then very deeply reddened: or it may cover the surface of the cartilages, as, for example, in the knee-joint. Leucocytes seem to be always discoverable in the exudation, but not in large numbers. In one case, which was examined by Dr Moxon, the sheaths of the extensor tendons of the wrist were full of opaque serum and of masses of greenish-yellow lymph. Articular cartilages have in rare cases been found eroded, but in all probability this was accidental and unconnected with the rheumatism.

That the anatomical changes in the joints in this disease must be slight in comparison with those which occur under other circumstances might, indeed, be inferred from its clinical history. One of its most striking features is the rapidity, and even suddenness, with which it flies from one part to another. Thus a joint which is extremely swollen one day may be quite normal on the next day; and a little later it may be again affected as severely as before. There is not always any obvious reddening of the skin; generally some ill-defined pink patches or striæ are seen, but they are often near the articulation rather than over it. Even if, as occasionally happens, the joint shows a redder surface, which looks like gout, the surrounding veins are not dilated.

The patient dreads the gentlest touch or the jar of a passing footstep; with his head and limbs immoveably fixed he turns his eyes with anxiety as a stranger approaches his bed. He lies perfectly helpless, unable to feed himself, or even to turn in bed, paralysed by the dread of pain; so that hospital patients often speak of having lost the use of their limbs. The pain is usually worse by night than during the day. The face is pallid, the tongue thickly furred, and the face and whole body covered with profuse sour-smelling sweat.

The different joints are sometimes attacked in regular order: the right ankle, then the left; the right knee, then the left; and so on. But more often the distribution of the disease is quite irregular. All four limbs usually suffer more or less.

Of all the joints, the knees are most affected in rheumatic fever, next come the shoulders and ankles, then the wrists and hands, and then the elbows. The hips are less frequently attacked and the toes often escape, even in severe cases. The mandibular joint has almost complete immunity, and probably the same is true of the articulations of the vertebræ and pelvis, but they are too deeply seated to be examined during life, and no traces would be left if they were dissected after death subsequent to the subsidence of the attack. According to Lebert, acute rheumatism sometimes attacks the synchondroses of the pelvis.

This distribution differs from that of gout, gonorrhœal synovitis, and osteo-arthritis; moreover, in no other kind of arthritis does the inflammation shift rapidly from joint to joint.

In some exceptional cases rheumatic inflammation of a joint, instead of subsiding, passes on into a chronic stage, and ends in hydrops articuli, most likely of the knee. Ankylosis or disorganisation of the articular structures is so extremely rare that its occurrence throws doubt on the diagnosis of rheumatism.

There is no evidence whatever that rheumatic inflammation attacks any

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but synovial and serous membranes. It has no predilection for fibrous tissues, whether the fibres be white, elastic, or muscular.

The *pyrexia* of rheumatism is in the majority of cases severe in proportion to the number of joints involved, and to the intensity of the inflammation in them. But to this rule there are many exceptions. Sometimes the articular affection is well marked while the temperature is scarcely raised at all. In other cases there is considerable fever, whereas few of the joints suffer, and these but slightly. Indeed, as Graves long ago pointed out, it is possible for the fever to occur alone, without any joint-affection at all. The case which he cites was one of relapse, the patient having previously passed through two attacks of the usual kind; but the converse may occur,—an attack of pyrexia, with pericarditis or endocarditis, or perhaps even with pleurisy alone, may be followed by a relapse which is attended with synovitis, and thus reveals its real nature. It is therefore certain that acute rheumatism is a *general* disease with localisation in the joints, not a local disease of the joints with symptomatic fever. Yet even Wunderlich was unable to recognise any typical course; the maximum temperature, which is usually about 104° F., sometimes occurs as early as the fourth or even the second day, but more often not until the end of the first or the beginning of the second week. The *pulse* is often extremely rapid, and is commonly large, full, and bounding, with a short systolic period; and sometimes it is markedly dicrotic. The breathing is not proportionably hurried. The *tongue* is generally flabby, marked by the teeth at its sides, and coated with a thick, white fur. There is usually thirst and anorexia, but the patient can sleep if not kept awake by the pain. He is not apathetic as in other fevers, and is seldom delirious unless his temperature runs very high. If, however, he has been a drunkard, a form of delirium tremens is often developed. There is no tendency for the fever to assume a "typhoid" character.

The *urine* is characteristically febrile: scanty, dense, high coloured, acid, depositing pink lithates in abundance, and often crystals of lithic acid; occasionally it contains a trace of albumen for a day or two. It does not contain sugar, but often reduces copper when salicin or salicylic acid is being taken.

One of the most striking symptoms of acute rheumatism, though not invariably present, is *sweating*. The patient lies bathed in water, so as to make even the blankets damp. This must, of course, carry away much heat by evaporation from the surface of the body, but it has no obvious influence in lowering the pyrexia or in relieving the articular pains. The perspiration has usually a very sour smell, which is often taken into account in diagnosis. This, however, seems to be due to the quantity of sweat rather than to its containing an unusual quantity of free acid. Sir William Gull used to show that the reaction to litmus paper often varies upon different parts of the skin, being acid, alkaline, and neutral, in the same patient. Sweat when first secreted is not acid and only becomes so by decomposition of the sebaceous compounds mingled with it. Senator suggests that the sweat may become alkaline by conversion of urea into carbonate of ammonia, particularly between the toes and in the armpits.

There is frequently developed a copious eruption of *sudamina*—minute transparent vesicles, containing an acid fluid which is no doubt sweat. They glisten when a bright light falls upon them, but they are colourless, so that they often can more easily be felt than seen. In many cases, however,

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their bases become slightly inflamed and reddened, and their contents opalescent and alkaline: at this stage they are sometimes called *miliaria*. This eruption bears no special relation to rheumatism, for it occurs in other diseases attended with sweating, and also in healthy people during hot weather.*

In some cases of rheumatism there is another remarkable eruption, which assumes the form of erythema, urticaria, or purpura. It has often been described under the name of *Peliosis rheumatica*, originally applied by Schönlein. Dermatologists have named varieties of the same rash, Erythema tuberculatum, Roseola marginata, Erythema nodosum, and Purpura urticans. These are all forms of *Erythema multiforme;* and will be described under that name in the chapter on diseases of the skin.

Cardiac inflammation.—It is not surprising that the important relation of rheumatism to disease of the heart was not recognised before the discovery of auscultation. Scudamore in 1827 seems to have been the first to appreciate the gravity of this complication, and Bright and Hope taught it. But to Bouillaud's work, published in 1840, is due the universal recognition of pericarditis and endocarditis, not as accidents, but parts of rheumatism.

In every case of acute rheumatism it is necessary from day to day to watch with the stethoscope for indications of inflammation of the pericardium or of the cardiac valves, for these affections often give rise to neither pain nor dyspnœa nor any other symptom.

The frequency of inflammation of the heart in rheumatism cannot be stated with numerical accuracy. The results of autopsies are not available for the purpose, because cases, which prove fatal, are more severe than the average; and the auscultatory signs, which afford our best means of detecting cardiac complications during life, are open to sources of fallacy, particularly the difficulty of distinguishing between organic and functional murmurs.

Among forty-five cases of rheumatic fever which ended fatally in Guy's Hospital, and in which there had been no previous chronic disease of the valves, the heart was found to be healthy in eight only, both pericarditis and endocarditis existed in nineteen, pericarditis alone in ten, endocarditis alone in eight.†

Turning to clinical records, we find that Budd put the proportion of cardiac lesions at 48 per cent., Fuller at only 23 per cent. It is remarkable that the same proportion, 23 per cent., was independently arrived at by Lebert at Breslau, and by Wunderlich at Leipzig.

Mr Manser collected 400 cases for the writer's paper in 1875, and Mr Shadwell 100 more for the second edition of the present work. Of these 500 cases from the clinical records of Guy's Hospital, only 221 were free from cardiac murmurs; while in the remaining 279 cases, 110 bruits were

* The "febris miliaris" which occurred as an epidemic on the Continent, and especially in France, throughout the eighteenth century and down to the year 1856, and may very likely have included cases of rheumatic fever, was attended with a red papular or vesicular rash. The name is still used in Italy, but probably for scarlatina, enteric, and other febrile diseases attended by sweating and sudamina. In the English "sweating sickness," of which there were five terrible epidemics between 1485 and 1551 in this country, there seems to have been no such eruption (cf. vol. i, p. 225).

† These figures differ from those which Dr. Sibson gave as the result of an analysis of 326 unselected clinical cases in his elaborate article in the fourth volume of 'Reynolds' System.' According to these, endocarditis was present nearly three times as often as pericarditis. Bouillaud estimated its occurrence as 80 per cent., but that was in patients who were bled and leeched remorselessly.

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believed to be pericardial and 169 endocardial. At St Bartholomew's Hospital, out of Dr Church's 574 cases in which the state of the heart was ascertained, it was affected in 371. Dr Peacock ('St Thos. Hosp. Rep.,' 1873) recorded cardiac murmurs in only a third of his cases.

There is no doubt of the fact that acute inflammation of the heart is sometimes, and especially in children, the sole indication of a rheumatic attack, and that in other cases it is attended with only slight and fugitive articular pains, which may easily escape notice altogether. Nevertheless, as a rule, there is in acute rheumatism a direct relation between the degree of severity of the articular affection and the frequency, as well as the intensity, of cardiac complications. Thus Sibson found that the affection of the joints was severe in only one fourth of those cases in which the heart showed no sign of inflammation, but in two fifths of those in which endocarditis was present, and in three fifths of those in which there was pericarditis, with or without endocarditis.

Age has a very marked influence upon the frequency of inflammation of the heart in rheumatism. The younger the patient, the greater the liability to it. This is strikingly shown by the third table in Dr Church's paper in the 'St Barth. Hos. Rep.,' vol. xxiii, p. 273. The percentage of cardiac affections in successive decades from under 10 to 50 runs thus:—83, 69, 51, 30, 21.

In young female servants, who make up a large proportion of the cases of rheumatism seen in hospital practice, the heart scarcely ever, according to Sibson, fails to show some signs of being attacked. Latham also said that maidservants with rheumatic fever in hospital always get pericarditis.

Older patients escape more often; and here a curious difference is found to exist between men and women. Pericarditis seems to be three times as frequent in men above the age of twenty-five as in women of corresponding age. The reason for this is believed to be that a laborious occupation increases the liability to cardiac complications in rheumatism; most women work less hard than men of the same age, but the girls of the lower classes are very apt to have their strength overtaxed.

It is probable that, taking all cases of acute cardiac implication in rheumatism, only the minority affect the pericardium, either alone or with the endocardium; but this cannot be certainly affirmed because pericarditis is often very transient and leaves no trace behind it. Adherent pericardium is certainly much less commonly found after death than valvular lesions, excluding those which in later life are due to atheroma or chronic sclerosis: and a pericardial rub is also less frequent than an endocardial murmur as estimated by auscultation during life. Judging by the same clinical method, the mitral valve is much more often affected in rheumatic endocarditis than the aortic, and, since the murmur is most frequently apical and systolic, mitral imperfection is more common than mitral stenosis. In fact, when a præsystolic bruit or other signs lead to the diagnosis of the latter lesion we may be almost sure that there have been one or more previous attacks of rheumatic endocarditis.

Of *other complications*, pleurisy, affecting chiefly the left pleura, is most frequent. Lebert speaks of it as occurring in 10 per cent. of all cases; and it would probably be more often diagnosed than it is, if the pain produced by moving rheumatic patients did not interfere with stethoscopic examination of the back.

Bronchitis is only occasionally present; the lungs are often œdematous;

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and, in fatal cases, the microscope may show them to be affected with slight catarrhal pneumonia. Lobar pneumonia is of somewhat rare occurrence, but when present commonly leads to death.

Rheumatic peritonitis and iritis are both very doubtful, and rheumatic meningitis, cerebral or spinal, may be said not to exist.

In one fatal case that occurred at Guy's Hospital the *tonsils* were suppurating, and acute tonsillitis sometimes begins an attack.

Rheumatic nodules.—An effect of rheumatic fever, of both pathological and diagnostic interest, consists in the formation of small, subcutaneous, fibrous nodules, usually but not always in the neighbourhood of joints, often upon prominent points of bone, like the knuckles, the olecranon, the tibia, and the acromion, or on the superior curved line of the occipital bone. They were first described by the late Dr Hillier in his 'Diseases of Children' (1868), and afterwards by Meynet, of Lyons, in 1875, by Rehn in Germany (1878), and by Hirschsprung in Denmark (1881). A full account of them by Dr Barlow and Dr Warner will be found in the 'Transactions of the International Congress of 1881,' vol. iv, p. 116, with twenty-seven cases.*

These nodules are rarely seen in adults, but are frequent in children. They are but very little tender, and seem to disappear soon after the attack of rheumatism is over. They have been found in the pericardium after death, and when numerous on the surface are in Dr Cheadle's experience indicative of severe inflammation of the pericardium, and of a fatal result from that or from endocardial lesions.

The *course* of rheumatism, uninfluenced by treatment, is tedious and uncertain. The pyrexia and pain gradually subside and revive again as a fresh joint is attacked, and thus the attack drags on for several weeks and leaves the patient excessively weakened and anæmic. Even when the heart has escaped, convalescence is slow and often interrupted by relapses. These are less severe and prolonged than the original attack, but may be repeated again and again, until the disease becomes chronic in duration though *still* acute or subacute in symptoms.

Sequelæ are very rare; and when the rheumatic synovitis is once cured, local deformity seldom results. In this particular true rheumatism differs remarkably from gout, and from gonorrhœal synovitis.

So far from protecting against recurrence, one attack of rheumatic fever is a predisposing cause of another. Apart from relapses, the interval is usually from three to five years, and sometimes much longer.

Ætiology.—*Cold* is generally regarded as the most frequent cause of *acute* rheumatism. But this is open to question. Exposure to cold winds, or travelling in winter or in cold climates, or insufficient clothing does not cause it. The only efficient kind of cold is getting wet through. The patient is not infrequently attacked within a few hours after being out for several hours in the rain; but when there has been an interval of a week or even a fortnight since such an occurrence, the causal relation of events is more doubtful. Sometimes the exposure may have been repeated again and again for a considerable time before any harm seems to result; we may either suppose a cumulative action, exerted slowly and from day to day; or *that*

* Numerous cases have since been recorded by Dr Cavafy ('Path. Trans.,' xxxiv, p. 41). Drs Lees, Duckworth, Money, Drewitt, Stephen Mackenzie and Fowler, in the 'Transactions' of the Pathological Society for 1883 and of the Clinical Society for 1883–84. A remarkable case in a woman of thirty-nine is described and figured by Dr G. S. Middleton, of Glasgow, in the 'American Journ. of Med. Sci.,' October, 1887.

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the patient does not suffer until his power of resistance is so diminished by over-fatigue, or in some other way, that he now for the first time gets "a chill." Careful inquiry as to previous exposure to wet and cold has led to a negative statement in probably half of the writer's patients; and there is no question that many, and perhaps the majority, of cases of acute rheumatism are not due to any tangible exposure.

Rheumatic fever is stated to be less common during the months of July, August, and September than at other seasons of the year. Haygarth, who in 1805 first published exact statistical observations of cases of rheumatism (170 in number), found as many of them to have begun in the five cold months (December—April) as in the seven warmer months—not more, perhaps, than an accidental difference. The number of patients admitted into the wards of hospitals in London at different times is liable to wide variations which are not attributable to changes in the weather; for several weeks hardly a case may present itself, whereas afterwards a great many may appear within a few days of one another.

A review of the statistics at Guy's Hospital during ten years collected by Mr Capes, shows that the largest numbers of admissions were in September (158), and November (136); the next in October, January, and April (127 each). There were not so many in May, June, and August (108—112); fewer in December, February, and July (about 100); and fewest in March (69). Dr Archibald Garrod gives a series of similar observations by nine different writers in the fifth chapter of his recent work on 'Rheumatism,' and finds great discrepancy in the results.

The *geographical distribution* of the disease is as yet too imperfectly known to enable one to state positively its bearings upon the question of its relation to climatic conditions. Hirsch gives a large amount of information; but with regard to many of the statements which he cites, it is difficult to tell whether acute rheumatism is meant, to the exclusion of the vague affections which are so often labelled "rheumatic." Thus, after stating that rheumatism is prevalent among the Esquimaux in Iceland, and in Polar regions generally, he concludes that acute rheumatism is a disease mainly of the temperate zones. One striking fact is that in Cornwall and South Devon it is comparatively infrequent. In a dispensary practice during four and a half years in Cornwall, only four cases occurred; and medical men practising in Exeter say that it seldom is seen in that city. The Isle of Wight and Guernsey are said to enjoy a similar degree of immunity from the disease. On the other hand, in the United States "rheumatism" appears to be far more frequent in the Southern than in the North-Eastern States, where the climate is comparatively cold and changeable. It is said to be rare in Canada and in Australia, both dry climates, though very different in temperature. Palgrave found both rheumatism and rheumatic disease of the heart common in the pure, keen, and dry atmosphere of the Nejid in Central Arabia.

Next to cold, muscular *exertion* is, according to some authors, an exciting cause of rheumatism. It not infrequently sets in immediately after a long march, and its great frequency in servant-girls is supposed to be due to their being so often sadly over-worked. Some of the occupations which furnish a large number of cases involve hard muscular work; thus it is frequent in smiths and in carpenters.

There can be no doubt that the members of certain families are especially liable to suffer from rheumatic fever; several children of the same

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parents are often affected by it, and others have chorea. Garrod traced an *hereditary predisposition* in about a quarter of his patients, Fuller in 27 and Chomel in 33 per cent. Among 400 hospital patients, the writer found it in 68, *i. e.* in 17 per cent. In his recent treatise, Dr Archibald Garrod reports that, among 500 cases of patients at St Bartholomew's Hospital who had never had rheumatism, 105 or 21 per cent. gave an account of rheumatic fever in near relatives; while, compared with this general coincidence, he found that, of 500 patients with acute rheumatism themselves, more than 150 (30—35 per cent.) could give a history of the same disease in near relatives.

Whether the disease is more apt to occur in persons of one *complexion* than in those of another is very doubtful. According to Laycock, those of "the rheumatic diathesis" are usually well built and well nourished, the complexion is of a healthy florid tint, the teeth are regular, sound, and firm, the hair is abundant, and the skin greasy and thick. Mr Hutchinson (following Bazin) is convinced that there is a "diathesis" which is termed by him arthritic, and which is common to gout, rheumatism, and arthritis deformans. A very general impression is that most patients with rheumatic fever have light hair and grey eyes. But this applies to all diseases in England, because our population is chiefly xanthochroic.

Rheumatism not infrequently follows *scarlet fever*, usually during the stage of desquamation.* A similar affection sometimes occurs as a sequel of dysentery (p. 235), but probably this is not true rheumatism. The *puerperal* state is also believed to bear a causal relation to rheumatic fever, which is not infrequently developed in women who have been recently delivered, or have miscarried, especially when there has been profuse hæmorrhage.

Sex.—Men are somewhat more liable than women to rheumatic fever, in about the proportion of 4 to 3; a difference which perhaps may be explained by their harder work and greater exposure to weather. This seems to be shown by Dr Goodhart's cases in children below 14, where of 69 patients there were 27 boys to 42 girls.

Among 400 cases at Guy's Hospital the numbers were 223 men to 177 women. Of 654 cases recorded by the Collective Investigation Committee ('Brit. Med. Journ.,' Feb. 25th, 1888), were 375 men, 279 women.

Age.—Rheumatism is a disease of *youth:* a large proportion of first attacks take place in children and in young adults; and it seldom occurs for the first time after the age of fifty. In infants, however, it is very uncommon; only a few cases have been recorded, one at the age of twenty-three days by Widerhofer, another at four weeks by Stäger, and one at ten months by Henoch (all quoted by Senator).

About four-fifths of first attacks occur between 11 and 30. At Guy's Hospital, out of 365 first cases, 22 fell between 5 and 10, 179 between 10 and 20, 118 between 20 and 30, 34 between 30 and 40, and only 12 above 40. Even second and third attacks become less frequent as the patient grows older. The writer has met with one case in a woman of 61, and one in a man of 73.

Putting together 683 cases (whether first or later) from St Bartholomew's Hospital and 620 from Guy's Hospital:—Out of the total 1303, 48 occurred under 10, 521 between 10 and 20, 441 between 20 and 30, 195 between 30 and 40, 69 between 40 and 50, and 30 above 50.

On the whole, we must confess that the causes of rheumatism are

* See papers by Dr Thos. Barlow and by Dr Ashby, 'Brit. Med. Journ.,' Sept. 15, 1883.

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obscure. Practically all that can be said is that those who are young, those who are poor, and those who have suffered from it already, are the most likely to be attacked by the disease.

Theory of rheumatism.—The pathological allies of rheumatic fever are chorea and erythema. Its only known antecedent is scarlatina, its only known consequence disease of the heart.

Difficult as it may be to distinguish it in certain cases from gout, or osteo-arthritis, or even from gonorrhœal synovitis or pyæmia, this is no evidence of the diseases being related, any more than the same or greater difficulty of diagnosis in cases of fracture or dislocation, tubercle or typhoid fever, intestinal obstruction or peritonitis. As for mixed or hybrid forms of disease, not only is their existence unproved, but the notion of a hybrid between two diseases is equally absurd, whether we regard them as produced by a clinical poison, by a local injury, by a spore-bearing microphyte, or as an abstract expression for a constantly recurring combination of symptoms.

The essential nature of the disease is quite unknown. It has been ascribed with equal confidence to diseased humours, to a disturbance of nervous centres or of trophic nerves, to the presence of a bacillus, and to an organic poison in the blood.

Dr Fagge, like other physicians, was attracted to the view, originally suggested by Prout, that the poison of rheumatic fever is *lactic acid.* Analogy, a fallacious guide, argues that rheumatism, like gout, is due to some acid in the blood; but, in spite of its peculiar sour smell, we have seen that the sweat of rheumatism is not more acid than usual. In 1853 Dr B. W. Richardson published a series of experiments upon dogs, which were believed by him to show that the injection of lactic acid into the peritoneal cavity was capable of setting up endocarditis; but eight years later Reyher, in 'Virchow's Archiv,' pointed out that appearances precisely similar are constantly seen in the cardiac valves of healthy cats and dogs. After this the lactic acid theory languished, until, in 1871, Dr Balthazar Foster, of Birmingham, recorded in the 'British Medical Journal' two cases in each of which the administration of this acid (in doses of ♏xv to ♏lxxv), with a view to check diabetes, was followed by the occurrence of painful swellings of the joints; one patient had no fewer than six well-marked attacks, the symptoms of which seem to have resembled those of acute rheumatism. Külz in his 'Beiträge zur Path. u. Ther. d. Diabetes' related a case in which lactic acid set up pains called "rheumatic" in the hip and thigh. It is true that the same drug has been repeatedly administered to other patients and to healthy persons without result; but, as Senator remarks, such negative facts perhaps only prove that a "personal susceptibility" is necessary. Lastly, a plausible hypothesis is that under the influence of cold the lactic acid which is always formed as the result of muscular exertion fails to be destroyed by oxidation, as it should be, and that when so accumulated it acts as an irritant to the joints. Until better evidence than imperfect observation, hypothetical chemistry, and vague conjecture is given, we may defer acceptance of the last of the humoral theories.*

It has been often suggested that rheumatism is a *specific fever*, and

* As ingenious theories on the chemical origin of rheumatism should be mentioned Dr Latham's 'Croonian Lectures' of 1886, and Dr Haig's more recent papers. The inhalation of sewer-gas as a cause of rheumatic fever has been put forward by Dr B. N. Dalton, in a practical paper in the 'British Medical Journal' for 1890.

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within the last few years the discovery of its infective microbe has been repeatedly announced. None of the criteria stated in the first volume (p. 13) have been satisfied; and, apart from the question of a bacterial origin, the natural history of the disease is strikingly unlike that of a specific fever. It is not contagious, it does not run a definite course, and it does not protect against itself.

A relation between *malaria* and rheumatism was a pardonable guess at the beginning of the present century, but there is not the least evidence in its favour as a serious hypothesis.

Prognosis: duration and relapses.—In the great majority of cases an attack of rheumatism when left to itself slowly and gradually improves, the pains diminish, the joints recover, and the fever subsides. This takes place in a variable time, occasionally in a week or ten days, often not until three weeks have elapsed, and sometimes only after six or eight weeks. The duration of rheumatism, however, is now happily so much modified by treatment, and the estimation of its natural length so much affects the judgment we form as to the results of treatment, that this question will be more particularly considered in the following section.

Of all acute diseases, rheumatism is the most prone to relapse. Its relapses are not like those of the disease known as relapsing fever (vol. i, p. 124), an essential part of the process; for in most cases they are absent. But in a large proportion—varying in different times and places and under different modes of treatment from a sixth to a fourth or a third of the whole number—only a few days after the first attack has subsided, a second comes on; this runs a similar though usually a milder and a shorter course, and the heart, if it has escaped before, is seldom affected now. A second relapse is common, and a third and a fourth are sometimes seen. Dr Church has proved that relapses are much more common after second than after first, and still more so after third attacks. The question of how far they can be prevented by regimen and drugs will be presently considered (p. 712), for it is one important object of treatment. There is no doubt that prolonged care and continued treatment during convalescence are influential in preventing relapses. Hence they are comparatively rare among patients who can afford a prolonged period of easy convalescence, while it is one of the disappointments of hospital practice to see a patient, who has been sent out perfectly well after an attack of rheumatism, return to the ward a week or two later with all his symptoms renewed.

The joints are very rarely permanently injured. Occasionally hydrops articuli remains behind, and after many attacks the joints may undergo the changes described in a subsequent chapter as osteo-arthritic. Anæmia is a marked effect, in proportion to the severity of the attack. It is scarcely ever absent, and return of muscular strength is often very slow.

The liability to peri- and endocarditis, with their subsequent effects, has been already considered.

Mortality.—Apart from the ultimate effects of rheumatic diseases of the valves, a fatal result is very rare. When not due to acute pericarditis, or to some accidental cause, death usually results from hyperpyrexia, a remarkable condition to be presently considered, or from pneumonia.

Senator puts the average mortality at from 3 to 3·7 per cent. of those who are attacked, and at Guy's Hospital in the 400 cases collected by the present writer, it was nearly the same, 3·75. So also in the 655 cases of

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the Collective Investigation Report the mortality was 3·3 per cent. But at St. Bartholomew's Hospital Dr Church found only 10 deaths in 693 cases.

The number of deaths in our wards varied widely in different years. From 1855 to 1867 (thirteen years) only *ten* fatal cases altogether appear in the records. From 1868 to 1884 (seventeen years) there were *fifty* fatal cases. The numbers in the several years were as follows:—Two in 1868, three in 1869, five in 1870, two in 1871, seven in 1872, two in 1873, seven in 1874, six in 1875, three in 1876, six in 1877, one in 1878, three in 1879, one in 1880, two in 1881, and none in 1882–3–4.

In three of these *sixty* cases the fatal termination was due to stenosis of the mitral valve, which had resulted from previous attacks of acute rheumatism. In only four cases was there found a severe and recent lesion of the valves as the direct result of the rheumatism.

In eighteen instances death was attributed to *pericarditis*, which was sometimes severe, the heart being covered with lymph, and there being from ten to sixteen ounces of fluid, deeply stained with blood. In several of these cases there was also effusion into one or both of the pleural cavities; twice there was inflammation of the mediastinum; in seven cases the muscular substance of the heart was obviously involved in the inflammatory process. Each of the complications in question may be supposed to have helped in bringing about the patient's death; and it is not improbable that myocarditis was sometimes present when it was not noticed. Although endocarditis existed in eleven of the eighteen cases, it was so slight that it could not be considered to have affected the issue.

In twenty-seven fatal cases of acute rheumatism collected by the writer, death occurred from *hyperpyrexia* with delirium in five, the ages of the patients ranging from twenty-three to thirty-one. One patient died from delirium tremens with acute *pneumonia*, four from pneumonia with cardiac lesions, thirteen from severe pericarditis or valvular disease or both, the immediate cause of death in two being embolism, complicated in both cases by chorea. The remaining three died from accidental causes (diphtheria, enteric fever and epilepsy) which supervened while under treatment for rheumatism.

Rheumatic hyperpyrexia.—That cerebral symptoms sometimes develop themselves in the course of rheumatic fever, and rapidly prove fatal, has long been known. By Sir Thomas Watson and Dr Latham it was thought that they depended upon cardiac complications, and particularly on pericarditis; but in many cases the heart is found free from all signs of inflammation. The existence of meningitis has also been disproved.

In 1867 Dr Ringer related three cases of rheumatic fever in which extremely high temperatures were observed, the thermometer having risen from 104° to 105° to 109·2° or even 110·8° Fahr. Dr Kreuser noticed the same fact in Würtemberg about a year previously. Many similar cases have been since recorded, both in England and abroad; at Guy's Hospital in seven years fourteen patients died of hyperpyrexia in acute rheumatism.

The most probable pathology of this terrible complication is that while the development of the high temperature is itself consequent upon an exhaustion of the heat-regulating centre in the bulb, the cerebral symptoms in their turn result from the action of the heated blood upon the brain.

Unlike the occasional high temperatures observed in severe cases of pneumonia or fever shortly before death, the hyperpyrexia of acute rheumatism may suddenly supervene in an apparently favourable case. One of

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Dr Ringer's patients was supposed to have recovered, and was to leave the hospital the next day, when cerebral symptoms set in, and he died in two hours, with a temperature of 110°.

The first indication of the onset of hyperpyrexia is often that the patient loses his pains and finds that he can move his limbs freely. Unless the temperature falls at the same time, this is a warning of impending danger, and should lead to the use of the thermometer at regular intervals of ten, twenty, or thirty minutes. Another warning symptom is that the skin ceases to perspire.

The cerebral symptoms of rheumatic hyperpyrexia vary in character in different cases. Sometimes the patient becomes drowsy, appears to fall asleep, and so passes into a state of unconsciousness, with contraction of the pupils, which ends in death. Sometimes he grows violently maniacal, jumping out of bed, and fighting with nurses and attendants. Sometimes he is seized with convulsions, or with tonic spasms and opisthotonos. His pulse becomes very rapid, from 140 to 136, and towards the last it may become imperceptible. His breathing is much accelerated, and his face sometimes exhibits the dusky purple flush of congestion, with eyes suffused, and more or less complete cyanosis.

The interval which elapses from the commencement of the hyperpyrexia to the fatal termination is very variable, as is well shown by an analysis of twenty-two cases made by Dr Wilson Fox. In one instance the thermometer rose from 103·5° to 109° in two hours; in two other instances a period of twenty-four hours passed before a similar point was reached. Even when the temperature is 107° or 108° the patient may now and then continue to live for some hours; and at 110° there may still be an interval of from one to two hours before the fatal termination.

Dr Fox thought that hyperpyrexia is more frequent in first attacks of rheumatic fever than in subsequent ones; and subsequent experience has confirmed this. As shown in a valuable report based on 67 cases, rheumatic hyperpyrexia is nearly twice as common in men as in women ('Clinical Trans.,' vol. xv). It very seldom occurs under puberty.

Diagnosis.—Acute rheumatism is, as a rule, very easy to recognise. But there are not a few diseases which may be mistaken for it, and in some exceptional instances it may be impossible to decide.

The author was called into the hospital one Sunday by the house physician to see a girl who had been just admitted for rheumatic fever, and whose temperature was very high. The peculiarity of her case was that she was covered all over with a bright scarlet rash. She had had acute rheumatism on a former occasion, and the real nature of her illness was not suspected until the following day, when the papular eruption of *smallpox* was found upon her. In several instances the pains in the limbs produced by the growth of *multiple sarcomata*, especially about the vertebræ, so as to compress the spinal nerves, have been supposed to be due to acute or to subacute rheumatism (vol. i, p. 83).*

Ulcerative endocarditis sometimes complicates rheumatism. In some cases so regarded, it is permissible to doubt, whether the rheumatism was really present, or whether the endocarditis was not primary.

* In a case of multiple sarcomata of the skin, secondary to a growth in the cæcum, under the writer's care, there were first pains and swelling about the joints which he mistook for rheumatism, and when the sarcomata appeared he supposed them to be purpuric erythema—peliosis rheumatica.

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Affections of the spinal cord in the early stages have been regarded as rheumatic on account of the pains to which they gave rise, until paralysis supervened. But it must not be forgotten that in some spinal cases, and notably in tabes, the joints may actually suffer. The same remark applies also to *syphilis*, the effects of which are not infrequently set down to rheumatism. Lastly, *scorbutus* and *hæmophilia* must be mentioned as diseases with regard to which it is by no means difficult to make a similar blunder.

The diagnosis from an acute attack of *gout* is occasionally difficult when the latter occurs for the first time in a young subject, but such cases are mostly hereditary, and the heart being unaffected would be unusual at that age in rheumatism. The degree of fever, the locality of the arthritis, and, above all, its shifting from joint to joint without leaving traces behind, are the chief additional marks which distinguish rheumatism from gout. The diagnosis from gonorrhœal synovitis and from acute osteo-arthritis will be given hereafter (pp. 719 and 725).

A useful means of diagnosis of doubtful cases in children is afforded by the presence of the "rheumatic nodules" described above (p. 692).

The writer had once a man of about fifty under his care, who appeared to be suffering from rheumatic fever; but soon diarrhœa and then a rose-rash appeared, and the diagnosis was altered to that of enteric fever. He died at an early stage of the disease from perforation, and after death it was clear, from the condition of the joints, the heart, and the intestines, that the two diseases had coexisted.

Perhaps the most common and serious error is mistaking for rheumatic fever one of those forms of *pyæmia* which come particularly before physicians because they are not secondary to a wound or obvious source of suppuration. The most frequent primary lesion is caries of the petrous bone, which may be recognised by otorrhœa, or by fœtor of a fragment of cotton-wool left in the meatus, even when there is no obvious discharge of pus, by perforation of the membrana tympani, and by tenderness over the mastoid process or the jugular vein. Sometimes the pyæmia is due to acute osteomyelitis of a long bone, when the affected limb will be found hot, tender, and œdematous; or acute periostitis with suppuration and secondary synovitis may in like manner simulate rheumatism.

Sometimes, in spite of all care, diagnosis is impossible till after death, and difficult even then. In 1879 Dr Goodhart made an autopsy in the case of a boy aged sixteen, who had been lying for five days in a medical ward in a typhoid condition. He had a systolic murmur; his temperature had been 103·8° on admission, his pulse 132, his respirations 48. He had been attacked with pains and with chilliness five days before his admission. Numerous abscesses were found in the lungs and in the kidneys. Evidently, therefore, the case was one of pyæmia; but it was not until after nearly every other bone had been examined that Dr Goodhart discovered suppuration beneath the periosteum of the lower end of the right fibula. On the tricuspid valve there was a vegetation of the size of a pea, with a little ulcer beneath, which had torn through some of the chordæ.

How closely pyæmia may resemble acute rheumatism is well shown by a case which occurred to Dr Moxon in Guy's Hospital in 1877. A patient was admitted who had nine days before been attacked with headache, sickness, and rigors, and in whom these symptoms were followed by profuse sweating and by pains in the joints. Salicylic acid was prescribed, but the

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temperature rose and delirium set in, so that cold baths were employed on several occasions. The question whether the disease could be pyæmia was formally discussed, and negatived in favour of the diagnosis of acute rheumatism. He made no complaint of the thigh, as being more painful than other parts, when he was being moved into, or out of, the bath. Yet at the autopsy not only was there osteitis of the lower part of the shaft of the right femur and of the adjacent epiphysis, but the bone was denuded of its periosteum, and there was a large collection of pus beneath the muscles. There was also suppuration about the shoulder, and pyæmic abscesses in the lungs and heart.

An important distinction between acute rheumatism and pyæmia is that in rheumatism the pain and swelling fly about, leaving one joint, and after a few hours attacking another. But that this criterion is not perfect is proved by the case of a boy who died of pyæmia in a surgical ward in 1870, and who for a time had flying pains in the joints.

It is worthy of notice that almost all the instances that have occurred at Guy's Hospital within the last few years of pyæmia from osteomyelitis have been in boys or young men between ten and twenty years of age. As a rule, the diagnosis is rendered comparatively easy by the severe constitutional disturbance which is present, by the presence of rigors, and by the skin being dry instead of sweating; the joints, too, show a deeper blush, and are more frequently hot.

Certain eruptions, which are sometimes associated with pyæmia, may simulate rheumatic erythema. Beside the scarlet rash which is comparatively common in surgical wards, and which has now been proved to be really scarlatina, there are cases in which the skin presents appearances of a still more remarkable kind. Thus in 1861 a boy of thirteen was admitted into hospital who had for a week been treated for rheumatic fever, but whose disease was at once recognised to be pyæmia, there being a large abscess of the thigh. Towards the last his body became covered with "a purplish rash, resembling the mottled rash of typhus, partly consisting of petechiæ (probably fleabites), partly of papules, which became vesicular at the apices and slightly scabbed." In another patient, in 1874, pustules are said to have appeared on the back and on the abdomen two days before death. Pustular eruptions are mentioned in two instances of "spontaneous pyæmia," collected by the Pathological Society's Committee in 1879.

In regard to all such cases, one must be careful not to overlook the presence of *glanders*. That disease is itself sometimes mistaken for acute rheumatism at its commencement.

Another class of cases which are likely to be set down to acute rheumatism are those of *pyæmia from gonorrhœa*. In 1872 a young woman in advanced pregnancy was admitted into Guy's Hospital with what was supposed to be rheumatic fever; an hour later she was delivered of a child, which survived for some days; she was now seen to be suffering from pyæmia, and four days afterwards she died. At the autopsy an abscess was found in the subserous tissue near the right ovary, and there were softening thrombi in the adjacent veins; but it was thought that these lesions were themselves secondary, and that the starting-point of the disease was a vaginal discharge from which she had been suffering, and which was probably of a gonorrhœal character.

In ordinary surgical practice it seldom happens that pyæmia is mistaken for acute rheumatism. But sometimes the converse blunder is com-

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mitted, and when rheumatic synovitis appears after an operation or accident, the patient is supposed to have pyæmia.

Rheumatism in children.—In the case of patients under puberty, rheumatic fever assumes somewhat special characters, which may sometimes prevent its recognition. It is very frequent, and often subacute without severe pain or synovitis, and with only moderate fever. Moreover, gout, gonorrhœal synovitis, and osteo-arthritis, are unknown at that age, so that if multiple arthritis with pyrexia is present we have only to consider the possibility of pyæmia or of tubercular disease—pulpy synovitis or caries—affecting more than one joint. The articular inflammation is sometimes so slight, that pain and swelling have to be inquired for. The heart is, however, extremely apt to become affected; there is a bruit to be heard in much more than half, some writers believe in nearly all, cases of rheumatism in children. Hyperpyrexia is very rare. Tonsillitis is common, and erythema is not infrequent. Cases of rheumatic pericarditis or endocarditis, or pleurisy with only slight pyrexia, and no discoverable synovitis, are very rare in adults, but not uncommon in children. Chorea is a frequent complication or sequela. Lastly, rheumatic nodules (p. 691) are far more frequent and numerous than in adults. An hereditary origin is far more often present, or more easily detected, and one from cold or exposure much less frequent.

Treatment.—Formerly the treatment of rheumatism was not satisfactory. A great variety of medicines were prescribed, each of which appeared to be highly successful in some cases, while it utterly failed in others. Some physicians, and especially the late Dr Fuller, of St George's Hospital, recommended the administration of alkalies, in such doses as to maintain an alkaline reaction of the urine; some, following Dr Rees, trusted to lemon-juice. Some gave bark (as Haygarth recommended), or quinine, others the tincture of iron, others colchicum, others ergot, others propylamine (or rather trimethylamine). The late Dr Herbert Davies advocated the employment of blisters to all the affected joints; surrounding the knees, for example, with strips of emplastrum cantharidis three inches wide. It seems to be certain that the blistering plan is often followed by a rapid subsidence of the local inflammation, but there is the disadvantage that when carried out fully it is apt to produce strangury, and (as Senator has shown in vol. lx of 'Virchow's Archiv') it sometimes leads to inflammatory exudation in the urine, which may not only contain flakes when voided, but even coagulate afterwards. Moreover, it may produce local sloughing; and, after all, the local synovitis is not the disease.

It would be difficult to say when physicians first began to entertain the suspicion that the apparent success of various methods of curing acute rheumatism might, after all, be fallacious, and that the result would have been the same if nothing had been done. Sir Thomas Watson used to cite, but without assenting to it, the dictum of Warren, who, when asked what was good for acute rheumatism, replied, "Six weeks." Within the last thirty years careful observations have been systematically made and recorded of cases left to themselves, without active medication. Such an "expectant treatment," as it has been termed, seems to have been first tried by Lebert, who in 1860 published the results in nine patients. A few years later Sir William Gull and Dr Sutton, in the 'Guy's

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Hospital Reports' for 1866, placed on record the details of twenty-five cases.*

From these observations it is clearly apparent that acute rheumatism is altogether unlike those among the specific fevers which have a fixed or definite duration. Among Sir William Gull's patients the length of time during which active symptoms continued, including that passed before admission as well as subsequently, varied from nine to thirty-four days. On striking an average, the course of the disease, as measured by the period at which there is freedom both from pyrexia and from pain, is much shorter than that named by Dr Warren. Gull and Sutton made it nineteen days; Lebert gave sixteen days as the average time for marked improvement, twenty-two days as that for convalescence.

In attempting, however, to compare cases of acute rheumatism left to themselves with those submitted to treatment by drugs, there is a fundamental difficulty. Strictly speaking, those cases only are comparable, in which the treatment was begun on the very day on which the patient was taken ill. But to include only such cases would be not only to limit the field of observation almost entirely to private practice, but also to ensure that the cases accepted should be of far more than average severity, since only patients seized with violent symptoms are likely to seek medical advice at once. It is therefore impossible to reject cases which have come under treatment at varying periods of the disease. But now arises the question whether one should take into account the time which the disease has lasted before the patient is seen. It is clear that if treatment is effective the fact must be brought out most strikingly when this time before treatment is left out of consideration; on the other hand, if it was ineffectual, we should take the whole duration of the cases as a basis for comparison.

It is indeed easy to prepare both sets of figures, as was done by Gull and Sutton; and, in dealing with average results, no serious error is likely to arise whichever set be adopted—provided the cases are numerous, so that each group may contain its proportion of patients brought under treatment at an early stage, and of those in whom the disease was already far advanced.

But there remains an insurmountable objection to the employment of averages, which invalidates the comparisons made by Gull and Sutton of the results obtained by the "expectant" with those by other methods. The objection is that some cases of acute rheumatism run a very protracted course, which, instead of terminating in a few weeks, is measured by months. One cannot be surprised that no such instances appear to be found in Gull's and Sutton's lists; for it would be scarcely practicable to keep the patients week after week under observation without attempting something for their

* It is, however, wrong to consider the "expectant" treatment carried out by Dr Gull at Guy's Hospital as doing nothing. It was the same rational treatment used in cases of pneumonia, of enteric or scarlet fever, and of fractured limbs. The patients were kept perfectly undisturbed, no strangers were allowed to approach their beds, they were screened from draughts, and the light was shaded. Their joints were covered with cotton wool dipped in laudanum, and protected from pressure by cradles. Except under very special circumstances, no laxative was allowed. They were given an effervescing julep, or any cooling drink preferred, and a grain of opium was administered every night, or oftener if the pain was severe. Under this treatment, if there were few brilliant recoveries, there was on the whole far less pain and distress, a shorter average course, less cardiac disturbance, and a more safe and rapid convalescence, than under the antiphlogistic treatment of Bouillaud, the lemon-juice of Dr Rees, the alkaline treatment of Dr Barlow (each of which the writer had the opportunity of comparing with Dr Gull's), or any other plan which was in use before the introduction of salicin.

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relief; and afterwards in tabulating results, the very fact of treatment having been adopted would be sufficient to exclude them. But when one is dealing with a series of cases submitted to any particular treatment, it becomes impossible to reject the cases in question, some of which may perhaps have been discharged from hospital still uncured; and when an average is taken, a few such cases would swamp the rest in which treatment may have been successful.

TABLE I.—*Natural Course of Twenty-four Cases.*

Duration of Symptoms while in Hospital.	Number of Cases.	Duration of Symptoms before Admission.	Average of the same.
3 days	1	21 days	Average 21 days.
4 days	1	5 days	„ 5 days.
5 days	1	Uncertain	—
6 days	2	7 days, 5 days	„ 6 days.
7 days	3	3 days, 3 days, 14 days	„ 6·3 days.
8 days	1	5 days	„ 5 days.
9 days	1	9 days	„ 9 days.
10 days	2	8 days, 5 days	„ 6·5 days.
11 days	3	8 days, 5 days, 5 days	„ 6 days.
12 days	2	6 days, 10 days	„ 8 days.
13 days	1	6 days	„ 6 days.
14 days	1	14 days	„ 14 days.
16 days	2	12 days, 6 days	„ 9 days.
18 days	1	12 days	„ 12 days.
21 days	1	4 days	„ 4 days.
27 days	1	7 days	„ 7 days.
Duration before admission of 23 cases in which it could be determined—180 days; giving an average of			„ 7·8 days.

It appears that the only fair way of using Gull's and Sutton's cases is to tabulate the length of time that symptoms lasted after admission: the list is then as follows, a fatal case being excluded (see Table I).

It is, of course, to be wished that the number of cases had been larger; but from the regularity with which they are distributed over different parts of the column one may perhaps conclude that the result represents an average. A point which comes out clearly from the last column is that a comparatively long duration of the disease after admission is by no means peculiar to patients admitted at a very early period of their illness. On the other hand, it may be noticed that the case which subsided most rapidly while in the hospital had already been running on for three weeks; and this was not possibly a mere accident, for on tabulating ten other cases in which rapid recovery took place without treatment (or under ineffectual treatment), we find that the average duration of the disease before admission was 13·7 days.

Let us now compare the natural course of the disease with the results of three different methods of treating acute rheumatism, as recorded by their several advocates (see Table II). It seems clear from these figures that no striking success can be claimed for any of the three plans of treatment. The most favourable seems to have been the alkaline method, but even under this the cases in which the symptoms subsided within five days amount to only eighteen out of fifty-one, or less than 36 per cent.

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TABLE II.—*Results of Treatment by Lemon-juice, Alkalies, and Blisters.*

Duration of Symptoms after Commencement of Treatment.	Lemon-juice. Dr Owen Rees ('Guy's Hosp. Rep.,' xii).	Bicarbonate of potass. Dr Garrod ('Med.-Chir. Trans.,' xxxviii).	Free Blistering. Dr Davies ('Lond. Hosp. Rep.,' i).
1 day	1	—	—
2 days	—	1	—
3 days	—	3	—
4 days	1	8	—
5 days	—	6	1
6 days	1	6	2
7 days	2	7	1
8 days	—	5	2
9 days	1	9	1
10 days	2	—	—
11 days	—	2	—
12 days	—	4	—
13 days	—	—	1
14 days	1	Doubtful	5
Total 73 cases	9 cases	51 cases	13 cases

Salicin.—Happily, it has now been conclusively shown that the duration of rheumatism may be greatly shortened by the administration of salicylic acid or its alkaline salts or salicin. It was in January, 1876, that Dr Stricker, assistant in Traube's Clinic in Berlin, drew attention to the use of salicylic acid in rheumatism. It had been used there for some months, and the same medicine had been employed at Basle by Buss. Salicin was originally advocated by Dr T. J. Maclagan, in March, 1876, in the 'Lancet.' He had first prescribed it in 1874.*

After a period of reasonable scepticism and trial, the salicyl treatment became firmly established in this country and in Germany, and was introduced into France by Professor Sée. The immense majority of physicians in Europe and America now use it.

Salicylic acid, as originally used in Germany, is a very unsuitable form of the remedy. It is insoluble, bulky, unpleasant, and irritating. It is now always neutralised by ammonia, potash, or soda, and the soda salt is the one generally used. It is probably less depressing to the heart than salicylate of potass, but it is often well to add ammonia or alcohol so as to obviate this result. Salicin is an agreeable remedy, and now scarcely, if at all, more expensive than the salicylates. It is also less irritating to the stomach and bowels, and milder in its general effects. But it sometimes fails when other salicyl-compounds succeed, and therefore is best used as a substitute for sodium-salicylate when its effects are found to be undesirable.

The author took pains to tabulate not only his own experience at Guy's Hospital, but also that of all his colleagues, in the treatment of acute rheumatism with salicin or salicylates from the spring of 1876 to the end of 1880; and obtained the following results (see Table III).

These figures obviously compare very favourably with those given in Table I as the result in Gull's and Sutton's cases. The symptoms were arrested within five days in no fewer than 180 of our 355 patients, at which period only three of their twenty-four patients had lost their symptoms.

* Among the Hottentots and the Boers of South Africa willow tea has, according to Mr F. Ensor, long been a traditional remedy for rheumatism.

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TABLE III.—*Results of Treatment by Salicin or Salicylates at Guy's Hospital.*

Period at which Patient became free from Pyrexia, as well as articular Pains, reckoned from beginning of Treatment.	Number of Cases.	Number of Cases in which Relapses occurred.		
		1 Relapse.	2 Relapses.	More than 2 Relapses.
1 day	7	2	—	—
2 days	41	8	1	1
3 days	40	11	4	—
4 days	42	10	3	1 (six relapses)
5 days	50	17	7	3 (in 1, five relapses)
6 days	23	4	2	—
7 days	21	6	1	—
8 days	20	9	2	—
9 days	13	7	3	1
10 days	14	3	2	1
11 days	14	2	1	—
12 days	6	2	1	—
13 days	5	2	—	—
14 days	6	0	—	—
15 days	4	2	—	—
16 days	2	1	—	—
17 days	2	0	—	—
18 days	3	1	—	—
19 days	0	—	—	—
20 days	0	—	—	—
21 days	2	—	—	—
22 days	1	—	—	—
23 days	3	2	1	—
24 days	2	—	—	—
25 days	1	—	—	—
26 days	2	—	—	—
27 days	0	—	—	—
28 days	2	1	—	—
29 days	3	2	—	—
30 days	1	—	—	—
32 days	1	—	—	—
35 days	1	—	—	—
36 days	1	—	—	—
37 days	1	—	—	—
41 days	1	—	—	—
49 days	1	1	—	—
55 days	1	—	—	—
60 days	1	—	—	—
Indeterminate	15	—	—	—
Fatal cases	2	—	—	—
Totals	355	93	28	7

It must be added that the rapidity with which relief is afforded to the articular pains is really far greater than appears in the table. In many of the cases which are set down as relieved at the end of five or six or seven days, the patient within two or three days was almost without pain, or was almost free both from pyrexia and from pain. It is no uncommon thing for the patient to be conscious of experiencing great relief from the first two or three doses of the medicine; and house physicians have repeatedly remarked the striking contrast, even on the first night after admission, between patients treated with salicylic acid or salicin and those to whom no medicine had as yet been administered; the former lie quiet, even if they

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do not sleep; the latter often cry out during the whole night. Moreover, in many of the cases placed lower down in the list, the drug was given for a few days, or for twenty hours only, and was then withdrawn; or the doses administered were probably too small to arrest the disease; for in the table every case has been included in which as much as sixty grains of salicylic acid or of salicin was given in the twenty-four hours. With a single exception Table III is drawn up so as to place the facts in the light more adverse to the success of the treatment.*

Relapses.—The exception just alluded to has reference to the relapses which occurred in ninety-three of the 355 cases. This list of ninety-three does not include certain instances in which during convalescence (perhaps when the patient first got up) pain returned for a day or two in a single joint, or in which without pain the temperature rose once or twice to a point between 99° and 100°. Such occurrences were not infrequent, but as no treatment was required, and as recovery was in no way retarded, the author left them out of consideration. The decided relapses more or less closely resembled primary attacks of rheumatism, so that the patients had to be kept in bed and on low diet. In one case a relapse lasted twenty-one days; but the average duration of thirty-one relapses (some treated, some left to nature) was only between five and six days.

Now, there are two ways of looking at the relapses of acute rheumatism. One is to regard them as continuations of the original illness, and to suppose that it has been interrupted and postponed, but not really cut short by the administration of the remedy. This view especially applies to cases in which the symptoms return very soon after the discontinuance of treatment. Thus, in four instances the disease reappeared within about twenty-four hours, and in nine others before a week had elapsed. In one case the urine was tested with perchloride of iron for several days after the salicylate of soda had been left off, and the purple reaction indicative of the presence of a salicyl-compound was obtained as late as the eighth day; it is therefore possible to set down all the thirteen cases as examples of recrudescence of the primary attack. But on the other hand there were six cases in each of which the relapse began when a period of from three weeks to two months had passed after the subsidence of the primary attack, and when salicin had long been discontinued. Such cases must be looked at in the same light as those which relapse when no treatment at all has been adopted; this happens not infrequently, though in what exact proportion of cases it is not possible at present to state. It does not seem likely that relapses are more apt to occur when salicin or salicylate of soda has been given than when the disease has been treated in other ways, or left to run its own course; although those observers who consider salicylic acid a very depressing agent would perhaps believe so. In twenty-eight of the 355 cases treated with salicin or salicylic acid at Guy's Hospital the original attack was followed by two relapses, in five of them by three, in one by

* Among other statistics of the effect of salicylates on acute rheumatism, the reader is referred to an elaborate analysis of 210 cases at St George's Hospital by Dr Isambard Owen ('Lancet,' 1881), to the 158 cases reported in the 'Lancet' for 1879 by Mr R. H. Lucas, of Bury St Edmunds, and to the results of 536 cases reported in the 'Brit. Med. Journ.' for 1888, vol. i, p. 395. See also Dr Bristowe's paper read at Cardiff in 1885, and the discussion which followed ('Brit. Med. Journ.,' August 22nd).

Dr Hermann Weber's early paper in the 'Clinical Transactions' for 1877 is of interest from its account of the introduction and tentative use of the new remedy (x, 63).

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four, and in one by six. It may easily be imagined that such patients spent many months in the hospital wards. It is probable that the frequency of relapses is greatly diminished by systematically continuing the administration of moderate doses of salicylate, or of salicin, until after the lapse of several weeks.

Doses.—The dose of salicylate of soda which is adequate to arrest acute rheumatism with rapidity appears, as a rule, to be about twenty grains, given at intervals of two or three hours; but sometimes a larger quantity is required. One patient of the author's took twenty grains of salicylate of soda every two hours without any marked result for two or three days, but the disease at once yielded when thirty grains were given. On the other hand, there are cases in which a dose of ten grains, repeated every six hours, seems to be effectual.

Salicylate of soda may be dissolved in aqua menthæ piperitæ, or in aqua carui. Or the acid may be dissolved in solution of citrate or of acetate of ammonia (the proportion being gr. xx of acid to ʒiij of liq. amm. acet.), and sweetened with extract of liquorice.

It is generally necessary to use a larger quantity of salicin; Dr Maclagan recommends that from twenty to forty grains should at first be given every hour. It may be prescribed as a powder stirred up in cold water, or twenty grains may be dissolved in an ounce of warm water.

Influence on the heart.—As regards the possible influence of salicylic acid in preventing the development of cardiac complications in acute rheumatism, it is very difficult to make any definite statement. One cannot forget that of each new method of treatment introduced—even of Dr Davies' treatment by local blistering—it has been asserted that the liability to inflammation of the heart is thereby lessened; and Gull and Sutton showed that in cases in which the organ is healthy at the time of the admission of the patients into hospital, it seldom becomes subsequently attacked.

Still, one may reasonably expect that any remedy which possesses the power of arresting acute rheumatism, so that after its administration fresh joints no longer become affected, must also hinder the development of what is believed to be an analogous inflammation in, and around, the heart. And although in sixty-nine of our 355 cases, auscultation revealed some changes in the character of the cardiac sounds while the patient was in the hospital, there was hardly one in which there was reason to believe that pericarditis set in at a time when the action of the remedy was fully established.

On the other hand, it would certainly seem that salicylic acid has no power of controlling or arresting the cardiac complications of acute rheumatism when they have once developed themselves.*

Drawbacks.—In certain cases the administration of salicylates is attended with inconveniences, and sometimes with symptoms which are alarming. A not infrequent effect is nausea and *vomiting*, accompanied with pain at the epigastrium. But this is seldom so severe as to make a change of treatment necessary.

Another effect is *enfeeblement of the heart's action.* At Guy's Hospital this has rarely attracted attention; in a few instances it is noted that the pulse became weak, and sometimes that it was irregular or intermittent; in

* Dr Church, comparing his cases under salicylic treatment with those of the late Dr Latham at the same hospital, long before its introduction, believes that the liability to endocarditis is much the same, but that pericarditis is less frequent, and pleurisy and pneumonia far less common and far less severe.

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one case it fell, after nine days, to fifty-two beats in the minute; in two the first sound of the heart became inaudible, and the heart's impulse could no longer be felt. The administration of stimulants has, however, been very seldom deemed necessary. On the other hand, Dr Greenhow ('Clin. Trans.,' 1880) says that in his patients, who were treated with salicylic acid, "more or less weakening of the pulse, requiring the free administration of brandy, occurred in nearly every case. This was accompanied by great weakening of the impulse of the heart, and in ten cases by almost complete obliteration of the first sound." Dr Goodhart recorded a case in the same volume in which sudden death in the night took place, probably as the result of failure of the heart, for the pulse had been rising in frequency. He was disposed to attribute this result to the administration of salicylic acid, but only sixty grains in all had been given, and none of the known effects of the drug were observed. At the autopsy early pericarditis was found.

Far more obvious than the cardiac are the *cerebral symptoms* to which salicylates (but very rarely salicin) sometimes give rise. Deafness is a very frequent and an early effect of the remedy. It is often accompanied with a sensation of giddiness, and with noises in the ears, which are described by the patients as buzzing or ringing, or are compared with the noise of a train or the rushing of water. In some cases there is headache, which may be intense; or patients become delirious, screaming and struggling to get out of bed. It often happens that, when such effects are produced, the patient has already lost his pains, and that his temperature has fallen to 99° or to 100°; the latter circumstance distinguishes the cerebral symptoms due to salicylic acid from those of hyperpyrexia. The face may be deeply flushed and bathed in perspiration, and in one case the delirium is reported to have been attended with "typhoid symptoms, so that the prognosis for a time was grave." As a rule the patient becomes rational in a few hours, or in a day or two, after the medicine is discontinued. A fatal result has very rarely followed, and in these exceptional cases we have usually found some other cause after death. The drug has often been resumed after a few days' interval without further ill-effects; but sometimes delirium has set in on successive occasions.

Another occasional effect, whether of salicin or of salicylic acid, is *epistaxis*. It was particularly noticed by Dr Greenhow, and has occurred in many cases treated at Guy's Hospital. It often recurs several times, but is not attended with evil consequences, except that it must no doubt tend to increase anæmia, and so to retard convalescence.

Dr L. E. Shaw has recorded in the 'Guy's Hospital Reports' (vol. xliv, p. 125) three cases of severe *hæmorrhage* occurring during the administration of salicylate of soda for acute rheumatism. In the first patient, a boy aged fifteen, taking a scruple of the drug every three hours, delirium appeared on the fourth day (eleventh of the disease), with epistaxis and retinal hæmorrhage. He recovered, but the other two cases proved fatal. In one, a wardmaid aged twenty-one, after taking the same dose of salicylate every two hours, became delirious and passed blood in her urine. After death there was no cardiac lesion found, and the kidneys were normal, but the renal pelvis and the bladder were covered by ecchymoses. In the third patient, a woman aged twenty-six, taking the same gr. xx dose every three hours, for a severe febrile attack of doubtful nature, delirium and hæmaturia appeared, and after death, beside lesions due to latent enteric fever, a precisely similar condition of kidneys and bladder was found.

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Taking the cases of rheumatic fever which occurred in our wards in two years (1881 and 1886), as specimens, Dr Shaw found that in these 174 cases, all treated by salicylate of soda, except a few by salicin, and in nearly the same doses, there were more or less toxic effects in no fewer than 111. Of these, deafness was present in sixty-one cases, delirium in thirty-three; headache, vomiting, and singing in the ears in about the same number, a slow or irregular pulse in thirteen, epistaxis in eleven, and hæmaturia in the two cases above quoted.

Some have supposed that the salicylic treatment causes *anæmia*, and leaves the patient exhausted, so that he regains health and strength more slowly than if the disease had been left to run its natural course. We may admit that the stay in hospital of cases treated with salicylic acid is little if at all shorter than it used to be before the remedy was used. But it must be remembered that for a long time after active symptoms have ceased one keeps the patient on low diet and confines him strictly to bed, for fear of the occurrence of a relapse.

It only remains to consider the facts which led Dr Greenhow to express, on the whole, an opinion unfavourable to the treatment of rheumatism by salicyl-compounds ('Clin. Trans.,' vol. xiii, pp. 244—346). On throwing his sixty cases (ten treated by salicin and fifty by salicylate of soda) into a form to be compared with the preceding table, we obtain the following results (Table IV).

TABLE IV.—*Dr Greenhow's Results with Salicylic Treatment.*

Period at which Patient became free, both from Pyrexia and Joint-Pains, dating from the commencement of Treatment.	Number of Cases.	Number of Cases in which Relapses occurred.
1 day	5	2 (in 1, two relapses; in 1, three).
2 days	11	3 (in 1, two relapses).
3 days	14	6 (in 2, two relapses; in 2, four).
4 days	5	3 (in 1, two relapses).
5 days	1	1 (two relapses).
6 days	1	1
7 days	1	—
8 days	2	—
12 days	1	—
14 days	1	1
18 days	2	1
20 days	1	—
22 days	1	—
Of indefinite duration	13 (Cases 1, 2, 4, 5, 6, 7, 9, 17, 29, 31, 38, 40, and No. 10 salicin)	3
Affording no evidence as to treatment	2 (Cases 26 and 32)	—
Totals	60	21

These results do not seem to be unsatisfactory. In most of the cases which are set down as of indefinite duration, it is doubtful whether the value of the medicine was fully tested; either too small a dose was given (Cases 2, 4, 6, 9, 38, 40) or the persistence of pyrexia may be fairly

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attributed to the presence of pericarditis (Cases 1, 5, 7, and the last of the salicin series). Of the remaining two cases (29, 31), in one the articular pains, and in the other the pyrexia rapidly disappeared.*

Treatment of hyperpyrexia.—The discovery that an excessively high temperature is the real cause of the supervention of dangerous cerebral symptoms in rheumatism was soon followed by the employment of active antipyretic treatment. The administration of salicylic acid or of quinine fails in serious cases; and the newer drugs, as antifebrin and antipyrin, described in vol. i, p. 205, are too transient in their effects and too depressing in their action on the heart, to be either efficient or safe. The only satisfactory treatment is by the direct application of cold.

The first instance in which this treatment brought about recovery from hyperpyrexia in rheumatism seems to have been recorded by Dr Meding in the 'Arch. f. Heilkunde' for 1870; the temperature was 108·6°; the means employed were cold affusion and enemata of iced water. In the following year Dr Wilson Fox published two cases which drew the attention of the whole profession in this country. The first occurred in a woman, aged forty-nine, who was in the fourteenth day of her illness, and who had been five days in University College Hospital, when her temperature began to rise quickly. At 3 p.m. it was 105°; at 6, 106·4°; at 8.5, 107·1°; at 9.15, 108·4°; at 9.50, 109·1°. She was then completely unconscious, her pulse was imperceptible, her face cyanotic, and she appeared to be drawing the few last gasping respirations which commonly precede death. There had been delay in preparing a bath, into which, at a temperature of 96°, Dr Fox had intended to put her when her temperature reached 107°. However, she was lifted into it at 9.50, and five minutes later the temperature in the rectum was found to be 110°. With admirable decision, Dr Fox sent for some ice; two large lumps were placed, one on her chest, and the other on her abdomen; a bag filled with ice was tied down the length of her spine; two assistants baled the warmed water out of the bath, and two others poured iced water over her as fast as the pails could be filled. The temperature in the rectum gradually fell until at 10.25 it was 106·2°. The pulse now became perceptible, and some slight signs of consciousness were manifested. At 10.35 the temperature in the rectum was 103·6°, and she was removed from the bath. At 10.55 the temperature in the rectum was 100·6°; and she was able to speak. The bath had to be repeated on the following morning, but she finally recovered.

Dr Fox's second case was that of a man, aged thirty-six, in whom on the sixth day after his admission (the seventeenth of his disease) the

* Dr Greenhow had laid down the rule that "no patient should be put on the treatment with salicin or with salicylic acid until he had been from twenty-four to thirty-six hours in the wards, and then only if it seemed clear that the illness was running an acute course." His object in giving these instructions was to exclude such cases as would, without any medicine, "improve rapidly after admission into the hospital, and become convalescent in three or four days." His plan, however, was not always strictly carried out, and the consequence is that he dismisses as valueless no fewer than twelve cases in which the subsidence of symptoms under treatment was most rapid. But surely there is no evidence that so large a proportion as this (in addition to an indefinite number of other cases withdrawn from treatment on the ground of their mildness) would get well in from one to four days without medicine. Probably, therefore, Dr Greenhow did in reality obtain good results from salicylic acid and salicin, but in his anxiety to weigh strictly the therapeutical claims of these drugs he did them less than justice. That the sixty cases which he submitted to treatment were of more than average severity, is supported by the fact that in about twenty-five of them signs of pericarditis were discovered on admission, or within a day or two afterwards.—C. H. F.

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temperature rose to 107°, having before been always below 104·5°. He showed signs of pericardial effusion, and also of inflammation at the bases of both lungs; he coughed and expectorated thin mucus stained with blood. This did not prevent Dr Fox from having him placed in a bath at 89° for twenty-five minutes, during which time it was cooled down to 66°. The rectal temperature fell from 107·3° to 103·1°; and after removal from the bath became normal. He ultimately got well, after eight baths in all ('Lancet,' 1871, vol. i, pp. 213 *et seqq.*).

During the time which has passed since Dr Fox recorded his cases, the treatment of hyperpyrexia by cold has been repeatedly adopted, and with very satisfactory results. A striking instance occurred in the person of one of the pupils of Guy's Hospital, who in 1875 had a very severe attack of acute rheumatism, during the course of which his temperature on twenty-six occasions, from the ninth to the twenty-fifth day of March, rose to a point between 105° and 107·2°, and was each time brought down by immersion in an iced bath. He recovered, and is now engaged in medical practice. Full particulars of this case may be found in a paper in the 'Liverpool Medical Reports' for 1876, by Dr F. T. Paul, who was house physician at Guy's Hospital at the time, and who carried out the treatment ordered with unwearied patience and determination.

Unfortunately, even when the bath is perfectly successful in lowering the patient's temperature, it does not always restore his consciousness, still less save his life. Thus Dr Paul records the case of a man aged thirty-two, one of the porters in Guy's Hospital, who after a week's illness with rheumatic fever, became extremely delirious and then comatose, and was found at 9 p.m. with a temperature of 108·8° in the axilla. As he lived out of the hospital, there was a delay of at least an hour before a bath could be procured. When he was put into it his temperature was 110·9°, and he was violently purged. The bath was at 90°, and he was kept in it for thirty-five minutes, during which time it was reduced to 66°, by cold water. His temperature on removal was 106·6°; he was still perfectly insensible, with contracted pupils and with noisy and rapid breathing. Half an hour later the temperature in the rectum was 101·3°, and an hour after the bath it was 99·3°. Subsequently it rose slightly, but it never reached 103°. He died in the afternoon of the following day, the only change being that the contracted state of the pupils changed to wide dilatation.

Another instance, which may also be found in Dr Paul's paper, is that of a woman who died after having had twelve baths during a period of nine days. For the last two or three days mucous râles were audible widely over the chest; but at the autopsy there was only a little bronchopneumonia at the bases of the lungs, and some mucus in the tubes. We have had five other cases at Guy's Hospital, which ended fatally, notwithstanding that the hyperpyrexia had been overcome by baths; in only one of them did the *post-mortem* examination reveal an adequate cause of death in severe pleurisy with pericarditis. Such patients seem generally to sink by failure of the circulation. Indeed, in both of Dr Wilson Fox's successful cases it was deemed necessary to give large quantities of brandy after the baths, and also to apply hot bottles to the feet and warmth to the back; his first patient took six ounces of brandy within an hour.

In all probability the best method of averting collapse after hyperpyrexia is to have recourse to a bath early, before the heart and the tissues generally have been too much damaged by the heat. When the bath is too long

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delayed there is always some risk of death during immersion, a mishap which has twice occurred at Guy's Hospital, once in 1874, and again in 1877. When the temperature is rising for the first time to a dangerous height, it should not be allowed to reach 106° before the bath is used. The necessary preparations should be made as soon as the thermometer indicates 104·5°, and if convulsions occur the patient should be immersed at once. Subsequently, the bath should be repeated as often as the temperature rises to 105·5°. It is best to let the water have a temperature of 90° at the time when the patient is immersed; if it be much colder than this he is likely to shiver and complain; whereas when it is from 90° to 100° he often finds it exceedingly pleasant, so that he will afterwards beg to have the bath repeated. He should be lowered into the water upon a sheet. As soon as this has been done, the temperature of the bath should be reduced to 75°, or even to a still lower point, by the addition of ice, which is more convenient than cold water because it occupies less space, so that no baling out is required. The patient should not be left in the water after the temperature in his rectum has reached 102°, as it will continue to fall after his removal. When he has been lifted back upon the bed, he should be lightly covered with a blanket and allowed to sleep.

The milder methods of employing cold, which were described in the treatment of enteric fever (vol. i, p. 204), sponging, wet packing, &c., are insufficient to meet the unusual danger of excessive temperature in rheumatism. The only efficient substitute for the cold bath, if circumstances make its administration impossible, is rubbing the patient's body with ice.

Treatment during convalescence.—The treatment of rheumatism, apart from the special necessities of hyperpyrexia or other complications, is much simplified by the good effect of the salicyl treatment. Relief is so soon obtained that in most cases we need only guard our patient from a relapse.

With this object, our duty is, first, to keep the patient in bed for many days after the fever has left him and his joints are free from pain; and secondly, to feed him still on strictly low diet, and only when his temperature is normal on farinaceous food. Many physicians believe beef-tea to be injurious, and undoubtedly no meat nor even fish should be allowed for a week, or, better, a fortnight after convalescence is established. Nor should the patient be on any account allowed to leave his bed until the same period has elapsed. This rigorous system is often difficult to carry out, but one has rarely been tempted to relax it by the entreaties of the patient without regretting one's compliance. Moreover, in order to prevent a relapse, salicylates in less frequent doses should be continued for three weeks or even longer. When convalescence is well established, and before the specific drug is left off, it is usually well to administer tincture of steel to combat the anæmia which usually follows rheumatism, but it is probably injurious if given before a week or ten days of freedom from pain and fever have elapsed.

The only exception to the above rules which should be allowed is in the case of patients who have suffered repeatedly from the disease, and whose hearts are already damaged beyond hope of repair. The pyrexia in these cases is often moderate, and the pain not severe; but relapses are frequent, and the cardiac symptoms are more important than those of the rheumatism. For such patients strong soups, eggs, and wine or brandy are

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often harmless and useful, while steel and digitalis may be given with benefit to the heart and with no apparent ill-effect on the rheumatism.

CHRONIC RHEUMATISM.—Although this phrase is in common use for a very frequent disorder, there is reason to doubt whether rheumatism, in the only exact sense of the term, is ever a chronic disease.

Formerly any ache or pain was "explained" as the result of a *distillation* or *catarrh* of cold, moist, peccant humours, and such a *rheum* might affect one part as much as another. In France it appears that medical usage in the seventeenth and eighteenth centuries applied the term *rhumatisme* particularly to pains in the limbs rather than in the trunk or head, and in the bones and muscles rather than in the joints, while the nomenclature of the classical writers was preserved in calling all articular pains, *i. e.* all cases of arthritis, *podagra* or gout. In England, from the introduction of the term rheumatism into medical literature by Sydenham, it has been chiefly restricted to inflammation of the joints, although the phrases articular rheumatism and rheumatic fever show with how little precision the word was used.

At the present time, in all schools of medicine, rheumatism is recognised, in its most characteristic and typical form, as the acute febrile disease, which has been described in the preceding pages. It is very desirable to restrict the use of the word to this, its only exact and definite sense. By "rheumatism" we should always mean the disease which appears as acute multiple synovitis with fever, and "rheumatic" should refer to this disease alone.

Chronic rheumatism, then, ought to mean a chronic arthritis of the same pathology as the acute outbreaks of rheumatic fever. Such a disease, we may affirm, does not exist.

Occasionally, as above stated, attacks of rheumatism relapse and recur so often, that the intervals are shorter than the paroxysms, while the duration and severity of the latter become less and less. Such successive subacute attacks are most often seen in young persons, and accompanied by cardiac lesions; they are often rebellious to treatment by salicylates and are more benefited by opiates and by steel. But their origin is always in an acute attack; there is always more or less febrile movement, while the age of the patient, the aspect of the case, and, above all, the cardiac implication, show that the case is one of genuine rheumatism.

Occasionally when rheumatic fever has occurred frequently and severely, and particularly when the arthritis has lingered longer than usual in a particular joint, there may ensue *hydrops articuli*, and this will run a more or less chronic course. The articulations most liable to this sequela of rheumatic fever are the knee and the wrist; still more rarely, under the same conditions, some distortion of the affected joint may result. This is most likely to happen after a long succession of subacute attacks as above described, and the fingers are the parts more likely to suffer. Lastly, there is no reasonable doubt that some cases of the disease, to be described in a subsequent chapter as osteo-arthritis, originate in unusually severe or unusually protracted synovitis of genuine rheumatic character.

But, however they may end—and all these chronic affections are rare and exceptional, not ordinary and frequent results of rheumatic fever—each of them starts with a typical acute attack of genuine rheumatism, and never by an insidious, non-febrile, and ingravescent disorder.

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Muscular rheumatism.—This has been considered a variety of chronic rheumatism, but here there is no articular inflammation at all. The disease, *i. e.* the pain—for there is no other symptom—is in the muscles. At one time this was, as we have seen, the type of "rheumatism;" then the patient's feelings and the physician's hypotheses made up the whole of pathology. But now that the Galenical or humoral theory is exploded, the disorder in question, having no morbid anatomy, no ascertained chemistry, and no decided ætiology or clinical alliances, must find a new name; and that of *myalgia* is distinctive, convenient, and affirms nothing beyond the fact of pain and of its localisation.

Myalgia has nothing pathologically in common with true rheumatism. We do not know whether its exact seat is in the muscular fibres themselves or in the perimysium and fibrous sheaths. Apparently it is not localised in tendons or in ligaments, for the pain is more often present in the thick fleshy masses of the neck and loins than in the tendinous muscles of the limbs. Movement always aggravates the pain; but if persevered in, will not infrequently prove the best remedy.

The so-called "growing pains" of children are probably not muscular; they are often situated in the shins, and sometimes in the shoulder. Occasionally they are really articular and rheumatic; sometimes they are symptoms of tubercular or syphilitic disease of the bones or periosteum, and often they seem to be symptoms of anæmia, and are quickly removed by the administration of iron.

Lumbago or pain in the loins has been already described as a symptom of gravel and stone in the kidney (p. 517); when constant and aching it is in its severest degree an early symptom of smallpox and other fevers (vol. i, pp. 105, 201); in a less acute form it is a common result of fatigue and malnutrition. This is the kind of backache with which some hard-worked people wake of a morning.

In women it is a frequent sign, not only of menorrhagia, but of uterine disorders unattended with hæmorrhage. Here, however, its characteristic seat is not so much the loins, as the sacral region.

The lumbago which is only called forth by movement is often called "a stiff back," but the stiffness is not from mechanical immobility; it only depends on the patient's instinctive avoidance of what causes pain. It is sometimes produced by unusual exercise—a first day's shooting or riding—and is then the direct effect of muscular contraction, like the pain which follows prolonged movement of any kind. The erector spinæ, which has the most constant work to perform, suffers most. Sometimes it follows definite exposure to wet, and this is the nearest approach in causation to true rheumatism. More often it can be clearly associated with thick and acid urine, and may thus be a symptom of lithæmia or of gout. But a large number of cases of lumbago are without ascertainable cause, come on suddenly, and as suddenly pass away. These are very like the painful contraction of muscles known as cramp, and, as in a severe case lately under the writer's notice, may be accompanied with evident contraction amounting to slight clonic opisthotonus.

In the majority of cases, lumbago is a disorder of later life, and (if distinguished from sacral pain) is much more common in men than in women.

Myalgia of the nuchal muscles, or a stiff neck, is usually ascribed to "cold." Similar pains about the muscles of the scapula are more often

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associated with dyspepsia. The fleshy masses of the glutæi and the calf are seldom the seat of continuous aching pain. In the former situation pain is usually unilateral, and depends on sciatica or hip disease; in the latter it is more often spasmodic, and due to the irregular contractions of cramp.

The *treatment* of the various forms of myalgia is not satisfactory. Firm pressure, as with a hard pillow, gives relief in lumbago and sacral pain. A hot bath is an excellent remedy for general aching after the fatigue of a prolonged railway journey or a day's hard riding, or exposure to cold and damp; and many persons find a Turkish bath still more efficacious. Shampooing and hot ironing are extremely grateful in "rheumatism" of the shoulders and lumbago.

As local applications, mustard plasters and friction with turpentine or hartshorn and oil (Lin. Camph. Co.) have not undeserved reputation. Equal parts of Lin. Bellad. and Lin. Chlorof. have often succeeded in the writer's hands when other remedies have failed. In a recent severe case of lumbago, the local use of methyl-chloride on lint proved remarkably efficient in removing pain.

Internally, alkaline and diuretic medicines such as citrate, carbonate, and acetate of potash are generally indicated when the urine is thick with lithates; and chloride of ammonium in full doses is sometimes not less efficient. It probably acts like sodic chloride, nitre, and other neutral salts in favouring transudation. Guaiacum is a valuable medicine when the pains are relieved by warmth.

But whenever possible the myalgia should be traced to its source; and if there is no local cause, a knowledge of the patient's previous illnesses may occasionally lead one to use iodide of potassium, colchicum, salicylate of soda or quinine with signal success.

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GONORRHŒAL SYNOVITIS*

"The gods are just, and of our pleasant vices
Make whips to scourge us."
King Lear.

Distinction from rheumatism—Sex and age—Relation to gonorrhœa—Distribution and local effects—Cases—Symptoms—sclerotitis and complications—Prognosis—Diagnosis—Pathology—Treatment.

SIR ASTLEY COOPER'S 'Lectures on Surgery,' published in 1824, seem to contain the earliest notice of the fact that gonorrhœa may be followed by a painful disease of the joints.

This is commonly called "gonorrhœal rheumatism," but the present writer, in a paper in the 'Guy's Hospital Reports' for 1874, proposed to term it *gonorrhœal synovitis.* Whether that name or "gonorrhœal arthritis" be preferred, we shall see that it differs in origin, course, prognosis, and treatment from true rheumatism described in the preceding chapter; its name therefore should be no less distinct. The relation between the affection of the joints and that of the urethra is far too frequent to be accidental. Moreover, we shall see that the clinical course, concomitants, and sequelæ of the disease are quite characteristic and different from those of any other articular inflammation.

Ætiology.—The present writer collected twenty-nine cases from the medical records of the hospital in 1870–72; and during that time many others must have presented themselves in the surgical out-patient room.

All these cases occurred in men; Mr Brodhurst, however, in 'Reynolds' System,' says that he has met with a few instances in women, and Mr Davies-Colley and Dr Church have since observed the same. Senator, in 'Ziemssen's Clyclopædia,' suggests that the toughness and thickness of tha vaginal mucous membrane may afford an explanation of the rarity of gonorrhœal synovitis in the female sex. It would be interesting to know whether urethritis is always present in the exceptional cases when it occurs in women; in some it undoubtedly is.

Since the twenty-nine cases above mentioned were published, more than eighty others, sufficiently reported to be available, have been admitted into our medical wards, and they were summarised for the present writer by Dr T. F. Rickets. Of these, seven occurred in women (two being open to some doubt), and seventy-six in men. Adding the twenty-nine earlier cases, we have a total of one hundred and five male and seven female patients.†

These 112 patients were, with few exceptions, in early adult life;

* *Synonyms.*—Gonorrhœal Rheumatism, Arthritis, or Syndesmitis.—*Fr.* Rhumatisme blennorrhagique.—*Germ.* Tripper-rheumatismus, Trippergicht.

† Mr Burghard, then Surgical Registrar at Guy's Hospital, abstracted the reports of a considerable number of cases of articular disease, combined with urethral or vaginal discharges, of which twenty-one are sufficiently complete to make it tolerably certain that they were examples of this disease, and not of accidental coincidence of gonorrhœa with inflammation of a single joint. Of these twenty-one surgical patients, seventeen were men and four women.

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11 were between sixteen and twenty; 66, or more than half, were between twenty and thirty; 30 were between thirty and forty; 4 were between forty-one and forty-seven; and one was fifty-two.

A urethral discharge was present in every case, but it was often not recognised until looked for to explain the synovitis, when a gleet was always found. The ætiological relation of the synovitis was again and again proved by the patient having previously suffered from "rheumatism" after gonorrhœa, sometimes three and even four times. Among our 112 patients, eighteen gave a history of a similar painful affection of the joints having followed a previous attack of gonorrhœa; fourteen had twice, and one had three times, suffered in the same way before.

Sir Benjamin Brodie recorded an instance in which, after two attacks of synovitis from gonorrhœa, two later ones were attributed to irritation of the urethral canal by the use of a bougie.

According to Mr Brodhurst, exposure to cold and wet may be an exciting cause. He mentions the case of an officer serving in a tropical climate, who, while suffering from gonorrhœa, slept out of doors until after sunset, and awoke in such pain that he could with difficulty be removed to bed.

In fourteen out of 112 cases the patient had previously experienced what was called rheumatic fever; but in two of them this was pretty clearly the same complaint as that from which he was then suffering, and in all but one there was no sign of cardiac disease—an improbable exemption after rheumatism at an early age. With a disease so common as rheumatism in children and young adults, its occasional occurrence previously to gonorrhœal synovitis is probably a mere accident.

In a considerable number of cases there was a history of "rheumatism" or "gout" in the parents or brothers and sisters of the patient. In thirteen they were "rheumatic" or "subject to rheumatism," in ten the father was "gouty," or had "rheumatic gout," and in seven cases a near relation had suffered from "rheumatic fever" or "acute rheumatism." Whether this shows any predisposition to inflammation of the joints generally—an "arthritic diathesis"—is very dubious.

The length of time which elapses between the commencement of the gonorrhœa and the development of the synovitis is put by Mr Brodhurst at from ten days to three weeks; but we found it to range from three or four days to six months, most often falling between one week and three months. Sometimes the urethral discharge continues to be profuse after the appearance of the joint-affection, but in most instances there remains only a slight gleet. Indeed, the patient is very apt to omit all mention of its presence, or even to deny that he has had any venereal complaint.

It is a curious question whether multiple arthritis ever occurs as the result not of urethral suppuration, but of otorrhœa, purulent ophthalmia, leucorrhœa, or other discharges from mucous membranes. Some cases of the kind have been published by Dr Ord, Mr Lucas, and other observers, but they must certainly be rare, and need careful criticism before the proposed explanation is admitted.

Locality.—Writers generally say that the knee is the most frequent seat of gonorrhœal synovitis; but at Guy's Hospital it has long been taught that the feet are the parts most apt to suffer, and this was borne out by analysis of the first twenty-nine cases above mentioned; for in more than twenty of them the ankle, sole, heel, or instep was attacked, while the knee was affected in only fourteen, the wrist in six, the shoulder in three, the hip and

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the elbow in one each. This pain in the heels and soles of the feet has to be inquired after, for it is not attended by swelling or other signs of inflammation. Hence it is often overlooked in reports, while stress is laid on the more obvious changes in the knee and elbow. The "plantar fascia" is the traditional seat of these pains, which are almost always worse at night, and of an aching, grinding character; but it seems probable that the local lesion here, as elsewhere, is in the synovial membrane and ligaments of the joints.

However this may be, the much larger number of cases collected between 1873 and 1887, including the twenty-one from the surgical wards, give results more in accordance with general belief, so far as the pre-eminence of the knee-joint is concerned. For among 102 patients, one or both knees were affected eighty-two times, the ankle fifty-five times, the sole, heel, instep, or metatarsus only thirty-seven times, and the hip twenty-six times. Of the joints of the upper extremity, one or both shoulders were affected thirty-four times, the elbow twenty-two, the wrist twenty-six, the metacarpus (and occasionally the fingers) twenty-five, and the sterno-clavicular joint twice. In six cases the cervical vertebræ suffered, and in two the tempero-mandibular joint.

The great toe is never affected alone; but there is often pain referred to the metatarsus generally and to the smaller toes, which usually escape in gout. The joints of the fingers are very seldom attacked, a point of distinction from subacute cases of osteo-arthritis in young subjects.

There is no rapid "metastasis" from joint to joint, as in true rheumatism; each suffers for many days, or even weeks. There is less local tenderness than might be expected from the pain, and but little redness or œdema; but effusion may almost always be detected in the larger and more superficial joints, particularly in the knee.

In a certain number of cases one joint only is attacked, but this is certainly rare; when it does occur, one knee or one ankle seems usually to suffer. But often, after the other joints are well, one remains as bad as ever, and this appears to be most frequently the elbow or the shoulder.

Cases.—A healthy, well-built young policeman, aged twenty-one, who was in Philip Ward for nearly three months in 1885, had almost every joint in the body successfully affected: first the ankles, heels, and instep, then one knee, then the elbow, shoulder, temporo-maxillary, atlanto-axial, and probably the cervical articulations.

There was severe sclerotitis first of one and then of the other eye. From being stout and florid he became pale and emaciated, but with time and the free use of opium he gradually recovered, and went out free from pain, though stiff and weak. In February of the present year (1891) this patient came to see the writer. He had very slowly recovered his flesh and colour, but about two years after his discharge from hospital was as well as ever. He had married six months ago, and since then had felt return of pain in one hip. The right shoulder had never completely recovered its mobility.

Another patient was a man of about twenty-five, who came into hospital in July, 1890, with a third attack of multiple arthritis, after a third gonorrhœal infection. Here, again, the extent and severity of the disease was remarkable. All the joints of the limbs, some of the neck, and one of the temporo-maxillary articulations were successively, and many of them simultaneously, affected.

A moderate gleet was present, and also double otorrhœa, but this last discharge dated from long before puberty. It was successfully healed, but

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the urethral inflammation was only aggravated by attempts to cure it by local means, and at last he begged it might be left alone: the synovitis was not made worse when the gonorrhœa was most severe, nor benefited by its subsidence. The eyes were not affected, though they had been in a previous attack. Relief was obtained by full and increasing doses of opium, but the case was very obstinate and afforded a good opportunity of demonstrating the effect, which was negative, of treatment by free and continued exhibition of iodide of potassium, guaiacum, and quinine. In his previous attack he had taken a great deal of iodide and had found it quite useless. The temperature was never above 102°, and after a time became normal. In October a new house physician tried new methods of treatment but with no more success than before. The patient ate well and did not become anæmic, and the pains gradually subsided, but the joints were as stiff as ever. By slow degrees, helped by friction, shampooing, and passive movement, power of motion was restored, but he was only able to leave his bed about Christmas; and when at last he walked out of the ward in January, 1891, though in good health several of the joints still remained more or less stiff.

Symptoms.—The *pain* of gonorrhœal synovitis is usually worse at night, and is always described as of a dull, constant, aching character.

The *pyrexia* is almost always moderate, and in some instances the temperature remains normal. Hyperpyrexia is unknown.

This disease differs much from true rheumatism in the fact that even in young subjects it shows little or no tendency to produce *cardiac inflammation*—pericardial or endocardial. Among the 112 cases on which this chapter is based, there was in two a slight systolic basic murmur heard for a time, probably pulmonary and anæmic, in three there was a systolic apex murmur (described as doubtful in one case), and in one only a diastolic, presumably aortic, bruit, which, however, disappeared in a day or two. In the very severe first case above mentioned there was a basic bruit during the height of the illness, but this had disappeared before he left hospital, and on his re-appearance after nearly six years there were neither signs nor symptoms of cardiac disease. In no case does it appear that permanent cardiac lesions have resulted from gonorrhœal synovitis, however young the patient and however protracted the disease.

It is, however, frequently accompanied by a peculiar form of *inflammation of the eye.* Thus, what first drew Sir Astley Cooper's attention to the subject seems to have been the case of an American gentleman, who came to him on account of a gonorrhœa, and said that two previous attacks had each led to inflammation of the eyes, and a few days later to swelling of the joints. Precisely the same sequence occurred on the third occasion, under the observation of Sir Astley himself. The writer observed this affection in eleven out of nineteen cases under his own care, and it was noted twenty-one times in the eighty-three cases collected for him by Dr Rickets. It usually attacks first one eye and then the other. It is commonly attended with injection of the small radiating vessels of the sclerotic which surround the cornea, sometimes with marked iritis and often with some degree of catarrhal ophthalmia. It usually subsides in a few days, under treatment by cold bathing, covering from light, and applying atropine drops; but in more than one instance it returned, after having disappeared. This *sclerotitis*, as it may be distinctively called, is quite separate from the purulent ophthalmia which results from inoculation with gonorrhœal pus, and also from syphilitic iritis and choroiditis.

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Course and event.—Gonorrhœal synovitis commonly runs a tedious and protracted course, lasting for several weeks, and even for months, notwithstanding treatment. In hospital practice the obstinacy of a supposed "subacute rheumatism" has often led to the discovery of the fact that the patient had a gleet.

Recovery seems to have occurred in each of our 112 medical and 21 surgical cases, although some of the patients left the wards before they had entirely lost the pains and stiffness of their joints. Fibrous ankylosis is not an uncommon sequela.

Mr Brodhurst relates a case in which the hips, the knees, and the jaw were all fixed; and another in which, during five years, ancylosis invaded the articulations of the vertebræ, the atlas and the occiput, so that the head could not be moved. That patient had suffered from three several attacks of gonorrhœa, each followed by "rheumatism." About six months after the last attack, and when he was only just able to walk about, he was unfortunate enough to marry a woman who had an occluded vagina. Soon afterwards, although there was no urethral discharge, the articular inflammation recurred; and on this occasion it led to the terrible results just described.

Though peculiarly tedious and obstinate, gonorrhœal synovitis when once cured does not return—another difference from true rheumatism. Numerous instances, however, prove that a fresh urethritis will produce a fresh synovitis, and in this way the same patient may suffer twice, thrice, or even oftener from "gonorrhœal rheumatism."

The most frequent seat of fibrous ankylosis is in the shoulder and elbow; but when the adhesions have been broken down under chloroform, the joint generally regains its usefulness. In the more protracted cases it is not very uncommon for eburnation and the other changes of osteo-arthritis to supervene, but the deformity thus produced is, as a rule, only slight.

Diagnosis.—The mere presence of a urethral discharge is obviously not enough for the determination of the disease, though the presence of a stricture or a gleet is essential. A man suffering from gonorrhœa may be attacked by rheumatic fever, and the two diseases run their course apart. Or a man subject to gout may be infected with gonorrhœa. But it must be admitted that some cases of osteo-arthritis, as we shall find in the next chapter, have their probable origin in severe gonorrhœal synovitis.

The distinction from *gout* depends on the age of the patient, the absence of tophi, the distribution of the synovitis and the freedom from return, except from a fresh urethral excitation. Sclerotitis when present is a valuable help to diagnosis.

It is more difficult to distinguish gonorrhœal synovitis from *rheumatism*, with which it is still commonly confounded. The ætiology, the different relation to sex and age, the continuous instead of transient affection of the several joints, the much longer course, the freedom from relapses or spontaneous recurrence, the affection of the eyes and the immunity of the heart —are amply sufficient grounds for the pathological distinction between the two diseases, and with care and attention are almost always sufficient for a right diagnosis in practice. The difference in prognosis from both rheumatism and gout, and the no less important difference in treatment, make the discrimination between these diseases of great importance.*

* See an excellent paper by Dr Thomas Bond published in the 'Lancet,' March 23rd, 1872, before the writer's paper in the 'Guy's Reports.'

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The absence of pyrexia and the normal state of the other organs, together with the fact that the joints do not suppurate, are distinctions from *pyæmia* no less than from rheumatism.

It is a question whether we ought to distinguish the cases above described from certain much more severe and localised inflammations of a single joint following gonorrhœa, which come under the notice of the surgeon rather than the physician. They have been described by Duplay and Brun in France ('Arch. Générales de Médecine,' 1881), and by Mr Davies-Colley in England ('Guy's Hosp. Rep.,' 1882). In both articles they are accounted as belonging to gonorrhœal synovitis, but Mr Colley proposes to distinguish them as acute gonorrhœal arthritis or syndesmitis. The joint is very prone to ankylosis after this severe local inflammation.

However this question may be decided by further experience, there is no doubt that we must exclude from the disease now under consideration the rare cases of true pyæmia arising from gonorrhœa, with suppuration of the affected joints, of which an example was recorded in the 'Pathological Transactions' for 1885, by Mr Pollard.

Pathology.—The physiology of gonorrhœal synovitis is quite unknown. Whether in its milder or in its more severe forms, it is certainly not a modified pyæmia, a process from which it differs at every point. Nor is it a "hybrid" or "modification" of gout or rheumatism. Possibly it may be connected with the abundant nervous distribution over the prostatic part of the urethra, and so be related on the one hand to the rigors which sometimes follow the passage of a catheter, and to the arthropathies of tabes on the other. But this is mere speculation. The micrococcus of gonorrhœa has been found in the effused serum in the knee-joint, by Petrone and Kammerer, but it is not certain that this microbe is truly specific.

Treatment.—Some authors recommend iodide of potassium in full doses; but the writer has tried this drug in many cases for a long time, and is convinced of its inutility. Colchicum, quinine, guaiacum, and salicyl-compounds have proved equally useless.

The patient must be kept in bed and upon light diet during the first week or two. Mr Brodhurst insists upon the importance of placing the affected joints in splints, and says that leeches sometimes do harm. He has seen the Turkish bath very useful, and instances a patient who was lodged in a house attached to one of these baths, so that he could be carried down into the hot chamber every day; when profuse perspiration was obtained, the pain, which was very acute, left him for the time.

Opium should be given in sufficient doses to relieve the pain and procure sleep. It probably is useful in other ways also, and no other drug is of so much value. Good feeding, bark and nitric acid, quinine, and a moderate use of alcohol, particularly porter, are necessary as soon as the first severity has subsided. Steel is sometimes of use, and arsenic is perhaps still more valuable during the long and tedious convalescence. It is important that every effort should be made to cure the urethral disorder, but the synovitis is often little benefited by success.

In the chronic stage, blisters may be applied with advantage, or liniment of iodine, or mercurial ointment. When one or more of the joints has become fixed, it is often advisable to give chloroform and to break down the adhesions by force.

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OSTEO-ARTHRITIS*

"If I were feeble, rheumatick, or cold,
These were true signs that I were waxen old."
DRAYTON.

History and nomenclature—Pathology and relation to gout and to rheumatism—Anatomy of the joints—Symptoms and course—acute cases—resulting deformity—Age and sex—Ætiology—Diagnosis—Prognosis—Treatment by drugs and external applications, by diet and climate.
Charcot's arthropathie ataxique—its relation to osteo-arthritis and tabes dorsalis.

THIS remarkable disease appears to have been first recognised by Sydenham. He says that rheumatism, when free from fever, is often called arthritis (*i. e.* gout), though really distinct from it:—"Unde forsan petenda est ratio cur tam sicco illum pede transiverint scriptores medici; nisi forsan arbitremur hanc morbi speciem ad reliquam malorum Iliada de novo accessisse." He goes on to describe the chronic course of the disease, with its remissions and exacerbations, and concludes his description as follows:—"Potest fieri ut æger omni membrorum motu ad mortem usque privetur, digitorum articulis quasi inversis, et protuberantiis, ut in arthritide, nodosis, in interna magis quam externa digitorum parte se prodentibus: stomacho nihilominus valeat, et cætera sanus vitam toleret."† The affection was clearly described by Heberden in 1782 as "the chronical rheumatism," which, he writes, "is in reality a very different distemper from the genuine gout, and from the acute rheumatism, and ought to be carefully distinguished from them both. Being so very different in its symptoms, as well as in the event, it would be useful if it were distinguished by a peculiar name, which might prevent its being confounded with other disorders by being called a "spurious or wandering gout" or a "chronical rheumatism." Haygarth in 1805 wrote as follows:—"This disease has hitherto passed under the name of gout or rheumatism, or perhaps has been most commonly called rheumatic gout. But as several advantages would result from a separation of this disorder from others with which it has been confounded, I have ventured to call it the nodosity of the joints." Cruveilhier gave the first accurate anatomical account of osteo-arthritis under the title "arthrite avec usure des cartilages articulaires." Virchow and most German pathologists call it "arthritis deformans." By many English authors it is still known as "rheumatic gout," by Adams in his beautifully illustrated monograph as "chronic rheumatic arthritis," and by Garrod as "rheumatoid arthritis." It is also supposed to correspond to the "poor man's gout" (*arthritis pauperum*) of older writers; but true gout is far from uncommon among the poorer

* *Synonyms.*—Arthritis deformans, Nodi digitorum, Malum articulorum senile; Chronic rheumatic arthritis, Rheumatoid arthritis, Rheumatic gout, Irish gout, Chronic rheumatism (in part), Nodosity of the joints.—*Fr.* Rhumatisme noueux, Goutte asthénique, Arthrite sèche.—*Germ.* Arthritis nodosa sive deformans, Deformirende Gelenkentzündung.

† 'Obs. Med.,' sec. vi, cap. 5 (p. 256 of Syd. Soc. ed.).

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classes of this country. In popular language this disease is generally called "rheumatic gout."

Objection may be made to all the above names, and it is perhaps best to adopt the term *osteo-arthritis*, which is used in the "nomenclature of diseases" of the College of Physicians, and in the Registrar-General's Reports. The course is most often "chronic," but sometimes subacute, hence it is better not to use the adjective as a constant epithet. *Arthritis deformans* is a distinctive and expressive name, but *rheumatic arthritis* is ambiguous and *rheumatic gout* misleading.

Pathology.—There are still some pathologists who maintain that all articular diseases, including even acute rheumatism, are closely related to one another, and in common depend upon what is termed an *arthritic diathesis*. This view is held by most French writers, and in this country by Mr Hutchinson, who thinks that he can identify an "arthritic" iritis, and even an "arthritic" pneumonia, by their recurring again and again in the same individual. He brings forward instances in which different members of the same family, belonging to successive generations, are said to have suffered from different "arthritic" affections; but one cannot attach exact significance to a statement that a mother, or an uncle, or a grandfather had "rheumatic gout," or "chronic rheumatism," or even "gout," unless we have a more detailed account of the case than can generally be obtained.

It is true that the lesions characteristic of gout and those which belong to arthritis deformans are sometimes found in different joints of the same person, or even in the same joint; and, apart from mere coincidence, eburnation and "lipping" probably follow repeated gouty arthritis. Between 1874 and 1879 four examples of this association were met with in the autopsies at Guy's Hospital. Three of the patients were men, aged fifty-two, fifty-four, and sixty-two respectively. The fourth was a woman aged thirty-six; and it is an interesting fact that she was said to have had rheumatic fever at the age of twelve, which in all probability was really the case, for she died of mitral stenosis. In each instance the great toe joints contained urate of soda, showing that gout had been present, while the lesions indicative of arthritis deformans were found in the knees or in the hip-joints. Once the left knee showed both kinds of change, the right that of arthritis deformans alone, and the two great toes that of gout alone. In a fifth and later instance the writer observed the edges of the patella thickened, and other signs of osteo-arthritis present, together with patches of urate of soda.

Again, there is no question that osteo-arthritis may be preceded and apparently caused by a single attack, or more often by a series of attacks of rheumatic fever. In the great majority of cases true rheumatism leaves no deformity behind, and this is a most remarkable and distinctive point in its natural history; but that the synovitis of rheumatism, when unusually protracted, or when frequently repeated, should produce no structural change would be most improbable.

The same results are produced more frequently by gonorrhœal synovitis, as we should expect, since here, as in gout, there is a far more persistent arthritis than in true rheumatism. They may also follow traumatic synovitis. In a case reported by Mr. Hutchinson in the 'Med. Times and Gaz.' for 1881, of a young woman whose thigh was amputated by Mr McCarthy for a myeloid growth in the tibia, bony lips were observed on the edges of

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the condyles of the femur, apparently as the result of irritation caused by the proximity of the tumour. So that if we define osteo-arthritis by the peculiar anatomical condition of the joints, to be presently described, we must admit that it may be the consequence of rheumatic, gonorrhœal, gouty, or traumatic arthritis if long enough prolonged or repeated. It is, on the other hand, never caused by the more acute and destructive arthritis of tubercle, syphilis, osteomyelitis, or pyæmia.

Nevertheless, it remains true that in the great majority of cases this characteristic multiple articular lesion does not follow either rheumatism or gout, or any other form of articular inflammation, that in fact such a sequence is exceptional and rare, and that, on the contrary, it far more often appears independently. Sometimes it may be traced to hard usage of the affected joints; more frequently it depends upon the ordinary wear and tear of laborious work during many years; and often it appears in young subjects who have never worked hard, and have never suffered from any kind of arthritis.

Moreover, the primary or idiopathic anatomical change in the joints is associated with a definite and constant pathological process, and with characteristic clinical symptoms. It has its own course, prognosis, and treatment, and therefore is, on every ground, practical and theoretical, entitled to be considered as an independent "disease."

Anatomy.—The process of arthritis deformans is often limited at first to a small area of one of the cartilages of a joint. Sometimes (as in several specimens exhibited to the Pathological Society by Mr Hutchinson in 1872) it begins round the margin, and spreads irregularly inwards; more often it starts in the middle of the articular surface.

The cartilage first becomes soft and velvet-like in appearance; afterwards it softens and gradually melts away, so that a depression is formed, in the floor of which the bone may be exposed. Histologically the change consists in a proliferation of the cartilage-cells, so that each becomes replaced by from eight to twenty corpuscles; the matrix at the same time splits into fibres in a direction perpendicular to the articular surface. Presently the enlarged alveoli rupture into the cavity of the joint; the fibres then remain for a time as shaggy projections, until ultimately they too disappear. There is at no time true ulceration, *i. e.* no suppuration and no formation of granulations.

The denuded bone is said occasionally to exhibit an open cancellous tissue, but as a rule it is converted into a very hard, compact substance, or, to employ the usual term, it undergoes *eburnation.* When, after the removal of the whole of the articular cartilages, the osseous surfaces everywhere come into apposition, with no soft material between them, they become scored and fluted with parallel grooves and ridges, corresponding in direction with some particular line of movement, to which they are henceforth restricted. The texture of the subjacent part of the bone becomes wasted, filled with oil-drops, and so gradually absorbed. Thus the neck of a femur may be gradually shortened until what represents the head lies in a hollow between the two trochanters.

Intra-articular fibro-cartilages, as in the temporo-maxillary joints, resist the disease no better than the cartilages which cover bones. So also the ligamentum teres becomes lost when the hip is affected, and in the shoulder the long head of the biceps disappears; it seems to undergo

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fusion with the capsular ligament of the articulation, so that it cannot be traced upwards beyond a certain point.

Formative changes are generally, though not always, associated with the destructive and atrophic process from a very early period. From the edges of the articular cartilages there arise a series of nodulated outgrowths—or "ecchondroses," as they are termed—which form a raised lip or border. These are at first small, but they afterwards increase in size, and soon lime salts are deposited in them, so that they become converted into bony plates or masses (*osteophytes*), which not infrequently grow into the capsular ligament, or into the tendons round the joint, and unite to form a complete osseous shell around the joint. In like manner outgrowths from the edges of the bodies of the vertebræ often cohere together across the intervertebral discs, so as to constitute a number of bony splints, which may immoveably fix a large part of the spinal column.

The synovial membrane of the affected joints is more or less thickened, especially near its lines of attachment to the bones. The folds, which project into the joint-cavity, are greatly enlarged and very vascular; they often form long villous processes, with numerous bodies like melon-seeds hanging from them; these bodies may ossify, become detached, and lie as "loose cartilages" in the joint. They cause fresh accession of pain, and are named *Mäuser* by the Germans, from their slipping away from the touch.

A modification of the bony outgrowth of arthritis deformans was long ago described by Heberden under the name of *digitorum nodi*, which, as he says, consist of little hard knobs, about the size of a small pea, situated upon the fingers, near the joint; they have no connection with gout, continue for life, are hardly ever attended with pain, and are rather unsightly than inconvenient ('Comment.,' cap. 28).*

The changes of osteo-arthritis are attended with a gradual loss of mobility, and at length the joints become fixed by the bony splints around, although when these are removed after death the articular surfaces are found perfectly free and smooth. Neither bony union nor suppuration ever occurs, but occasionally fibrous ankylosis is established.

At an advanced stage of arthritis deformans the deformity produced is characteristic. Most of the joints, as a rule, are fixed in a bent position; but the wrists are commonly extended. The fingers almost always lie at an angle with the rest of the hand, and are deflected to the ulnar side, so that the knuckle of the forefinger projects towards the thumb. Some of the phalangeal joints are usually over-extended, so as to be concave on the dorsal aspect, as was noted by Sydenham. The degree of enlargement is very variable. Sometimes each articulation forms a bulbous swelling; sometimes the ends of the bones are almost of normal size. The elbows and knees are greatly enlarged, and are, next to the hands, the most obviously deformed.

Distribution.—The fingers and wrists are commonly first attacked (in about two thirds of the number of cases), then the knees. The hip may be affected alone, but only in the later periods of life (*morbus coxæ senilis*). Next to the hands and knees, the elbows and shoulders are most often affected, the feet less frequently. The vertebræ are very often found

* In Sir D. Duckworth's treatise on gout, and in that of Dr A. Garrod on gout, the relation of these *nodi digitorum* to gout is discussed. They are certainly not tophi but exostoses, and in the writer's belief are neither rheumatic nor gouty nor syphilitic, but osteo-arthritic.

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united by osteo-arthritis after death, and this is one cause of the stooping back and stiff neck of old people in the workhouse. Adams figures the disease well marked in the temporo-maxillary articulation, and it is stated to affect the bones of the ear and the ossified laryngeal cartilages of old age.

Symptoms.—Usually the disease causes more or less local pain from the beginning, but occasionally its early stages are without any subjective symptoms. This fact is well illustrated by one of Mr Hutchinson's cases in the 'Pathological Transactions.' A man, aged forty-one, had his thigh amputated for disease of the knee, it not being known that any other joints were affected; but the cartilage was found eaten away in every articulation of the foot, except the distal joints of three toes. The same writer speaks of having frequently been able to detect the presence of a projecting lip round the articular cartilage of the lower end of the femur, in persons who are not aware that the knee had ever suffered. He places himself in front of the patient, puts the finger-ends of one hand flat upon one condyle, and those of the other hand upon the other condyle, and then directs him to bend and extend the joint slowly several times in succession. In this way, he says, the edges can be easily found, and the degree of their elevation estimated. He admits, however, that practice is required to prevent one mistaking for a morbid condition a ridge which normally exists at the same spot in many healthy persons.

Dr Spender, of Bath ('Brit. Med. Journ.,' 1888, vol. i, p. 781), who has seen large numbers of patients in the early as well as in the chronic stages of osteo-arthritis, believes that it is marked at or even before its development in the joints by cold hands and feet, with a rapid and incompressible pulse (90 to 110 or 120) but no rise of temperature. He has also noticed as an early symptom pigmentation of the skin, particularly on the forehead and face and on the fingers, and afterwards maculæ over the legs, and freckles on the face. A constant dampness of the hands from perspiration, and liability to fits of neuralgic pain in the affected limbs, are other early symptoms on which Dr Spender lays stress.

It is probable that when a patient begins to complain of pain and stiffness in one or more of his joints, the disease has often been present for a considerable time. The pain is not generally constant; it "comes and goes," sometimes without obvious cause, sometimes in apparent relation to changes of the weather. In certain cases it has a shooting character, so as to resemble neuralgia. With many patients it is worse when the limbs are warm, in others when they are cold; in some by night, in others by day. It is very apt to be brought on by the use of the part; in the hip, for example, by walking even a short distance; in the shoulder, by carrying anything, however small, in the hand. The stiffness, however, is usually more noticeable after rest, as when the patient first attempts to get out of bed in the morning.

A sense of weakness and of distressing fatigue and impotence may be as marked a symptom as pain; and there is often far more wasting of the muscles than seems to be accounted for by mere disuse of the limbs; the thenar and the hypothenar eminences, for example, may be so hollowed that the case looks like one of progressive muscular atrophy.*

* "The chronical species of rheumatism equally partakes of the palsy, for there is always a trembling, weakness and numbness left for some time in the limb affected, and the use has at last in many been wholly taken away" (Heberden, 'Comment.,' cap. 79).

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Another very characteristic symptom, which, however, is not present during the early stage in the disease, is creaking or grating, which accompanies the movements of the affected joints; it can be plainly felt or even heard by the patient, and is easily recognised by grasping the joint with one's hand.

In some cases osteo-arthritis sets in from the first with well-marked symptoms; it produces swelling, heat, and even perhaps redness of the affected joints, and is attended with more or less pyrexia; so that Sir A. Garrod described an *acute variety* of the disease, which, he says, very closely resembles ordinary acute rheumatism, differing only in the greater length of the paroxysm, in the absence of profuse sweating, and in its having no tendency to attack the heart;* and Mr Hutchinson declares that it sometimes gives rise to paroxysms as short and as definite as those of gout itself. In one of Dr Ord's cases the temperature one evening rose to 102·8°. These cases, however, are quite exceptional, and the disease is in most persons insidious and gradual from its first onset.

There is no doubt that osteo-arthritis is often attended with effusion of fluid into the affected joints, and that the designation of *arthritis sicca* is inappropriate. Many cases of so-called *hydrops articuli* really belong to this disease. Analyses of the effused fluid from the hip-joint in arthritis deformans have been made by Hoppe-Seyler, and recorded in 'Virchow's Archiv' for 1872; he found a proportion of mucin which greatly exceeded that in normal synovia.

In other cases hæmorrhage takes place into the affected joints. Thus in 1875 Dr Goodhart, in examining the body of a man aged fifty, who had been admitted for arthritis deformans, but had died of hernia, found that each ankle contained several drachms of liquid blood, and that there was also blood in both knees, with swollen synovial membrane and rounded vascular œdematous fringes; all these joints, as well as the hips, showed the characteristic changes of arthritis deformans.†

Age and sex.—Osteo-arthritis is decidedly more common in women than in men. Of Haygarth's 34 cases all but one were in women. Of 75 well-marked cases which have come under the writer's personal observation, 48 occurred in women and 27 in men. Of Adams' 21 patients, 16 were men and only 5 women, but many of the former were cases affecting the hip alone. Among 500 private cases of Sir Alfred Garrod there were 411 in women and only 89 in men ('Med.-Chir. Proc.,' Nov., 1887).

This disease is very rare in children, and is not commonly met with under thirty. Of the 75 cases quoted above, 3 occurred before the patient

* Trousseau states that in four out of nine autopsies of "nodular rheumatism" made at the Salpêtrière by Cornil, pericarditis was present. It is, however, incredible that this was connected with a disease which rarely, if ever, produces cardiac lesions; and on looking into the cases it is at least probable that chronic cirrhosis of the kidneys was the real cause of the pericarditis which ended some of these long-standing cases of arthritis.

† Among the less common symptoms of arthritis deformans is the presence of fibrous nodules at a distance from joints, as, for instance, among the muscles of the arms or forearms. I once saw an old lady, aged seventy-one, who for about three months had complained of a curious affection of the tongue and cheeks, which perhaps belonged to this disease, inasmuch as she also had hydrarthrosis of each shoulder-joint, and a less marked affection of her knees. The tongue was uniformly enlarged, and had a peculiar firm, fleshy consistency, without being at all indurated; its surface was rather smoother than natural. At each corner of the mouth there was a button-like mass, which extended outwards for some distance into the substance of the cheek, and on which the mucous membrane adhered more closely than elsewhere to the subjacent tissues.—C. H. F.

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was twenty-one years old—one in a girl of sixteen—and 7 between twenty and forty; 3 patients were above seventy years old, 7 between sixty and seventy, and all the others were between forty and sixty. In many cases, as Haygarth remarked, it first develops itself in women at the climacteric age, and it has been supposed to depend on uterine disturbances. It is, however, by no means confined to any one period of life. Garrod says that he has seen it in children of ten or twelve years, and he has also met with instances in which it began in people above seventy years of age.

The characters of arthritis deformans differ somewhat according to the sex and age of the patient, so that two types of the disease may be recognised. One begins in the hands and feet, and subsequently spreads to the larger joints; this almost always occurs in women, and belongs to the earlier periods of life. Men are more liable to have the disease in one hip or one shoulder, before any other parts are affected, and this variety is especially frequent at an advanced period of life. Moreover, as Mr Hutchinson remarks, the change in the affected joints is not quite the same at different ages; under middle age the outgrowths of bone, which are so striking a feature in old people, are rare, and if present they are usually small.

Ætiology.—Osteo-arthritis is the disease commonly called "chronic rheumatism," which cripples the joints of old men in the country who have been exposed to wet and cold during a lifetime of hard work and often scanty food. These patients have never suffered from true rheumatism; the heart is unaffected (unless from chronic atheroma), and they live to an advanced age; but they suffer much from these crazy joints, and not infrequently are bedridden from the same cause during the last years of their lives.

This "chronical rheumatism" is much affected by the weather, usually worse on damp and better on dry and warm days, sometimes worst with an east wind. The patient feels the changes of the barometer, and suffers least in the settled summer season.*

The ætiology of arthritis deformans seems to differ altogether from that of gout or true rheumatism. Garrod was unable, after looking over a large number of cases, to find much evidence of its being transmitted by inheritance; one member of a large family not infrequently suffers severely from it, while the rest remain free. Again, it is not produced by indulgence in rich food or in alcoholic stimulants. On the contrary, it is apt to occur in weakly, ill-fed women, who are exhausted by repeated child-bearing, by menorrhagia or prolonged lactation, by grief or mental anxiety.

Dr Ord, in the 'Transactions of the Clinical Society' for 1877, has brought out the relation of osteo-arthritis to dysmenorrhœa and "ovario-uterine provocation," which the late Dr Fuller had insisted upon. In one of his patients it was regularly developed paroxysmally just before, throughout, and for a short time after each menstrual period, and underwent no less regular remissions in the intervals. He noticed three cases in which the arthritis was limited to, or began and remained excessive in, one side of the body, while the ovary on the same side was painful and tender. Haygarth remarked that his nodosities "usually begin about the period when the menses naturally cease."

* "The hollow winds begin to blow,
The clouds look black, the glass is low,
Hark how the chairs and tables crack,
Old Betty's joints are on the rack."
Signs of Rain, a Poem by EDWARD JENNER.

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Garrod believes that tubercular subjects are especially liable to be affected by osteo-arthritis; as also are "individuals of weak frame, whose circulation is languid, and whose extremities are habitually cold." Among thirty-four cases examined by the writer with this point in view, there were three in which phthisis was also present.

The nervous system has often been regarded as the origin of this remarkable disease. But there is not much pathological connection between embarrassment in business, mental distress and being frightened by shells in a siege, on the one hand, and changes in the spinal cord, like those of Charcot's disease, on the other. A nervous origin has also been suggested for gout and rheumatism (pp. 675 and 695), but we must remember that every disease may have a cellular (local, "solidist") theory or a "humoral" theory; in modern language, it may be ascribed either to a bacterium or to trophic nerves and sympathetic centres.

To some extent we must recognise any kind of slight but frequently repeated irritation as a cause of arthritis deformans. To a still larger extent we must admit that it is a degenerative or senile change. And, lastly, we shall find that in many cases there is a distinct traumatic origin, such as has often led to the lesions of this disease being confounded with those of fractures and dislocations (Adams).

Apart from actual injury, overwork, or the wear and tear of many years, is a cause or an occasion of osteo-arthritis. Senator insists that the parts most apt to be attacked are such as have been most used; as, for instance, the fingers and the wrists of watchmakers, and of women who have worked hard with the needle or at knitting. The result of different kinds of labour in producing pressure on certain joints with "usure des cartilages," eburnation of the articular surface and prominence of its edges, has been ingeniously analysed by Mr W. A. Lane in several interesting papers published in the 'Guy's Hospital Reports,' the 'Pathological Transactions' (1886), and the 'Journal of Anatomy' (vol. xxi).

The effects of pressure and irritation in producing the anatomical deformity of osteo-arthritis are not confined to the human race. A similar affection occurs in the feet of horses, probably as the result of overwork.*

The same lesions are also found in other animals, including cetacea —animals much exposed to wet and cold.

With regard to the *geographical distribution* of arthritis deformans in man, no extended observations have yet been made. But Trousseau, after stating that it is "a disease of rare occurrence" (in Paris), remarks that in "certain damp countries it is so common as to be almost endemic." A circumstance of some interest is that signs of osteo-arthritis were detected by Delle Chiaje in bones taken from Pompeii, and by Lebert in skeletons from the catacombs of Paris, so that the disease is clearly not of modern origin, although it has only been distinguished of late years. For an account of a specimen from a Roman tomb by Dr Norman Moore, see 'Path Trans.,' 1883.

There is no question that arthritis deformans is more common in Ireland than in England or Scotland, or that it is more frequent among agricultural labourers than in towns. Adams found specimens more numerous in Holland than anywhere else except Dublin.

Diagnosis.—The distinction of osteo-arthritis from other affections of the joints rests upon recognition of the characters above given. Its gradual

* This may perhaps be the affection described by Aristotle as ἡ τῶν ἵππων ποδάγρα.

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onset, the absence of pyrexia and febrile urine, the freedom of the heart from attack, and the age of the patient sufficiently distinguish it from true rheumatism. From chronic rheumatism it cannot be distinguished, if we define that term as Sydenham and Heberden did (p. 722); but from all other chronic affections of the joints it is distinguished by its characteristic deformities. The distribution of the lesions and the whole clinical *facies* of the disease separate it from gout; while the age if not the sex of the patient, and its gradual and insidious approach render it unlikely to be confounded with gonorrhœal synovitis.

Prognosis.—The tendency of osteo-arthritis is to grow more and more inveterate. The patient often becomes completely crippled, unable to dress or to undress, to carry food to the mouth, or to hold a pen in his hand.

It must not, however, be supposed that the prognosis is always unfavourable, nor that all patients who become the subjects of arthritis deformans, at least in its slighter forms, necessarily continue to suffer from it for the rest of their lives. The author knew an old gentleman who, when he must have been nearly seventy, was unable during one winter to move the right shoulder, so that he had to be shaved by his servant; but during the following summer he became free from the complaint, and it never returned. Dr Ord speaks of more than one of his patients having regained a fair state of health when menstruation became normal; but he does not say that joints which had been enlarged ever returned to their natural size.

The disease seems to have little or no tendency to shorten life, its duration from first to last being perhaps ten, twenty, or even thirty years. Haygarth's first patient lived to about ninety-three years of age; and death, when it arrives, is directly attributable to some intercurrent affection.

In ten years at Guy's Hospital (1875 to 1884) we had ten deaths with osteo-arthritis, but none of them as its result. One patient, who was only thirty-five, died from adherent pericardium, the result of several attacks of rheumatic fever; the changes in the joints were slight, and certainly secondary to true rheumatic synovitis.

Of the rest, the youngest was fifty and the oldest seventy-four; five were men and five women. Two died of strangulated hernia, three of cancer (one of whom showed signs of gout and of old tubercular phthisis), one of cerebral hæmorrhage, and the rest of chronic interstitial nephritis.

Treatment.—The medicines most serviceable in arthritis deformans are arsenic, guaiacum, and cod-liver oil. Trousseau, following Lasègue, recommended the tincture of iodine in doses of ♏x up to xxx. Iodide of potassium is more often prescribed in England; and it is most likely to be useful when warmth augments the pain. Garrod speaks highly of the syr. ferri iodidi. Dr Ringer says that actæa (cimicifuga) racemosa has yielded him very satisfactory results, and that it is most useful when the pain is worse at night, and when the disease is traceable to uterine derangement.

Given in full doses, and continued for a long time, arsenic is the most efficient drug in this disease, although no doubt cases occur in which it is useless: patients have told the writer that as soon as their eyes begin to itch they know the pains and stiffness will be relieved. Next to arsenic, steel and bark are the most useful drugs. But more important than either is cod-liver oil (which indeed was first introduced by Dr Laycock for "chronic rheumatism"). A generous diet, with porter or wine, is a great help to

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treatment. When the pain is severe, there is no objection to the administration of opium in doses sufficient to subdue it.

Hot douches, hot sand poured over the joints affected, hot baths, and blisters or other counter-irritation are all valuable. Warmth and flannel and powdered sulphur do good, cold and damp are harmful. Passive movement is necessary, and after the hot bath or douche the joints should be well shampooed. Moreover, the patient should be encouraged to persevere in exercising the affected joints, and to play the piano, or make any other exertion of the fingers.

Lastly, removal from Ireland, and if possible from England, to a warm, dry, and equable climate is invaluable. Perhaps most cases might thus be cured if the treatment could be early adopted.

A weak continuous galvanic current is sometimes applied with advantage. A narrow terminal, connected with the positive pole, may be placed over or below each of the affected joints in turn; a sponge, connected with the negative pole, higher up the limbs or nearer the spine.

Tincture of iodine may be painted over the swollen articulations; or they may be strapped up with plaster; or the belladonna liniment may be applied to them; or the linimentum cantharidis, which Garrod says is more convenient than an ordinary blister. Trousseau recommends that the affected parts should be buried in hot sand, at a temperature as high as can be borne, three times a day for an hour or two at a time. In certain cases stimulating applications, such as the cajeput oil, do good. Splints are decidedly harmful. The patient should use the joints as much as he can, sometimes even if it causes increase of suffering for the time. In this case rest is not indicated by pain.

The best climates are such as are warm, but dry and bracing. Drinking alkaline waters does harm; but the chalybeate springs of Tunbridge Wells or Schwalbach may sometimes be useful. The warm baths of Bath and Buxton, with internal use of the waters, are certainly often valuable means of relief and sometimes perhaps of cure.

CHARCOT'S JOINT-DISEASE.*—It is to the acumen of Professor Charcot that we owe the recognition in 1868 of a remarkable chronic affection of the joints which is liable to occur in the earlier stage of tabes dorsalis (locomotor ataxia). It is sometimes abrupt in its origin, but chronic in its course and remarkably wanting in local signs of inflammation. It was first described by Charcot in 1853 as a variety of osteo-arthritis, under the title "rhumatisme noueux d'origine nerveuse."

The joint affected, after a period of swelling probably produced by intracapsular effusion, gradually becomes completely disorganised. The synovial membrane and cartilage disappear, and the articular ends of the bone undergo singular atrophy. The whole shaft of the bone becomes porous and brittle, not only the ends, as in ordinary osteo-arthritis. In the knee, which is most often affected, the tuberosities of the tibia and the condyles of the femur are absorbed, or, when the hip is attacked, its entire head and neck. With this there is little or no hypertrophy. At last the ligaments are so relaxed and the ends of the bones so altered, that the joint swings in all directions like a flail. The shoulder and elbow are also liable to attack, but not, it appears, the joints of the fingers or of the vertebræ.

* *Synonyms.*—Arthropathie ataxique—Maladie de Charcot—Arthritis tabidorum atrophica—Chronic atrophic arthritis.

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Dr Buzzard has called attention to the frequency of the gastric attacks of tabes when this articular lesion is present. He found them present twelve times in twenty-six cases.

The remarkable affection known as perforating ulcer of the foot has sometimes been met with in cases of Charcot's disease.

Whether this affection is a variety of osteo-arthritis deformans, and what is its true pathology, are still disputed points.

Charcot's atrophic arthritis, as we may call it, was briefly alluded to as a complication of locomotor ataxia in the first volume of this work (p. 530). A discussion of its nature by Sir James Paget, Mr Hulke, Mr Hutchinson, many cases were recounted and specimens shown, will be found in the Mr M. Baker, Sir D. Duckworth, Mr Lucas, and other pathologists, in which eighteenth volume of the 'Clinical Society's Transactions,' and an earlier paper by Dr Buzzard ('Path. Trans.,' vol. xxxi, pp. 193, 202) should also be consulted. A good case was published by Mr Keetling ('Clin. Trans.,' 1882), two by Dr Charles Atkin in the Manchester 'Medical Chronicle,' April, 1885, and six by Dr Sydney Roberts in the Philadelphia 'Medical News,' Feb. 14th, 1885. The writer had lately a well-marked case affecting the knee in a man suffering from tabes in John Ward (1890–91). There was much effusion in the joint, and he complained of rather severe pain.

In Germany this condition has attracted little attention. The account in Prof. Eichhorst's text-book merely reproduces the statements of Charcot and his pupils.

It is remarkable that tabid arthropathy is so rare in this country, for it is not a late sequel of tabes, which might only be found in sick asylums and workhouse infirmaries; it belongs to the earlier stages of the disease.

It is believed to be a trophic neurosis, dependent on wasting of the cord; yet it appears to be nearly as common in the upper as in the lower extremity, whereas ataxy of the arms is very rare compared with that of the legs.

The chief distinctions between Charcot's arthropathy and osteo-arthritis are the absence of pain, the rapidity of the destructive process, and the excess of atrophy over hypertrophy—"incapability of repair" as Mr Baker puts it. Whether we may regard it as osteo-arthritis modified by occur ring in a tabid subject, it is difficult to say. That joints corresponding to Charcot's description may be met with in England is certain, and not less so that cases are seen of articular disease clinically combined with tabes dorsalis. But all affections of the joints which do not end in suppuration or pulpy degeneration tend towards the same result, eburnation and deformity. We have seen that gout, true rheumatism when frequently repeated, gonorrhœal arthritis and traumatic synovitis, together with the mere wear and tear of long-continued labour, may each and all produce an anatomical condition of joints, with thickened lips and atrophied cartilage, which is indistinguishable from what may be called primary or idiopathic osteo-arthritis. It may be that tabes is only another cause of what is essentially the same result. But if so, this is itself an important clinical and pathological discovery.

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RICKETS

AND OTHER GENERAL DISEASES AFFECTING THE BONES

> "Whence multitudes of reverend men and critics
> Have got a kind of intellectual rickets,
> And by the immoderate excess of study
> Have found the sickly head to outgrow the body."
>
> SAMUEL BUTLER.

RICKETS—*History—Definition—Anatomy of the rachitic limbs—of the thorax—skull—pelvis—craniotabes—Histology—The spleen in rickets—Symptoms and concomitant disorders—Course and event—Ætiology: age, sex, climate, food—relation to tubercle and syphilis—Pathology—Diagnosis and prognosis—Treatment, preventive and curative.*

MOLLITIES OSSIUM—*History—Ætiology—Symptoms and event—Diagnosis—Histological and chemical changes—Pathology—Treatment.*

Osteitis deformans—Acromegaly.

IN the middle of the seventeenth century the famous anatomist and physician, Francis Glisson, drew attention to a disease affecting the bones of children, which he supposed to have recently sprung up, where it was first observed, in the counties of Devon and Somerset. It was then known as "*the rickets.*"*

The term *Rachitis* (or Rhachitis) was proposed by Glisson on account of its similarity in sound, although in his work on this disease (the second edition is dated 1650) he offers his readers a Greek derivation, on the ground that the dorsal spine (ῥάχις) is one of the first parts to be attacked.

The word ῥαχίτις, *morbus spinalis*, having been suggested by a friend, he accepts it as expressive of the affection of the spine, as distinctive, and as near to the vernacular, either by accident or possibly by corruption of a physician's term.

He says that "the rickets" is the word common for the disease in the West of England, in London, and in the Southern and Midland counties, but that in the North it is hardly known.

In a thesis by Whistler (said to have been published at Leyden in 1645)

* This word has been derived from a verb in use in Dorsetshire—"rucket" (= to breathe laboriously); or from "rick" (= elevation or hump, as "hayrick"); or, according to Trousseau, from a Norman word—"riquets," applied to deformed persons, itself said to be derived from Alberiquet, dim. of Alberic, a dwarf in Gothic mythology (see Koch's paper, 'Arch. f. Gynæcologie,' 1885, and Virchow's criticism in the same year, 'Arch. f. Path.,' vol. cii). But the true derivation, according to Skeat, is from *wrikken*, to "wrest," to twist "awry;" whence the phrases a "ricked" ankle, a "rickety" chair.

In framing a Latin word Glisson bore in mind the following excellent rules: "(1) Ut nomen morbi notabilem aliquam ejus conditionem comprehenderet; (2) Ut id satis esset distinctum ab aliorum morborum et symptomatum nominibus; (3) Ut esset satis familiare, pronunciatur facile, memoriæ quoque accommodatum, non nimis longum, neque operosius decompositum."

The vernacular term in French is *chastre* (castrum), and in German *doppelte Glieder*.

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the complaint was spoken of as *morbus puerilis Anglorum*, and probably this led foreign writers to call it *morbus Anglicus*. It was hence supposed to have spread from England to the Continent; but there is little doubt that it had existed among children from time immemorial on both sides of the Channel, and it is as common in Holland and Germany at the present day as in England.

Only few and doubtful allusions to the disease are to be found in older works, but there is an antique statue of Æsop, figured by Stiebel in his monograph published in 1863, which is said to exhibit the deformities characteristic of rickets.*

In Grant's 'Observations on the Bills of Mortality' (1662), it is noted that "the rickets" first appeared in the returns for London in the year 1634, when fourteen deaths were ascribed to it. In 1658 the number was 476 (for the disease was better recognised), and in 1659 it was 441.

Definition.—Rickets is not merely a disease of the bones. It is like Rheumatism and Gout, a general, or, to use a disused term, a "constitutional" disease. But just as gout and rheumatism find their most marked expression in the joints, so does rickets in the bones.

It may be defined as a disease attending the period of development in the earlier stage of childhood, affecting the whole body, but particularly recognised by its effect on the growth of the bones, leading to enlargement of the ends of the long bones and deformity of the chest, pelvis, spine, skull, and limbs. In its extreme form it affects the whole skeleton of the child, but it often begins in some particular region, and it may remain limited to the chest, or to the head, or to some of the limbs, at least so far as its more obvious manifestations are concerned. But it is far from being confined to the skeleton. Its symptoms, as we shall presently see, extend to the organs of nutrition and secretion. It is a general disease or cachexia, probably of diatetic origin and certainly not contagious or specific. For some reason the particular form of malnutrition which shows itself in rickets can only be produced at a certain age. After two or three years old not only does no form of starvation lead to its characteristic changes, but when present it gradually passes away of itself.

Pathologically we may perhaps compare it best with Scurvy.

Anatomy: the limbs.—The earliest sign of rickets is swelling of the epiphyses of the long bones. This is particularly marked at the wrist; the radius and ulna form a flattened swelling which contrasts with the small hand. Something similar may be seen in the case of the ankle. The joint of the knee appears as a hollow or depression between the projections of the articular extremities of the femur and of the tibia, and hence the expression "doubling of the joints," which has sometimes been used as synonymous with rickets. If the affection advances the limbs become curved. In the forearm the bones almost always bend so as to be convex towards the extensor surface; in the upper arm the character of the distortion is less uniform. In the thigh the rule is that the femur is arched, with its convexity looking forwards and outwards. The knees may thus be thrown far apart, and the patient becomes bow-legged. The shape assumed by the tibia

* A cast of this statue from the Villa Albani at Rome has been lately added to the collection of antique casts in the South Kensington Museum (No. 229). It represents rather the effects of extreme angular curvature of the spine than of true rickets. Two of the children in Glisson's frontispiece have cyphosis, which is not obviously rachitic.

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and fibula varies in different cases. They often carry downwards and inwards the curve formed by the femur on each side, so that the ankles meet one another, although the knees do not; but in other instances they are themselves bent with the convexity outwards or inwards; so that in the latter case the feet are widely separated. Sometimes each leg presents a sharp angle, projecting forwards at the junction of the middle and the lower thirds, and producing with the narrow shaft what is called the sabre-shaped tibia.

These various deformities are the natural result of the bones yielding to the traction of the muscles and the weight of the body. They are, in fact, so soft that very little force is required to bend them, as can easily be demonstrated after death. The curves in the forearms and upper arms are probably due to efforts made by the child to raise itself by laying hold of fixed objects with its hands, and to other like movements: there is often a marked angle at the insertion of the deltoid into the humerus. The more common distortions of the bones of the thighs and legs seem to be caused by the weight of the body in the erect posture; but the angular bend which is found just above the ankles is probably due to pressure transmitted to the tibiæ from the insteps and feet in crawling about upon the floor, a favourite mode of progression with rachitic children.*

Another effect of the softening of the bones is that a very slight accident suffices to partially break them. Such "green-stick" fractures, as they are called, may be caused by abrupt movements; sometimes several of them are seen in the same child. Their effects, of course, complicate and alter in various ways the more regular distortions resulting from the disease, and all the more because, interfering but little with the movement of the affected part, and giving rise to no marked increase of pain, they are very apt to escape notice until a large quantity of callus has been thrown out.

The thorax.—Of far more importance as regards the patient'a health, though perhaps less conspicuous to the eye of an untrained observer, are the changes produced by rickets in the shape of the chest. Here, again. the first indication of the disease is an enlargement of the growing ends of the bones; namely, of the ribs just where they join their cartilages. The consequence is the formation of a series of little nodules, which can be easily felt, and may even be seen through the integuments, arranged in a vertical line, slanting outwards as it passes downwards on each side of the sternum. This "beading" of the ribs, as it is termed, is sometimes the only discoverable sign of rickets which a child may present; it must therefore be carefully looked for whenever the existence of this disease is suspected. But, further, there is in most cases a more or less considerable alteration in the form of the thorax, by which its capacity may be greatly reduced. If one watches a healthy child who is suffering from extreme dyspnœa dependent on obstruction of the larynx or trachea, one may observe that at each inspiration the middle parts of the ribs are forcibly dragged inwards. This is especially the case with those ribs which lie towards the base of the chest on each side, and cover the lungs; for the effect is not to be seen where they overlie the solid organs, the heart, and the liver. The cause of this is that the ribs are unable to resist the atmospheric pressure when they are no longer supported by the counter-pressure of air entering the lungs freely from the throat. Now, in rickets, it would seem

* It has been objected that in stillborn fœtuses, believed to be rachitic, similar changes have been observed in the shape of the limbs. But there is no evidence that the distortions are identical with those of ordinary rickets.

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that the mere elasticity of the lungs is sufficient to turn the scale and to prevent the lateral portions of the softened ribs from moving outwards when the child draws its breath; or it may be that this result is brought about by trifling and transitory affections of the bronchial tubes. In either case the effect is not transitory, as it would be under normal circumstances; but there arises a persistent flattening, or even a depression, of the chest walls. This generally runs, as a vertical broad and shallow groove, downwards and outwards from just below the fold of the axilla on each side; as it approaches the margins of the costal cartilages it forms an angle and slopes away to each side, now lying almost parallel with the diaphragm. Or one may describe two sulci—the one nearly perpendicular, the other more horizontal—meeting at an obtuse angle near the base of the xiphoid cartilage. The vertical groove is generally said to be formed by the ribs themselves, outside their beaded ends. But Dr Gee ('St Barth. Hosp. Rep.,' vol. iv) has pointed out that the beads sometimes occupy the bottom of the groove, and that in exceptional cases they may lie to its outer side, so that, in fact, it corresponds with the cartilages only and not at all with the bones. One result of the depression of the ribs is that the higher abdominal viscera are pushed out from below the ribs; the liver projects beyond the costal margins more than in a healthy child; and, as the intestines are commonly full of gas, the belly becomes protuberant and contrasts strongly with the narrow chest. Another effect, according to Sir William Jenner, is the production of a white friction-patch on the surface of the heart, just above the apex of the left ventricle, where the fifth rib presses on it (see his well-known Lectures in the 'Medical Times' for 1860). Yet another is an increase in the antero-posterior diameter of the thorax; the sternum is pushed forwards, and the dorsal vertebræ form a rounded curve.

These changes together constitute what is commonly called the *pigeon-breast*. They are almost always associated with the presence of emphysema in the anterior edges of the lungs, beneath the projecting sternum; while in correspondence with the flattened ribs, one may often notice a collapsed condition of the inferior edges of the lungs, and even of parts of their lateral surfaces. Another feature of the disease is that the clavicles are much more bent than in the normal state, and carry the shoulders further backwards, with the effect of increasing the apparent prominence and narrowness of the upper part of the chest.

The vertebræ.—The spinal column in a rachitic child becomes gibbous, curved with the convexity backward. In contrast with this *cyphosis* of the dorsal spine, the cervical and the lower lumbar vertebræ have their natural forward curves exaggerated (*lordosis*). The part especially affected is, as Mr Lane has shown in the 'Guy's Hospital Reports' (vol. xlii, p. 319), the junction of the thoracic and lumbar vertebræ, the eleventh and twelfth dorsal, and the first and second lumbar. The sacrum also becomes more flexed on the iliac bones, and its promontory moves downwards and forwards.

In the *pelvis* various deformities occur, but these are not obvious during the acute stage of the disease, and are only important because in females they may permanently narrow the cavity and obstruct parturition. In most cases the brim assumes an hour-glass or oval shape, the pubes being approximated to the sacrum, but the opening may be triangular, and the symphysis pubis rostrate.

The face is peculiarly backward in its development; the jaws remain narrow, and *dentition* is late and irregular. It is not uncommon for a

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rachitic infant a year old to have cut none of its teeth; and when two or more of the incisors have appeared before the commencement of the disease they are sometimes without successors for several months. The teeth themselves are imperfectly formed; their enamel is defective; in a year or two they turn black and break off, or fall out. Dr Gee has pointed out that the second dentition is also delayed.

The cranium.—In marked contrast with other parts of the skeleton, the skull is disproportionately large, so much so that until recently it was believed to be larger than in healthy children of the same age. Ritter von Rittershain has, however, shown by accurate comparative measurements that the enlargement is generally only relative.

The rachitic skull is square in shape, both looked at from above and from in front. The eminences which represent the centres of ossification of the frontal and parietal bones remain very prominent.

With regard to the state of the brain we find some discrepancies of statement among different writers. Trousseau maintains that the softness of the cranium allows of the more easy development of the hemispheres, and so accounts for the possession by rachitic children of intellectual faculties in advance of their age—a fact, if it be a fact, which it would be more reasonable to attribute to the habits induced by their unfitness for exertion and their habitual association with adults. Dr Gee thinks that the growth of the brain is really dwarfed, like the rest of the body, and that fluid is commonly effused into the ventricles to fill up the empty space within the skull. But it seems to be certain that, in many cases in which the head appears to be increased in size, there is no excess of fluid. On the other hand, hydrocephalus is of frequent occurrence as a complication of rickets, and the affection known as hypertrophy of the cerebral substance is sometimes met with (vol. i, p. 691).

The form of the cranium is also altered. It has been described as elongated; but in reality it is more often square, and flattened on the top, in consequence of the fact that the fontanelles fail to close at the proper time. Clinically, this is the most important of all the symptoms of the disease, except the beading of the ribs. The principal fontanelle not uncommonly remains open up to the age of three years or even longer; moreover, there is often separation of the bones, where they meet to form sutures; their margins, being the growing parts, are generally more or less thickened; so that sometimes one can feel a distinct ridge along the vertex and even down the front of the forehead.

There is often an irregular thinning of the occipital bone—a condition first described by Elsässer, in 1843, as *craniotabes* (cf. vol. i, p. 303). The way to detect it is to grasp the head with the two hands, and to make very gentle but firm pressure with the tips of the forefingers over all parts of the surface of the bone in succession. One may then find that certain small spots, generally near the lambdoidal suture, yield and become indented, just as though the osseous tissue were replaced by a piece of cardboard.

The growth of the body generally is retarded in rickets; a child two years of age may be taken for not more than six months old; a boy of twelve may be no taller than he ought to have been at three. Among forty-two cases in which Ritter von Rittershain (1863) made careful measurements at ages between four months and three years, there was only one in which the length of the body was not from one and a quarter to two and a half

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inches below the mean length in healthy children at the same ages. Rickety infants, however, are not infrequently fat, and sometimes excessivly so.

Histology.—The microscopical changes in rickets are very interesting. If with a strong knife one cuts through a rib and its cartilages, across the plane of the union between them—or if one divides the end of a long bone, so as to expose on the face of the incision the junction between its shaft and one of its epiphyses—certain deviations from the normal appearances are at once obvious, even to the naked eye. The "zone of proliferation" of the cartilage ought to be a well-defined, straight, narrow, bluish-white line, perhaps one sixteenth of an inch in width; and the yellow "ossifying zone" beneath it ought to be still narrower. Instead of this, in a rachitic bone the zone of proliferation is considerably thickened, reddened, and of a soft spongy texture. Moreover, the meeting line between them is most irregular and sinuous, with promontories and islands of bone and medullary spaces projecting far into the cartilage. Rindfleisch aptly sums up these changes by saying that the processes which prepare the way for the conversion of cartilage into bone are morbidly accelerated, without the actual ossification keeping pace with them. So, again, beneath the periosteum, instead of an almost inappreciable quantity of embryonic tissue, there is in rickets a soft, red, vascular layer, perhaps one twelfth of an inch thick, which has been compared with the substance of the splenic pulp. It sends processes into the superficial vascular canals, and often has embedded in it numerous minute osseous processes, which tear away with it from the shaft, leaving the latter rough. The whole of the interior of the bone also, including the medullary cavity, is unnaturally red and vascular.

In thin sections, and with the aid of a microscope, the exact nature of the affection can be traced more minutely. The broad, bluish-white zone contains long columns of proliferated cartilage-cells, thirty or forty deep. But, unlike what occurs in the normal process of ossification, these cells can easily be seen to be directly transformed into stellate bone-cells, each of which, however, remains surrounded by a delicate ring, corresponding with the former cartilage capsule. Rindfleisch says that the homogeneous chalky appearance of this "cartilage-bone" enables one to recognise it with the naked eye, even when it is embedded in normal osseous tissue.

The *chemical* composition of the bones in rickets has been several times investigated, and the proportion of inorganic to organic matter has been found much below what is normal. The analysis of Friedleben, however, published in 1860, made the percentage of earthy salts from 33 to 52, which is considerably higher than that given by earlier inquirers, although much less than the percentage of 63 to 65 obtained from the bones of healthy children.

Visceral changes.—Following Sir William Jenner, some English observers attach considerable importance to a change in the liver and spleen and lymphatic glands, which he described as an "albuminoid infiltration."* Dr Dickinson has investigated the microscopical characters of this affection, which he finds to be an overgrowth of the fibrous tissue in the portal canals of the liver and in the trabeculæ of the spleen respectively, with some excess of cellular elements also. The organs, he says, feel hard, dense, and elastic; the liver shows yellowish acini, each surrounded by a thin pinkish or grey line; the spleen, which may be so large as to extend below the umbilicus, is of a deep red or purple colour, besprinkled with smooth, white spots, or mottled into a pale buff. The lymph-glands are moderately increased in

* See 'Medical Times and Gazette' for 1860, vol. i, p. 259.

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size, tough, white, and opaque. Dr Gee states that when rickety children die with an enlarged spleen, its appearance differs in no respect from that of the spleen of ague, or of inherited syphilis, or of cachexia due to unknown causes. He thinks that the affection is really a result not of the rickets, but of the general state of ill-health which caused the rickets (St Barth. Hosp. Rep.,' vol. iv, pp. 69, 265). Dr Dickinson connects it more particularly with emaciation and anæmia, others with congenital lues. It is certainly a rare complication, and, when present, usually subsides under treatment.

General symptoms.—Before any changes in the bones can be discovered, rachitis may be ushered in by sickness, diarrhœa, and tumefaction of the abdomen, with languor, drowsiness, loss of appetite, and febrile disturbance. More often these symptoms accompany or follow the osseous disease. In any case it is of practical importance to remember when called to a child with croup or bronchopneumonia that the case is often one of advanced rickets, although the mother may have thought the child to be in good health before.

Some minor symptoms often lend considerable aid in diagnosis. One is a peculiar *restlessness* at night, which causes the child, even in cold weather, to kick off the bedclothes, as often as it is covered over. Another is the outbreak of profuse *perspiration* on the head and neck and the upper part of the chest, especially during sleep. Elsässer laid much stress on this in connection with his craniotabes. A third sign of rickets is extreme *sensitiveness* of the body and limbs, so that the child lies motionless, and dislikes being moved, or handled; and at once begins to cry if lifted by the arm-pits and tossed up and down. The tenderness appears to be partly in the bones and periosteum, but Sir William Jenner and Dr Gee have noticed that gentle pressure upon the loins or abdomen is sometimes no less painful.

The muscles are soft and flabby, and more or less wasted. In severe cases the child is unable to walk or even to stand, even though he may have been on his feet for some time before he became rickety, so that Dr Gee speaks of "pseudo-paraplegia" under such circumstances. Jenner relates the case of a girl, six years old, who could neither change her position in bed without assistance nor lift her arm an inch from the surface on which it lay; even at a later period, when she had greatly improved, she was obliged to be tied into a chair with a pillow at its back to support her head; and if the head fell forward the nurse had to raise it for her. She afterwards recovered so as to walk without assistance.

The rachitic process, like that of scurvy, is unaccompanied by fever. Any rise of temperature appears to be due to inflammatory complication.

Course.—Rickets generally runs a somewhat chronic course, but subsides, under favourable circumstance, at the end of a year or two. Some writers, however, have described an acute form of the disease. Senator records the case of a child, four months old, who became feverish, and in whom the epiphyses of several of the long bones of the limbs were swollen and very tender, but without redness; the affection subsided entirely in about six weeks.* Moller published three cases of "acute rickets" so early as 1859.

A remarkable form of what may be called "acute rickets associated

* Fürst is said to have recorded instances of this kind in 1832; but on referring to them, it appears that his were cases of rapidly fatal multiple abscesses of joints occurring in infants.—C. H. F.

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with purpura"—or scurvy (cf. *supra*, p. 659)—has been described by several authors: Dr Cheadle ('Lancet,' 1878), Dr Gee (who called it "osteal or periosteal cachexia," 'St Bart.'s Hosp. Rep.,' vol. xvii), Mr Thomas Smith ('Path. Trans.,' vol. xxvii), Dr Goodhart ('Dis. of Children,' p. 556), Dr Samuel West ('Clin. Trans.,' xxi, p. 209), and Dr Barlow, whose excellent account of eleven cases with two autopsies will be found in the 'Med.-Chir. Trans.,' vol. lxvi, p. 159. See also Dr Eustace Smith's remarks on the point in his 'Disease in Children,' p. 255.

Event.—After recovery from rickets, the bones lose their soft spongy character, and become denser and harder than natural. The articular ends are no longer enlarged, probably because they have been overtaken in their growth by other parts, so that the normal proportions are restored. Many of the deformities which are so conspicuous in young children seem slowly to disappear, at least when they do not exceed certain ill-defined limits. It used to be traditionally taught at the Hospital for Children in the Waterloo Road that although tibiæ which were laterally curved might become straight in the course of time, a similar improvement never occurred when they were sharply bent with the convexity forwards near the ankles, in the manner which is attributable to crawling on the floor (p. 735). The pigeon-breast is very generally permanent; and in too many cases the limbs, as well as the trunk, remain distorted for the rest of life. Even when there are no striking alterations in the shape of the bones, one can often recognise the fact that a person was rickety in childhood by his short stature, thick-set body, and large protuberant head.* Such persons are often erroneously supposed to have suffered from hydrocephalus.

Sequelæ.—That rickets is not a mere local disease of the skeleton is proved by its being frequently associated with disorders of distant parts either as complications or sequelæ. These are laryngismus stridulus, tetany, trismus, the slighter form of convulsion called carpo-pedal contraction, and more severe infantile eclampsia (cf. vol. i, p. 711); bronchitis, and broncho-pneumonia with collapse of the lungs; and lastly, an obstinate form of intestinal catarrh with severe diarrhœa.

Age.—Rickets is a disease of early childhood, but all observers are not agreed as to the exact time at which it is most apt to be developed. The common belief is that this period is from the sixth month to the eighteenth, or the end of the second year; corresponding, in fact, with the first dentition. If we inquire closely into cases which are said to have begun later than this, we usually find grounds for suspecting that a slight form of the affection had existed for some time previously, although it may recently have undergone a more rapid increase. Dr Gee, who collected 635 cases, is disposed to agree with von Rittershain in thinking that rickets does not often begin after the end of the first year. The latest case that he had himself observed was one which seemed to have begun at twenty months. The child, who showed considerable beading of the ribs, had cut the first tooth at six months, and at twelve months it had been weaned, and had walked; six weeks before it came under Dr Gee's notice it had begun to get weak in the legs and loins, and during the last three weeks it had sweated much. Even in that instance the absence of the disease at an earlier period was merely a matter of inference; and there can be no doubt

* This is the type described by Victor Hugo in Quasimodo, the hunchback of Nôtre Dame, who must certainly have had rickets in childhood.

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that most statistical tables are so largely open to the same objection that they have little value.

Some have supposed that rickets may develop itself in young adults, but this idea appears to have been based upon erroneous views as to the nature of certain cases of spinal curvature and of articular disease. Indeed, it seems scarcely possible for the morbid process described (p. 738) to arise when the process of ossification has been almost completed; and in future no such case of "late or retarded rickets" would be accepted without full details as to its histology.*

It is certain that rickets may occasionally be seen before the sixth month. Dr Gee speaks of unquestionable beading of the ribs in infants only three or four weeks old. In such cases it seems probable that the starting-point of the disease was in intra-uterine life; but whether it can ever be recognised at the time of birth is very doubtful. A few supposed instances of such an occurrence in the stillborn fœtus were recorded by Jules Guérin in his 'Mémoires sur les Caractères du Rachitisme' as early as 1839, but Urtel (1873) and Eberth (1878) have each found that the histology of the affection described under that name is altogether different; for the process by which the epiphysial cartilages normally undergo conversion into bone was arrested at a much earlier period, before the cartilage-cells had begun to proliferate and to arrange themselves in vertical columns. The most conspicuous character of such cases is the extremely stunted form of the limbs. Cases of so-called "fœtal rickets" have been published by Dr Felix Schwartz of Vienna ('Med. Jahrb.,' 1887), Dr Thomas Barlow and Mr Shattock ('Path. Trans.,' 1881, pp. 364, 369, *ibid.*, 1884, and 'Clin. Trans.,' vol. xxi, p. 291). The condition is probably allied to cretinism.

Sex.—With regard to the relative liability of boys and girls to the disease, authorities differ. Guérin, in Paris, recorded 148 cases in male and 198 in female children; von Rittershain, at Prag, 290 male to 231 female cases; Brunnicke, at Copenhagen, 108 male and only 55 female; and Dr. Ritchie, at Manchester, 128 male and 91 female. The general result would be about 53 to 47 in the 100; a difference so small that we may probably conclude that rickets is equally common in the two sexes.

Climate.—The frequency of rickets varies in different countries. It is greater where the climate is damp and cold than in the Mediterranean regions or the tropics; but the statistical results hitherto collected are not exactly comparable with one another.

Dr Gee found rickets in no less than 30·3 per cent. of all children under two years old brought to him at the hospital in Great Ormond Street in 1867. It is far less common among those who live in the country than among the inhabitants of crowded cities, where children are apt to get too little light and air.

The geographical distribution in England is approximately given in an interesting report drawn up by Dr Isambard Owen on the returns obtained by the Collective Investigation Committee ('Brit. Med. Journ.,' Jan. 19, 1889, p. 114). Rickets was common in London, in the great industrial districts of the Black Country, of Lancashire and the West Riding, and of the Tyne and Tees; also in the mining districts of South Wales, in Glasgow

* These conditions seem to have been fulfilled by a case of rickets in a boy of eleven, shown by Dr Drewitt ('Path. Trans.,' xxxii, 385), and examined after death by Drs Abercrombie and Barlow. Mr Clutton and Mr Davies-Colley have published similar cases at about the same age. But none of these patients were over puberty.

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and its neighbourhood, in Edinburgh, Dundee, and other large towns in Scotland, and in the only large Irish towns, Dublin and Belfast. It was rare in rural and sparsely-populated districts: in the Scottish Highlands, in Cumberland, North Lancashire, and the agricultural districts of Yorkshire and Scotland, in Derbyshire, North Wales, and generally in the southern, eastern, and south-western English counties. In Ireland towns as large as Cork, Limerick, Londonderry, and Galway are almost entirely free from rickets.

Efficient cause.—Many attempts have been made to find a definite exciting cause for the disease. One observer endeavoured to trace it to prolonged lactation, another to premature weaning; Guérin in 1839 supported the latter view by some experiments on young puppies, which he deprived of their mother's milk, and fed with meat; the more recent investigations of Tripier, however, have shown that although animals treated in this way become sickly and die, they are not really affected with rickets.

One cause of the malady is deficiency of phosphates and other earthy salts in the food. Chossat and Alphonse Milne Edwards succeeded in producing curvature of the bones in pigeons and dogs by cutting off the supply of their nutritive salts; but Friedleben has since found that even when atrophy of the osseous tissue is thus induced, the changes characteristic of rickets are wanting. The effect of insufficient and indigestible food in producing rickets is well shown by Dr Norman Moore in his thesis on the subject (1876) with two hundred cases in illustration.

Although rickets often occurs in children who have been improperly fed on patented foods, there is no reason to believe that starch is responsible for more than, perhaps, some of the diarrhœa. The fact is that feeding on starch without a due proportion of proteids and fats and salts, such as is given in milk, is a kind of starvation. (See an excellent paper by Dr Cheadle in the 'Brit. Med. Journal' for Nov. 24, 1888.)

Rickets is not hereditary; but it has been attributed to *other conditions in the parents*, such as syphilis, phthisis, emaciation, anæmia, and even old age. Sir William Jenner thinks it very doubtful whether impairment of a father's health has any influence in producing rickets in his children. Von Rittershain thought that he traced the disease to the presence of tubercle in the father more often than in the mother. But the truth is that among the poor it is impossible to isolate causes of this kind; a husband's illness may deprive the wife of nourishment, throw much heavy work upon her, and in many different ways render her likely to bear weakly infants. So, again, even when the parents of a rickety child have, one or both of them, had rickets in early life, it is very doubtful whether the disease is really transmitted. A point of great importance, on which Jenner has laid stress, is that the first child of a family, or even the first two or three, may be found free from rickets, where later ones are affected by it; and, again, that if once a woman has borne a rickety infant, those that follow are almost sure to have the same disease. This is due, not only to the progressive enfeeblement of the mother's health by repeated child-bearing, but also, among the poor, to the overcrowding and deficiency of food and clothing, which are implied by a large family; and perhaps among the middle classes to the way in which children are sometimes kept indoors, when there is but one nursemaid for several of them.

The relation of rickets to *tuberculosis* requires further investigation by pathologists untrammelled by previously formed opinions. Jenner, although he contrasts the two "diatheses," adds that rickets does not by any

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means exclude tubercle. Dr Eustace Smith says that rickets never occurs in children in whom "the tubercular disposition" is well marked; and Trousseau held rachitis and tuberculosis to be opposed diatheses. In contradiction to the statements of von Rittershain just quoted as to the inheritance of rickets from tuberculous fathers, Jenner refers to a table made for him by Dr Edwards, which appeared to show that phthisical parents are *less* likely than others to have rickety children.

The relation of rickets, and particularly of craniotabes, to *congenital syphilis* was discussed at the International Medical Congress of 1881 ('Trans.,' vol. iv, p. 35) by MM. Parrot, Guérin, and Bouchut, of Paris, by Dr Rehn, of Frankfort, and by other pathologists. See also on this point an admirable paper with tables, by Drs Lees and Barlow, in the 'Path. Trans.,' vol. xxxii, p. 323.

There can be little doubt that M. Parrot's view of the ætiology of craniotabes was wrong, and that congenital syphilis when present is an independent complication of rickets.

Pathology.—It was once supposed that the immediate cause of the changes in the bones in rickets was the action of lactic acid, dissolving out the lime salts from their substance. The acid was said to have been detected not only in the bones themselves, but in the urine; and it was supposed to be formed in excessive quantity in the alimentary canal from milk and other articles of food. Moreover, chemists stated that more than the normal amount of phosphate of lime was excreted by the kidneys. The modern investigations on the histology of the disease have rendered such a theory untenable; but recently another hypothesis has been framed, in which the acid plays a different part. In 1871 Dr Wegner, of Berlin, in the course of some experiments upon young animals with minute doses of phosphorus, found that if, while administering the poison, he withheld lime salts from the food, there arose an affection of the bones precisely like rickets, as it is seen in the human subject. He supposed that the phosphorus was a stimulant to the osseous tissue. Heitzmann has since stated that lactic acid is capable of acting in the same way. The hypothesis, therefore, as given by Senator, is that the disease is the combined result of the irritant influence of that acid upon the growing bones, and of a deficiency of phosphate of lime, consequent either on there being too little of it in the food, or on its being carried away through the bowels by diarrhœa. But at present this theory seems to rest upon too slender a foundation of facts, almost as slender as that which supports the theory of lactic acid as the cause of rheumatic fever. The two theories seem, indeed, to be mutually destructive.

Rickets is not uncommon in the lower animals. The lion cubs in Dublin were all born with cleft palate, which prevented their sucking the dam's teats until she was properly supplied with bones as well as flesh. Mr J. B. Sutton has reported some striking cases of rachitis from the Regent's Park Gardens, and finds that in animals the disease is cured by means of oleum morrhuæ and ground bone-earth.

Diagnosis.—The recognition of rickets is very easy when it is fully developed. As a source of fallacy in regard to the craniotabes of Elsässer, may be mentioned here the case of cerebellar tumour (referred to in vol. i, p. 611), in which a somewhat similar thinning of the occipital bone was observed.*

* I must confess that I have sometimes been in doubt as to what constitutes "beading" of the ribs, as distinguished from the slight roundness of their ends which is normal.—C. H. F.

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The only real difficulty is as regards early cases in young children; and here the mistake usually made is not to look for the signs of rickets. This should never be omitted in cases of infantile diarrhœa, of laryngismus stridulus, or of bronchitis in infants.

Prognosis.—Most writers express doubt as to whether death is ever caused by rickets alone, apart from complications. Dr Eustace Smith, however, says that he has seen it directly fatal, with extreme dyspnœa and lividity. As a rule, if the child succumbs, it is to diarrhœa, or bronchitis, or laryngismus stridulus; perhaps to croup or pneumonia, or to one of the exanthems.

If left unmodified by treatment, a case of rickets is more grave in proportion as the child is younger at the time of its commencement. It is therefore extremely important to be on the look-out for it whenever an infant at the time of the first dentition begins to fail in health, or suffers from any trifling disorder, such as relaxation of the bowels. For, as follows from what has been stated in regard to its ætiology, rickets is eminently a disease that can be prevented. It is also one that can be cured.

Treatment.—The chief thing is to attend to the food. When an infant is suckled, the breast should during the first six weeks be given every two hours, except from 11 p.m. to 5 a.m., during which interval the mother or the wet nurse should be allowed to sleep; at a later period every three hours, or still less frequently. It is very wrong to let a child lie asleep with the nipple in its mouth, although nothing is more common than for it to be kept at the breast all the night while the mother herself is in a sound slumber. If a baby does not thrive, one cause for it may be that its mother's milk is insufficient in quantity or too poor. According to Dr Eustace Smith, it is a sign that this is the case when the infant falls asleep while sucking; or one may notice that it sucks at its thumbs until they become raw. If something in addition to the breast-milk is required, one may employ cows' milk or asses' milk, sweetened a little, and perhaps diluted with water, according to the age. Neither biscuit powder nor any other farinaceous food should be administered to very young infants. The secretion of saliva appears not to be established, under normal conditions, before the third month; and it is believed that up to that time all starchy matters pass through the intestines unaltered, and are discharged with the fæces. But it must be confessed that some well grown children are seen whose parents had been ignorant of this rule, and had brought them up in direct opposition to it. Liebig's or Melin's malted food may safely be used mixed with milk, even a week or two after birth. Some infants, however, will not take it; and there is no doubt that what suits one perfectly may not do for another. Swiss milk and the other condensed preparations of milk are often given to young children. They undoubtedly fatten rapidly; but the more important tissues seem not to be well sustained, and infants so brought up are apt to succumb if attacked by diarrhœa or by other ailments. Probably these effects are attributable to the quantity of sugar contained in it: but one also feels a natural prejudice against the attempt to preserve artificially a substance so liable to decomposition as milk; and the same doubt would apply to Nestlé's compound.

After six months it is advisable that the mother's milk should be supplemented either by farinaceous food or by Liebig's maltine. At eight

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months, a little mutton- or chicken-broth, or beef-tea, may be given with advantage. At ten or twelve months the child should be weaned.

When a feeding-bottle is used, the most extreme care is required to keep it and its tube clean, so that it may not turn the milk which is put into it sour. It should be scalded out every time it is employed; and the tube should be always kept in water.

No one who had not witnessed it would believe how utterly wrong is in most cases the management of young children belonging to the lower classes. At a very early age they are allowed to have bacon, fried fish, pickles, potatoes, and beer. If brought up by hand they perhaps are given cornflour, or some other substance which is little better than pure starch; and it is often made up with water instead of with milk. If suckled, they are not weaned until they are eighteen months or two years old; but long before this they sit with their parents at meals, and share what they eat.

A rickety child after weaning requires certain precautions to be taken, which are unnecessary for others. Not only must its food be nutritious and digestible; it must also be easy of mastication, if the teeth are few in number or decayed. Thus one often has to direct that all the meat should be finely powdered in a mortar, and that the potatoes should be mashed, and all the lumps be carefully picked out.

A rachitic child should be kept lying down, and should not attempt to walk, so long as the bones are soft. Splints projecting below the feet may be used for the purpose of rendering such attempts impossible; but mechanical appliances seem to be of little service in straightening the spine or the limbs. Jenner found that when the ribs were inclined to yield, a well-adjusted bandage round the abdomen was useful by restraining the descent of the diaphragm.

At night the child should sleep on a hair mattress, and if its head is tender and inclined to perspire, it should have a horsehair pillow made with a hole in the centre so as to remove all pressure from the occiput. Dr West has seen this give quiet sleep for the first time for weeks. A thin linen nightcap may also be worn, which can be changed two or three times if necessary.

Bathing is of considerable importance. The child should be sponged with warm soap and water once or twice a day, or, according to the season, with tepid or even cold water, in which sea-salt may be dissolved. Dr West recommends tan baths, made by adding to the water a decoction of oak bark; the receipt is to take three handfuls of the bruised bark, and boil it in a linen bag in three quarts of water for half an hour.*

Drugs.—Among medicines the most valuable is cod-liver oil. It should be given even when the bowels are relaxed, unless it causes an increase of diarrhœa, which is not often the case. Small doses of ʒj or ʒij are more efficient than larger ones, which are apt to cause nausea and diarrhœa. Steel wine and quinine are also useful, and the *liquor ferri perchloridi* is often prescribed with advantage. Dr Eustace Smith has seen marked benefit result from Alison's prescription of tannic acid in doses of half a grain to a grain twice or thrice daily. If an occasional aperient is necessary, a little castor-oil or a powder of rhubarb and soda, or the syrup of senna, may be given.

* Beside the discussion on rickets in the International Congress of 1881, referred to above, an instructive debate will be found in the 'Pathological Transactions' for the same year (vol. xxxii, pp. 312—404). It was introduced by the author of this work and continued by Dr Norman Moore, Mr Haward, Dr Dickinson, Mr Parker, Sir William Jenner, Mr Hutchinson, Mr Lucas, the late Dr Baxter, Mr. S. Watson, and Dr Goodhart.

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Lime-water should be given if there is proof that the milk which the child takes becomes curdled in its stomach.

Jenner forbids the administration of repeated doses of mercury to rickety children; and he says that when they are attacked with acute diseases leeches should never be applied, nor antimony be given.

MOLLITIES OSSIUM.*—In the middle of the last century instances of extreme deformities produced by softening of the bones were recorded, almost in the same year, by three observers, Duverney, Morand, and Pringle; and the names of two of the patients—the Marquise d'Armagnac and Madame Supiot—have become historical. Similar cases have since been met with from time to time, but very rarely, except in certain districts bordering upon the Rhine, where, according to Senator's statement (in 'Ziemssen's Handbuch'), they have been somewhat less infrequent.

Sex and age.—Mollities ossium is far more common in women than in men; among 145 cases quoted by Mr Durham, in the 'Guy's Hosp. Reports' for 1864, thirteen only occurred in males; and one may suspect that the disproportion would have been still more marked if they could have been critically sifted. A case in a young man was reported by Dr Burgess in the 'London Medical Chronicle' for October, 1888.

The *age* at which it is least uncommon is between twenty-five and thirty-five; a few of the patients are said to have been under twenty years old, and a few over fifty. In a well-marked case which the editor saw with Dr E. O. Day, the disease began when the patient, a girl, was about sixteen. Dr Rehn has recorded one in an infant ('Internat. Med. Congr.,' 1881, vol. iv, p. 59); so also has Dr Bury, of Manchester ('Brit. Med. Journ.,' 1884, vol. i, p. 213).†

Ætiology.—A definite cause can seldom be assigned. Habitual exposure to cold and wet, as from living in a damp house, has sometimes been supposed to give rise to it, but to many instances such an explanation could not be applied. The patients have often been well fed and in easy circumstances. Sometimes it has been noted that they had been affected with rickets in childhood, but in all probability this was a mere coincidence. The occurrence of utero-gestation appears to play an important part in the ætiology of mollities ossium; in ninety-one of Litzmann's and Durham's 132 cases it began during pregnancy, or shortly after childbirth.

A case of extreme distortion of the limbs, thorax, and pelvis, recorded by Dr W. J. Webb, of Chicago, in the 'New York Medical Journal' for March 21st, 1885, occurred in a man some of whose brothers and sisters had symptoms more or less like his own.

Symptoms and course.—As a rule, the earliest symptom of the disease consists of pains in the trunk or in the limbs, which seem to vary in character in different cases, and which may appear to wander or fly about from part to part, so that they are usually supposed to be "rheumatic." The next thing may be that one of the bones breaks without cause, or during some slight effort, as in getting out of bed. Or a progressive change in the figure may be noticed, the body becoming short or stunted, the back rounded and distorted laterally, the neck stooping so that the chin may be

* *Synonyms.*—Malacosteon—Osteomalacia—Le rachitis des adultes.

† See moreover the report on Dr Goodhart's case ('Path. Trans.,' vol. xxxiv, p. 201), and Mr Davies-Colley's (*ibid.*, vol. xxxv, pp. 285, 292); also Mr Thos. Jones's 'Diseases of the Bones' (1888).

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brought close to the sternum. There is extreme lassitude with disinclination for any kind of muscular exertion. The patient waddles in walking, and has to help herself with sticks or crutches. Presently she is obliged to take to her bed. She now becomes a most pitiable object. Her bones may show numerous fractures, and these remain unrepaired, the broken ends being merely surrounded by a soft callus, and forming so many false joints. Her limbs also become bent in the strangest way, perhaps one leg outwards and the other inwards, according to the pressure to which they have been subjected while she is lying or half-sitting, propped up with pillows, and "all of a heap." Towards the last the softening of the bones may be so extreme, that one can bend them backwards and forwards with but little force, and without injuring them; bringing, for instance, the foot round so as to touch the back of the head. The more superficial bones, even the cheek-bones, can often be indented by the finger, or they may feel like egg-shells, as if they had merely a thin layer of osseous material on their exterior.

For a time the general functions of the body may appear to be but little interfered with; the appetite and the digestion may be good; and menstruation may go on naturally. Pyrexia may be occasionally present, but not to any marked extent.

Event.—Ultimately, however, the disease ends fatally, either by exhaustion or more commonly from inability to breathe, the ribs being dragged inwards at each inspiratory effort, so that scarcely any air enters the lungs. Another frequent cause of death is the obstruction to parturition caused by distortion of the pelvis. Usually the brim acquires what is termed a *rostrate* character, the pelvic symphysis forming a sharp angle between two prominences due to the pushing upwards of the acetabula and the parts adjacent. Cæsarean section has often been necessary, and has often ended fatally for the mother. It is a point of great importance that mollities ossium seems to advance step by step during successive pregnancies, the patient in the intervals regaining strength and being sometimes able to get about to work. The duration of the disease is usually from four to six years; but sometimes it has lasted eight, ten, or thirteen. Once, in a case recorded by C. Schmidt, it is said to have ended fatally in three months. In very exceptional instances it has ended in recovery.

Diagnosis.—Mollities ossium is very easy to recognise at an advanced stage of the disease, but it must be borne in mind that mere brittleness of the bones is not sufficient to determine it. In old people, and in persons who have been long bedridden, the ribs and some other parts of the skeleton are apt to undergo atrophy, so that one can very readily snap them with the fingers (*atrophia ossium senilis*). In the inmates of lunatic asylums this change seems to be particularly frequent. But even in young persons a somewhat similar state of *fragilitas ossium* is sometimes met with. The author once saw a young man who was dying of bronchitis, and in whom a large number of the ribs were found to be broken, as the results of muscular efforts in coughing. He had at different periods of his life had fractures of many of his bones from very slight injuries. Another affection which might be mistaken for mollities ossium is sarcoma, developing itself in a large number of the bones at once, and causing their spontaneous fracture.

Anatomy.—A marked distinction between these morbid states and malacosteon is that in it the bones are not only fragile but *soft*. After death they are found to be readily cut with a knife, and they feel like india-rubber or even like cheese. On section, the compact osseous tissue may have

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entirely disappeared, or it may be reduced to a very thin lamina beneath the periosteum, within which there may be nothing but a soft pulpy material. Or the cancellous tissue, instead of undergoing uniform absorption, may be hollowed out here and there into rounded or oval cavities. In the case which occurred at Guy's Hospital in 1864 this change in the bodies of the vertebræ was so striking that at first it almost seemed as though masses of a soft myeloid growth had eaten away the bone. The calvaria was considerably increased in thickness, and had a homogeneous texture, which was compared by Mr Durham to softened pasteboard.

On chemical analysis the composition of the bones is found to be greatly altered. Different observers give different figures, but it may be roughly stated that the proportion of inorganic constituents is reduced to about 30 per cent. The carbonate of lime is said to be diminished in quantity even more than the phosphate, and the character of the latter salt to be changed, the amount of lime being deficient in relation to the acids, so that it no longer forms the ordinary basic compound. C. Schmidt has also stated that gelatin is often absent. It must be remembered that the percentage given above represents not the definite actual constitution of osseous tissue affected with mollities ossium, but an average derived from some parts in which the change is extreme, and from others which perhaps deviate but little from the normal.

As a rule the morbid process seems to be most advanced in the interior of the bone, and least so towards the circumference. The microscope shows that there may be wide differences even within a single Haversian system, the lamellæ nearest the central vessel being completely decalcified, while the outer ones still retain their inorganic constituents. The orifices of the Haversian canal on the surface of the bone are widened, and a viscid fluid may exude from them when the periosteum is stripped off, which itself is very thickened and unduly vascular.

The substance which fills the interior of the bones in mollities ossium seems not to differ essentially from the normal medulla. It varies in appearance, being sometimes of a deep red colour, and spotted with ecchymoses, sometimes opaque, yellow, and fatty, sometimes mucoid and semi-translucent. These variations probably correspond with those which naturally occur in the same tissue. In the red material there are numerous cells, some of which may contain two or sometimes several nuclei. At least this is what Virchow and other histologists have generally stated. Rindfleisch maintains, on the contrary, that exceptionally few young elements can be seen.

The pathology of mollities ossium is as yet unknown. The decalcification of the bones has been supposed to be due to the action of some acid, and lactic acid is said to have been actually detected in the osseous tissue by several of those who have made analysis of it, and also in urine passed during life. In one case which ended in recovery Moers and Mück found that the acid gradually disappeared from the urine as the disease was subsiding ('Deutsches Archiv,' 1869).

Rindfleisch, on the other hand, suggests that the solvent is carbonic acid. Recent observers have failed to find in the urine an excess of phosphate and of carbonate of lime, which was at one time said to be present, and, according to Mr Solly, in one case reached four times the normal quantity, such phosphatic deposits occurring in urine having an acid reaction (?). Phosphatic calculi seem, however, to have been found in the kidneys or in the bladder in certain cases, and abundant deposits of phosphate of lime were

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constant in a case of a woman who was under the care of Dr Oldham in Guy's Hospital about 1866.

It is a point of some interest that osteo-arthritis deformans has sometimes been noticed in connection with mollities ossium. Moreover, in the muscles changes have been found which cannot easily be referred to mere disuse; not only do they become flabby, wasted, and fatty, but Friedreich has recently shown that the nuclei of their fibres multiply, and that there are other histological appearances identical with those which occur in progressive muscular atrophy. Trousseau and Lasegue observed in some cases that gently touching or stroking the surface of the limbs was capable of exciting painful contractions of the muscles beneath. It may therefore be that mollities ossium is, after all, something more than a mere disease of the bone.

Treatment.—No certain means of checking the progress of this remarkable disease has been ascertained. It is most important for women who show signs of the disease to avoid pregnancy, since we have seen that this condition aggravates the evil each time that it recurs. Phosphate and carbonate of lime have been given with a view of supplying the deficiency of earthy salts in the bones, but probably without benefit.

A more efficient treatment is the administration of cod-liver oil. Trousseau, who regarded the disease as essentially the same as rickets, narrates two striking cases (one published by Beylard in his thesis in 1852), in which complete recovery of health followed the administration of this remedy.

OSTEITIS DEFORMANS.*—In 1877, and again in 1882, Sir James Paget described in the 'Medico-Chirurgical Transactions' (vols. lx and lxv) a remarkable affection of the bones to which he gave the above name. His first paper contained five, his second seven additional cases. It seems, as the term denotes, to be of inflammatory nature, though very chronic, and without pain, fever, or signs of suppuration.

It befalls those who have reached or exceeded the middle period of life, and is insidious in its beginnings, gradual in its progress, and apparently unlimited by any natural check.

The bones affected are those of the skull and the limbs. The long bones enlarge, soften and bend, but afterwards appear to become harder and denser than before, acquiring a weight and a closeness of texture like that which used to be described as *osteo-porosis.*

The patient loses his previous stature from curvature of the back and bending of the knees, and even the ribs may sometimes become fixed by their own overgrowth. The skull is often monstrously enlarged; but the facial bones escape, so that the disease never presents the aspect of what is called *Leontiasis ossea.*

There are pains, vaguely styled rheumatic, in the affected limbs; but no internal lesion appears to accompany the disease of the skeleton.

The pathology of this remarkable affection is quite unknown. It is certainly not due to syphilis nor tubercle, nor is it allied to gout or osteo-arthritis.

A case was recorded and figured by Mr Bryant in the 'Guy's Hospital

* This title Sir James Paget states in his second paper had previously been applied to a local osteomalacia of the tibia and fibula by Professor Czerny of Freiburg i. B. The correct form is osteitis (*ὀστίτις* from *ὀστέον*), not ostitis.

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Reports' for 1877 (p. 337), and another by Mr C. F. Symonds in the volume for 1881. The former was a further stage of one of Paget's five cases.

No remedy is of any service.

The writer saw many years ago, in the University Museum at Prague, an enormously and uniformly thickened skull, which was taken from the body of a patient who died, still young, after narrowing of the bony foramina, had gradually compressed the several cranial nerves. Other remarkable cases of more or less limited osseous overgrowth have been recorded, one in the 17th vol. of the 'Path. Trans.,' and another more nearly resembling the one above cited, except that the growth was concentric instead of excentric, described by Sir Prescott Hewett, is preserved in the Museum of the College of Surgeons, and is figured by Mr Holmes in the second volume of his 'System of Surgery,' p. 330.

The remarkable affection called *acromegaly*, in which the head and limbs hypertrophy, affects not only the bones but the soft parts. It was briefly referred to in the first volume (p. 866), under Myxœdema, with which it might sometimes be confounded. Two cases have been lately described and figured in the 21st volume of the 'Clinical Transactions' by Mr Godlee, and by Dr Hadden and Mr Balance.

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DISEASES OF THE SKIN

"His native beauty is a lily-white,
Which still some other-coloured stream infecteth,
Least like itself; with divers stainings dight,
The inward disposition it detecteth:
If white, it argues wet; if purple, fire;
If black, a heavy cheer and fixed desire;
Youthful and blithe if suited in a rosy tire."

PH. FLETCHER: *The Purple Island.*

INTRODUCTORY CHAPTER

General pathology—Classification: examples—Nomenclature—Historical sketch—The elementary anatomical lesions—The local distribution of cutaneous affections: in depth (bathymetric) and over the surface (regional)—Symmetry—The circumstances: age, occupation, and habits of the patient, season, history of the malady, subjective symptoms, disturbance of other organs, effect of previous treatment—Arrangement followed.

IT is now well known that diseases of the skin differ in their origin and significance no less than diseases of the tongue, the eye, or the bladder. Some of them are symptomatic of specific febrile diseases, of which they form a part, and are therefore important chiefly for the sake of diagnosis. Others are produced by certain articles of food, by poisons, or by drugs. Some are examples of various pathological processes which are familiar in other organs, as cancer, hypertrophy, atrophy, hæmorrhage, pigmentation. But the majority are examples of the widespread morbid process called inflammation.

Dermatitis is sometimes the result of definite local irritation, but is often independent of such obvious cause, and therefore must at present be called primary or idiopathic. In this and in other respects it is analogous to the inflammations which affect the bronchial mucous membrane, the stomach, the intestines, and the urinary tract. Closely resembling these structures in its origin and development, its general anatomy, its vascular and nervous relations, and its glandular apparatus, the skin has also a pathological alliance with the mucous membranes.

Diseases of the skin are best studied as examples of general morbid processes, modified by the peculiarities of the affected tissue.

Classification.—Any arrangement of diseases is valuable so far as it helps the memory to retain useful facts; any arrangement is useless or mischievous if it pretend to be a universal or "natural" or "scientific" system. Diseases are not natural objects; they are physiological states, which we sometimes define by their cause, as plumbism and scabies, sometimes by their histology, as sclerosis of the cord and epithelial cancer of the lip, sometimes by their constancy in transmission, as measles and typhus, and sometimes by more or less constant concurrence of symptoms, as chorea and epilepsy.

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The following examples show how cutaneous disorders may be classified on different principles, each of which is important, but none exclusively eligible:

I. *Classification of Diseases of the Skin regarded as Physiological Processes.*
(*Pathological Arrangement.*)

Acute inflammations:
- Diffuse, *e. g.* scarlatina, morbilli, syphilis, roseola.
- With venous congestion—Erythema nodosum.
- With œdema—Urticaria.
- With necrosis—Furunculus, anthrax.
- Localised in papules—Enterica, syphilis, eczema, prurigo, lichen.
  - vesicles—Eczema, zona, variola, scabies, herpes, varicella.
  - pustules—Impetigo, variola, scabies, syphilis, sycosis, acne.
  - blebs—Pemphigus, scabies, rupia, pemphigus neonatorum.
- Desquamating during involution—Scarlatina, eczema squamosum, &c.

Chronic inflammations:
- With over-production of epidermis—Psoriasis.
- With fatty degeneration—Xanthelasma.
- With hypertrophy—Elephantiasis (Arabum), molluscum fibrosum, &c.
- With contraction—Cheloid (of Alibert).
- With œdema—Elephantiasis (lymphatic), œdema durum.
- With venous congestion—Acne rosacea, pernio.
- With ulceration—Lupus, syphilis, lepra.

New growths—Xanthelasma, lupus, lepra, tertiary syphilis, cancer.
Atrophy—The senile skin.
Hypertrophy—Ichthyosis, cornu cutaneum, clavus, verruca.
Hæmorrhage—Traumatic (flea-bites), typhus, scurvy.
Pigmentation—Syphilitic maculæ, melasma, chloasma, icterus, ephelis.
Congenital malformations—Ichthyosis, cutaneous nævus.
Neurosis—Pruritus.
Anomalies of secretion:
- Increased or diminished—Seborrhœa, xerodermia.
  - Hyperidrosis, anidrosis, chromidrosis, &c.
- Obstructed—Molluscum, comedo, milium, acne, sudamina.

II. *Diseases of the Skin regarded as the result of Antecedents.*
(*Ætiological Classification.*)

Due to the sun, wind, &c.—"Eczema" solare, and traumatic dermatitis.
Due to the irritation of friction—Intertrigo.
Due to animal parasites—Scabies, prurigo pedicularis, much of impetigo
Due to vegetable parasites—Favus, ringworm, &c.
Due to the irritation of scratching—Much of scabies, urticaria, and prurigo.
Due to local applications—Mercury, mustard, arnica, antimony, &c.
Due to the presence of poisons in the blood—The rashes caused by belladonna, copaiba, shell-fish, &c.; bromide-acne; hydrœa from iodide.
Secondary to general disorders—The rashes of syphilis, scarlatina, morbilli, typhus, enterica, herpes labialis, scurvy, rheumatic erythema.
Due to disturbance of innervation—Zona.

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III. *Diseases of the Skin regarded as objects of Cure.*
(*Therapeutical Classification.*)

By external applications:
- Sulphur—Scabies
- Mercurial ointment, &c.—Phthiriasis.
- Hyposulphites, and other parasiticides—Ringworm and other tineæ.
- Tar—Psoriasis and chronic scaly eczema.
- Caustics—Lupus.

By internal remedies:
- Mercury—Syphilodermia.
- Arsenic—Psoriasis, chronic eczema, pemphigus.

IV. *Diseases of the Skin regarded in their Subjective Effects.*

Peculiarly painful—Zona, erythema nodosum.
Itching—Scabies, prurigo, eczema, urticaria, icterus.
Negative—Syphilis, typhus, enterica.

V. *Diseases of the Skin regarded as Anatomical Conditions of certain Layers, Organs, or Regions.*

A. *Bathymetric Distribution.*

The horny cuticle—Ichthyosis, cornu, clavus, sudamina, xerodermia, psoriasis.
The Malpighian rete and papillæ—Eczema, scabies, secondary syphilis, exanthems, erysipelas; psoriasis . . . . . not leaving scars.
The deeper cutis and subcutaneous fascia—Zona, variola, phlegmonous erysipelas, late syphilis, lupus, lepra, carcinoma . . . leaving scars.
Elephantiasis, xanthelasma, sclerodermia . . . . not ulcerating.

B. *Distribution to Organs.*

Sweat-glands—Sudamina.
Sebaceous glands—Milium, comedo, acne, molluscum, lupus erythematosus.
Hair sacs—Ringworm, favus, sycosis, furunculus.
Nails—Onychia, onychomycosis, psoriasis, atrophy.

C. *Surface Distribution.*

Scalp—Eczema (especially impetigo), phthiriasis, favus, ringworm, area.
Face—Dermatitis from exposure, ephelis, rashes of smallpox, measles, and syphilis, erysipelas, lupus, acne.
- Forehead—Supra-orbital zona, chloasma.
- Eyelids—Xanthelasma, milium and sebaceous cysts.
- Nose—Lupus, acne rosacea.
- Bridge of nose and cheeks—Lupus erythematosus.
- Nose and lips—Eczema and impetigo of children.
- Upper lip—Symptomatic herpes.
- Lower lip—Epithelial cancer.
- Beard—Sycosis.
- Ears—Eczema, xanthelasma tuberosum, tophi.

Neck, front—Intertrigo.
nucha—Furunculi.

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Shoulders and back—Prurigo pedicularis, acne, anthrax.
Chest—Rashes of scarlatina, syphilis and other exanthems; tinea versicolor.
Breasts—Intertrigo, scabies.
Abdomen—Rash of enterica and other fevers.
Sides of trunk—Zona.
Genitals—Eczema, scabies, elephantiasis scroti, herpes præputialis.
Nates—Scabies (in children), furunculi, intertrigo, congenital syphilis.
Elbows, extensor side—Psoriasis, xanthelasma tuberosum.
flexor side—Eczema, xanthelasma planum.
Forearms and back of hand—Erythema, hydroa (erythema iris).
Wrist and between fingers and toes—Scabies.
Fingers and toes—Pernio.
Palms and soles—Syphilis (especially squamosa and bullosa).
Axilla and groin—Eczema.
Knees, extensor side—Psoriasis.
flexor side—Eczema.
Legs—Chronic dermatitis, elephantiasis, erythema nodosum.

VI. *Diseases of the Skin as they specially affect different Ages.*

Infancy—Syphilis, nævi, eczema, intertrigo, impetigo capitis, strophulus.
Childhood—Scarlatina, morbilli, roseola, erythema, ecthyma, molluscum sebaceum, eczema of face and scalp, ringworm and favus, pernio.
Adolescence—Lupus, acne, erythema nodosum.
Early adult life—Enterica, secondary rashes, psoriasis, sycosis, lupus.
Old age—Rodent ulcer, epithelial cancer, prurigo pedicularis.

Nomenclature.—We must abandon the binominal terminology which once extended to the whole of medicine, as founded upon a misleading analogy between natural organisms like plants and animals, and heterogeneous objects like diseases. Next to a false pathology and fruitless attempts at classification, nothing has been more injurious to the rational study of this group of diseases than a cumbrous, pedantic, and often barbarous nomenclature.

A good name should have the following characters:—(1) It must consist of a single word. (2) It must be distinctive, and easily recognised both by the eye and the ear. (3) It must be capable of forming an adjective. (4) Less essential points are that it should be short and, if possible, familiar, of Latin or Greek origin, or capable of easy reproduction in the former tongue, and as classical and euphonious as may be. (5) It should be unmeaning, or at least arbitrary and conventional in its application; or, if descriptive, should apply only to some obvious and constant feature of the malady.

Historical sketch.—The fact that many eruptions of the skin are closely attendant upon febrile and other general disorders early attracted notice; and the humoral pathology which pervaded medicine from classical times until almost the present day afforded a ready explanation of their occurrence. Hence cutaneous diseases were long regarded as mere symptoms of some hypothetical "dyscrasia" of the four Galenical humours: the blood which took its origin in the liver, the phlegm secreted by the pituitary gland, the bile by the gall, and the black bile by the spleen. From due mixture of

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these humours arose the four natural temperaments: sanguine, in which the blood was predominant; phlegmatic or pituitous; bilious or choleric; atrabilious or melancholic; and from their ill-mixture resulted dyscrasiæ, such as scurvy, scrofula, and gout.

When the doctrine of the four humours was given up they were thought to be due to disorders of the blood; then, when better knowledge of the chemistry and morphology of the blood began to stand in the way of so easy an explanation, they were ascribed to "diatheses" or tendencies, of which the eruption was at once the evidence and the effect. Explanation by assumed causes still took the place of inquiry into anatomical and clinical facts.

The skin was assumed, as it still too often is, to be a kind of chart or index to the state of the blood, as the tongue to the state of the stomach, and eruptions are still so regarded by popular opinion.

During the eighteenth century the causes and indications for treatment of an eruption were readily determined by learned physicians to be due to a strumous cachexia, or a scorbutic state of the blood, or vitiated humours from obstruction of the primæ viæ, or lues venerea, but the actual physical conditions of the skin were scarcely noticed. It was the great merit of Willan (1808) (shared in some degree by his predecessor, Plenck, of Vienna, 1783) that he accurately described the *anatomy* of the morbid skin. His "orders," the *elementary lesions* of later dermatologists, are the alphabet of the subject, and correspond with the "physical signs" of the diseases of the lungs introduced by Auenbrugger and Laennec.

From the English or anatomical school of Willan and Bateman (1813) sprang the French school of dermatology, which may be generally described as *ætiological* in aim. Biett, its founder, was a pupil of Willan, and introduced his system into France (1833). He had the great merit of perceiving that syphilis does not merely act along with other predisposing causes in producing diseases of the skin, but that it has as its direct consequence definite, constant, and recognisable lesions, the knowledge of which is all-important for diagnosis and for cure. He was succeeded by Cazenave (1843), Devergie (1854), and other systematic writers, who continued the work of clinical investigation and accurate description. Unfortunately Biett's success in tracing certain cutaneous affections to syphilis led to the formation of similar groups of "scrofulides" and "maladies dartreuses."

The attempt to define cutaneous diseases by their true *nature and cause* instead of by anatomical lesions had been already made by Alibert, a contemporary of Biett. His eloquence and power of picturesque description had much influence, which was increased by the publication of a magnificent atlas of plates, illustrating his Arbre des Dermatoses (1814). His pathology, however, was erroneous, his descriptions superficial, his nomenclature inaccurate, capricious, and unclassical. The same attempt to explain rather than to investigate, and to supply the nature and causes of disease by hypothesis when proof is absent, pervades the voluminous writings of Bazin (1853–70), who carried the hypothesis of "diathesis" to its extreme limit. The same principles were illustrated in the interesting lectures of Professor Hardy (1858–64), so long connected with the great hospital of St Louis.*

Meanwhile, another school had arisen at Vienna which was guided by

* Since these lines were written, M. Hardy has returned to his dermatological studies and has published a systematic treatise on the subject.

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the pathological doctrines of Rokitansky. Its founder was Ferdinand Hebra, whose writings have done more than those of any other man to put the study of dermatology on a sound basis and to extend its limits.*

The *pathological* school of Vienna represented by Hebra, the *diathetic* school of Paris represented by Hardy, and the *anatomical* and *therapeutical* school of England represented by Erasmus Wilson (1847–67) have all changed during the last twenty years. *Histological* investigation by the improved methods of the last twenty years has thrown much light upon the morbid processes of the skin, and the newest school of dermatology, that of America, is making important contributions to the subject.

In England since the long blank after Willan and Bateman's work, the late Erasmus Wilson alone made valuable contributions to dermatology; but of late a new school has sprung up and has produced such excellent works as those of Dr Liveing and Dr Crocker, besides the numerous important papers of Mr Hutchinson and many younger dermatologists.

Before entering upon the description of the several diseases to which the skin is subject, it will be well to say a word on the most important points in their description and diagnosis.

I. THE ANATOMICAL LESIONS.—The following are the most characteristic and important:

1. *Hyperæmia* or *Congestion.*—(*a*) Mere overfulness of the vessels from paralysis of the vaso-motor nerves with redness and heat, but without the exudation and tissue changes which accompany inflammation. This hyperæmic blush, readily produced in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e. g.* Trousseau's *tache cérébrale*, see vol. i, p. 683).

(*b*) *Active or arterial inflammatory hyperæmia*, varying in colour from brilliant scarlet to rose pink, and combined with heat, tingling, or other sensations. Such an early stage of inflammation is often called "erythema." The local swelling and the subjective symptoms distinguish it from the non-inflammatory hyperæmia just described.

(*c*) *Passive* venous or conjestive *hyperæmia* dependent upon retarded circulation and distended venules. The colour is purple, bluish, or livid, the surface is cold, and there are no painful sensations. This passive conjestion, frequently seen as the result of thrombosis, and also in long-standing affections of the heart and lungs, is often associated with the more chronic forms of inflammation in which œdema is present, and connective-tissue overgrowth is apt to result. The best example of this condition is in the congestive erythema of a chilblain.

2. *Pimple or Papule.*—A solid, small elevation of the skin. Under this name more than one pathological lesion is included.

(*a*) The acute inflammatory papule, more or less pointed, bright red, small, and very early seen with a lens to contain a minute drop of exudation. It is either abortive or ends in a vesicle or pustule.

(*b*) The chronic large inflammatory papule, never showing liquid

* His 'Acute Exantheme und Hautkrankheiten' was the third volume of the series of textbooks of 'Pathology and Therapeutics' superintended by Virchow, and was published in parts between 1860 and 1874. Much of the latter part is written by Hebra's son-in-law, Moritz Kohn, now better known as Kaposi. The translation into English for the New Sydenham Society (1866–75), begun by Dr Fagge and the present writer, was completed by Mr Waren Tay.

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exudation, but apt to become covered with minute scales. Sometimes, as in psoriasis, these papules increase so as to form a raised patch, and then become covered with scales, and sometimes they coalesce with the same result as in lichen planus. More often the papules remain discrete and without scales, as in prurigo.

(*c*) A solid non-inflammatory papule formed by true hypertrophy of the normal papillæ of the cutis. Such papillary growths produce the minute multiple warts which occasionally occur in immense numbers over wide surfaces of the body; large local warts and condylomata are much more common.

(*d*) Solid elevations of the skin, which may be called false papules; such as the heaped-up scales at the orifice of a hair-sac which form the so-called "lichen or pityriasis pilaris," and a sebaceous gland occluded by its own secretion, which is called a "comedo."

3. *Vesicle.*—A visible cavity in the skin filled with transparent liquid. In almost all cases the vesicle is inflammatory, and the liquid is exuded plasma, consisting of water, salts, albumen, and a few leucocytes with only a trace of fibrinogen. Where the epidermis is thin the vesicles rupture almost as soon as they form, but where it is thick, as in the palm and sole, they grow and coalesce into large bladders. Broad and flat vesicles, as those of zona, are usually distinguished from the smaller and more closely-packed vesicles of eczema. The vesicles of smallpox are remarkable not only for their size and depth, but for the exudation being so effused into the meshes of the papillæ and Malpighian layer that the cavity is "pocketed," and shows a central depression or *umbilicus.*

Non-inflammatory vesicles consist of retained excretion either of sweat-glands (sudamina) or of mucous glands. The latter are practically the only vesicles seen on the mucous membranes, for under the moisture and friction of the mouth, though inflammatory vesicles form, they are scarcely ever seen before they burst.

4. *Pustule.*—A cutaneous abscess, that is a cavity in the skin containing inflammatory exudations, water, salts, albumen, and abundance of dead leucocytes in a state of fatty degeneration, with usually only traces of fibrinogen. The distinction between a vesicle and a pustule is therefore often one of time only, and rests upon the abundance of the corpuscular element in the exudation; but while most vesicles become pustules the exudation remains serous in many cases of eczema. Again, in contagious impetigo, in furunculi, and in some other cases, the first visible exudation is opaque, yellow, and purulent.

5. *Bulla* or *Bleb* is the name given to a very large vesicle. It is, as a rule, inflammatory and of essentially the same pathology as a vesicle or pustule. It contains at first transparent serum, but this usually becomes more or less completely purulent. There are also almost always shreds of fibrin to be seen. The anatomical distinction asserted between ordinary inflammatory bullæ and those of pemphigus will be referred to in the chapter on the latter disease.

6. *Scab* or *Crust.*—A dried-up concretion of the contents of a vesicle, pustule, or bleb. Its form depends upon the inflammatory process ceasing, otherwise fresh exudation succeeds, and no dried-up mass is allowed to form. The size of a scab will always depend upon that of a pustule or bleb which formed it, its thickness upon the amount of fibrin and leucocytes in the exudation. Its colour is often characteristic; light brown or yellow when

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the exudation is serous, deeper yellow (compared by the elder anatomists to honey, whence the term *Porrigo favosa*), greenish yellow in some cases when the pus is thick, red or almost black when the exudation contains red blood-discs.

7. *Scale* (*squama*).—A dry flake of epidermic cells. When scales form in moderate amount and of small size as the result of inflammation which has passed, they are described as *furfuraceous;* when large, adherent, imbricated, and glistening silver-white from the refractive power of air enclosed in the spaces, scales have the characteristic appearance seen in psoriasis. Large, thin, and very abundant scales, which have been compared with dry hop leaves, and are sometimes termed *squames*, are almost characteristic of pityriasis-rubra. Beside the true epidermic scales desquamation often consists of dried-up sebum or of dried exudation mixed with epidermis. The microscope distinguishes the amorphous fatty material of the former and the leucocytes of the latter from the flat horny cells of true scales.

8. *Wheal* (*pomphos*).—A flat solid elevation of the skin much larger than a papule, and of ephemeral duration. Such wheals may be either traumatic or idiopathic; they are the characteristic effects of the poison of the stinging-nettle and of the form of erythema, hence called urticaria. They are formed by acute œdema of the skin producing local anæmia from pressure.

9. *Scratch-mark.*—An injury to the skin of linear form and curved outline, usually marked by dried-up blood, and having a definite relation to the range of the patient's hands. They are of diagnostic value as proofs of pruritus.

10. *Raw.*—A surface which has lost its horny layer of epidermis so that the moist and living Malpighian layer is exposed, from which more or less exudation oozes. Such a raw weeping surface is characteristically seen after the blister formed by cantharides has been broken. It also results from the rapid rupture of a number of vesicles as in the kind of dermatitis called eczema madidans.

11. *Chap* (*rima*).—A crack or fissure which goes through the epidermis to its Malpighian layer or to the vascular papillæ beneath. These rimæ or rhagades sometimes extend very deeply, are apt to bleed, and are always extremely painful.

12. *Sore* (*ulcus*).—The result of destruction by inflammation which has reached below the Malpighian layer and has destroyed the papillæ; characterised by the absence of any trace of epidermis by the granulations which cover its floor and the pus in which they are bathed.

13. *Scar* (*cicatrix*).—The result of the healing process after an injury or disease which has been deep enough to destroy the papillæ of the part. Accordingly the presence of a cicatrix, however superficial and slight, shows that the preceding process affected the deep layer of the cutis.

14. *Nodule.*—A solid elevation of the skin larger than a papule, and seated in its deep layer. The nodule was formerly called a tubercle, but the word "tubercle" should never be applied except with its present pathological meaning. A *node* is a large nodule, and there is no reason for restricting the term to syphilitic nodes or *gummata.*

15. *Stain* (*macula*).—A patch of increased pigmentation of the skin, either the result of long-continued preceding hyperæmia or occurring independently as a primary increase of pigment.

16. *Hæmorrhage* (*ecchymosis*).—When a blood-vessel of the cutis vera gives way, a dark red or purple mark is produced, which (like the macula)

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does not disappear on pressure. When small, the punctiform spots resemble flea-bites, and are hence called *petechiæ;* larger extravasations, particularly when elongated in form, resemble the bruises caused by a stick, and are termed *vibices.*

The mode of invasion should be noticed, whether by successive foci appearing and coalescing, or by a spreading *serpiginous* border. The earliest and most characteristic anatomical lesions will generally be found in this advancing edge.

II. DISTRIBUTION.—After determining the morbid anatomy of a disease of the skin, the next step is to notice its distribution.

(A) *In depth.*—In its pathology the skin does not follow the anatomical and embryological division into epidermis and cutis vera. It may be physiologically divided into three layers:

(1) The *horny layer of epidermis* or *cuticle,* dead scales, the only affections of which are increased growth, atrophy, dryness, desquamation, and other results which really depend upon perverted growth in the living layer of cells which lies immediately beneath it.

(2) The *living Malpighian layer* of the epidermis, together with the *papillary layer* of the cutis. These two tissues are constantly and inseparably united in their pathology. Their inflammation constitutes the enormous group of diseases which come under the head of superficial dermatitis. Affections confined to this part never leave scars.

(3) The *deep layer of cutis* with the *subcutaneous connective tissue.* Inflammation or new growths beginning below the papillæ are prone to spread to the subcutaneous tissue and not to stop until they reach subjacent muscle or bone or deep fascia. The deep affections of the skin which lie in this region are less numerous, but more severe, than those of the superficial layer, and are always marked by cicatrices.

(4) Lastly come the cutaneous affections which particularly affect the *sweat-glands,* the *sebaceous glands,* the *hairs* and hair-sacs, or the *nails.*

(B) In their distribution over the *surface* of the body, the diseases of the skin differ greatly. The earliest attempts at classification were between affections of the scalp and of the trunk. In Willan and Bateman's system this character did not receive due consideration; but it has met with still less at the hands of French and German dermatologists. Even in the best descriptions of Hebra and his successors it is sometimes impossible to learn what part of the body is affected by a particular disorder. But the fact is that very few diseases of the skin are indiscriminate in their extent, while many are at least as definitely and exclusively fixed to certain localities as the lesions of enteric fever in the intestine, of tubercle in the lung, or of tabes in the spinal cord. The skin is not uniform in its structure, the relative thickness of its layers, its vascularity, its nervous supply or the distribution of its glands. Its different parts are variously protected both by natural and artificial coverings, they are variously exposed to injuries, to irritants, and to moisture. It is, therefore, not surprising that their diseases differ so greatly. Psoriasis of a flexor surface and scabies of the face should be regarded, like carcinoma of connective tissue or phthisis of the base of the lung, as altogether exceptional.

It must be remembered that in childhood the several regions of the skin are not yet completely differentiated, and hence the local distribution of

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its diseases is less strictly adhered to than in adults. We find precisely the same rule in the localisation of pneumonia, of malignant disease, and of tubercle, in children.

The principal dermatological regions are :—The hairy scalp, the axilla and pubes ; the face ; the orifices of the eyes, mouth, nose and ears ; the front of the neck, the chest, the abdomen and genitals ; the back of the neck, shoulders and loins ; the outer side of the arm and forearm and back of hand ; the bend of the elbow, the wrist, palm of hand and fingers ; the buttocks, outer side of thigh and leg, and dorsum of foot ; the inner side of the thigh and ham, the sole and the toes.

We must here stop for a moment to explain the meaning of the word *symmetry* in pathology. It is nothing to the point to call universal eruptions like that of scarlatina symmetrical ; they are so only because the human body is itself bilaterally symmetrical. Nor is it enough that the same disease should be found in both right and left members, as is acute rheumatism. Symmetrical distribution means that exactly the corresponding parts on the right and left side are simultaneously affected, both ears, both elbows, the back of each hand, the under surface of each wrist, the popliteal space on each thigh, or the sole of each foot. This is bilateral symmetry, but we also see examples of serial symmetry in pathology, when the same condition is seen on the elbow and the knee, the wrist and the ankle, the palm and the sole.

III. Circumstances.—The third group of characters includes the natural history and surroundings of a case of cutaneous disease. They help in diagnosis, they throw light upon pathology and causation, and they frequently supply hints for treatment. We have to consider :

1. The *age* and *sex* of the patient. Some affections, like prurigo pedicularis, are scarcely seen except in the aged skin ; others, like impetigo, are extremely common before puberty and extremely rare afterwards ; while true acne begins, with rare exceptions, at the period of adolescence.

2. *General health*, and particularly the state of the stomach and bowels; the urine, and the temperature.

3. *Occupation*, especially when parts of the skin are exposed to cold, to great heat, to wet, or to chemical or mechanical irritation ; and also *intercourse* with other persons affected with a similar disease.

4. *Clothing.*—Some cutaneous maladies are caused by the friction of under-garments, or by their being saturated with sweat, or being coloured with deleterious dyes.

5. *Cleanliness.*—Pediculi and tinea versicolor are the result of dirt, and the same is true of some forms of eczema, intertrigo, and acne. But as a rule it is remarkable how little local mischief is done by uncleanly habits and neglect of the skin.

6. *Climate*, season, temperature, moisture or dryness of the air, sun, frost, and wind. Many diseases of the skin, as of other organs, which were formerly supposed to be endemic, are not really so ; but some are peculiar to hot countries, and others, notably leprosy, have, within historical times, become more restricted in their range.

7. *History* of the malady, its duration, the manner of its onset, and particularly, when obtainable, a knowledge of the primary lesion. The fact of recurrence is also of great importance.

8. *Subjective symptoms*, as pain or discomfort, itching, burning, smarting,

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tenderness, or neuralgic pains, alleviation by exposure to the air, or by covering, by heat or cold, by the application of water or oil, by pressure or by friction.

9. *Concomitant symptoms:* Pyrexia, insomnia, jaundice, albuminuria, and the varied lesions of past or present syphilitic or tuberculous disease.

10. Lastly, the effect of previous treatment, whether positive or negative, is sometimes a great help, not only in avoiding therapeutical errors but in deciding doubtful diagnoses.

Diseases of the skin should be arranged (as they should be named) like diseases of other organs; *i. e.* for convenience, either alphabetically or otherwise. The order followed in the present volume is to begin with the most common affections, the superficial forms of dermatitis, eczema and its allies, psoriasis and its allies, erythema and its allies. Then will follow affections of the hair-sacs and cutaneous glands, including ringworm. Next comes a chapter on the deep inflammations and the hypertrophic conditions which result therefrom. Closely allied to the deep chronic forms of dermatitis are the important and well-defined diseases known as lupus and leprosy, and the chapter which treats of these two subjects is naturally followed by one on tumours and new growths. Then comes a short section on abnormalities of the cutaneous pigment and of cutaneous innervation, and the subject concludes with a chapter on the practical diagnosis of diseases of the skin in general.

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ECZEMA*

AND COMMON SUPERFICIAL DERMATITIS

> "Her wrinkled skin, as rough as maple rind,
> So scabby was, that 'twould have loathed all womankind."
>
> SPENSER.

Definition—Willan's—Hebra's—Its distinction from other forms of dermatitis—Histology—Anatomical lesions—Course—Distribution and local varieties—General symptoms—Ætiology—Diagnosis—Prognosis—General treatment—Local applications—Diet and regimen, baths, &c.—Internal remedies—Special treatment of local varieties.
Impetigo—Its relation to eczema—to pediculi—to contagion—Its treatment.

BY far the most important of diseases of the skin, and perhaps the most important of all diseases which do not shorten life, from its frequency, its obstinacy and the misery it occasions, is the affection now universally known as eczema (ἔκζεμα), the "outbreak" or "eruption," as the Greek physicians called it. In its commonest form it is familiar to the profession and the public, and cannot escape instant recognition, but under many circumstances it is difficult to diagnose, and opinions have differed widely as to its pathology, its definition, and the extent to which dermatoses bearing other names are allied to or identical with it.

Definition.—Willan classed eczema among *vesicular* diseases, and this is a proof of his acumen and judgment; for, although the vesicles of eczema are so small and numerous, so short-lived and speedily supplanted by pustules or weeping surfaces or scales that one may see hundreds of cases before the vesicular stage can be demonstrated, yet there is no doubt that vesicles are characteristic and, if not a constant, the most nearly constant anatomical lesion of eczema.

The most important step in the pathology of this disease was Hebra's statement that eczema can be produced at will, for it is in fact identical with the *common superficial dermatitis* which is the result of ordinary irritants. As a result of this important statement, Hebra not only described under eczema much of erythema, intertrigo, the pustular form of dermatitis known as impetigo, and most cases of papular dermatitis previously classed under various species of lichen and strophulus, but he boldly included scabies itself as also a common inflammation of the skin and therefore a true eczema. All succeeding dermatologists have more or less followed Hebra in extending the bounds of eczema far beyond the definitions of Willan and Bateman.

But invaluable as was the new doctrine of Hebra, it has become clear that for clinical purposes we must seek again to narrow the definition of the

* *Synonyms.*—Moist Tetter—Common idiopathic superficial dermatitis.—*Fr.* Eczème.—*Germ.* Ekzem—Die nässende Flechte.

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word eczema. Inflammation, the reaction of the living tissues to injury, is the key-note of pathology. If to the doctrine of inflammation we add that of degeneration and new growths, of parasites and of contagia, almost the whole range of modern pathology is covered. It is quite true that the vast majority of diseases of the skin, like those of the rest of the body, are inflammatory, but for prognosis and cure we need much more than this elementary fact. Hebra himself had too much sagacity and practical sense to be led far astray by his own reform.

(1) Syphilitic diseases are most of them undoubtedly inflammations of the skin, but however closely they might approach in symptoms and appearances to some forms of eczema he separated them very widely. Scarlatina is a dermatitis not unlike some stages of eczema. Variola and varicella often approach impetigo still more closely in appearance, but neither Hebra nor any of his disciples have classed the exanthemata of Willan with eczema. These diseases are all separated by our knowledge of their ætiology, by their combination with definite symptoms in other organs than the skin, by their course, and by the practical measures for which they call.

(2) Scabies, again, is distinguished from all other forms of dermatitis, not by the pathological process, but by the peculiarity of the irritating agent, by the consequent characteristic distribution, and by the special mode of treatment.

(3) Lastly, we must separate from true eczema diseases like psoriasis, which, though undoubtedly inflammatory, are special in their characters, in their anatomy, in their chemical products, in their results, and (above all) which cannot be produced or even simulated by an external irritation. In other words, they are not "common superficial dermatitis," such as results from the natural reaction of the healthy skin against a common mechanical, thermal, or chemical irritant.

But now comes a more fundamental definition of the term, which is absolutely necessary for those practical objects which are the end and justification of all refinements of nomenclature. If we call eczema common superficial dermatitis, and assert with Hebra that we can produce eczema at will by rubbing in an irritant ointment or by exposure to the sun, we run the danger of forgetting what is, after all, a most important character of the disease which we agree to call eczema, whatever else may be included under the name. Undoubtedly "wet tetter" is in the majority of cases *not* the direct and immediate result of local irritation. It is therefore preferable to say that a scorching sun or a mustard plaster will often produce a common superficial weeping dermatitis which is histologically and chemically absolutely identical with eczema, which may, if we please, be called artificial or traumatic eczema, but which yet differs from the true disease by the very fact that it is the physiological reaction of the healthy skin to a definite known irritant; that it further differs in its course, in its distribution, in its whole natural history, from idiopathic, typical, true eczema, and demands as a consequence a different prognosis and different therapeutics. The distinction, however subtle in theory or difficult to draw in practice, is of direct and paramount importance.

For instance, the surgeon of a gaol is shown an eruption on a prisoner's arm, which, by every anatomical character, is a "common superficial dermatitis"—is what might have been made by Hebra's eczema-producing liniment. He diagnoses eczema, prophesies the course it will take, its obstinacy, and its probable recurrence, and prescribes what he calls appro-

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priate treatment. But whether verbally correct or not, he has made as great a practical blunder as is possible. The common superficial dermatitis is traumatic; the eczema is not *like* that produced by an irritant, but was actually and designedly so produced; the subject of it is not a patient with a disease, but a skilful impostor who has inflicted injury upon his skin; the course of the eruption will not be guided by the natural history of eczema, but by the will of the patient, it will not recur except by his wish, and will not be cured by "appropriate treatment." Since, therefore, the name which follows a diagnosis should connote as much knowledge as possible in brief, it is much better not to name factitious dermatitis "eczema."

In the same way we should exclude all common superficial dermatitis which is the direct and immediate result of local irritation, for what is remarkable is not that the skin should inflame when irritated, but that the skin of many people is liable to undergo the exact pathological changes produced by irritation *without* any demonstrable irritant.

Eczema, therefore, may be defined as "idiopathic, common, superficial dermatitis." We must, however, fully admit the difficulty or impossibility of drawing a line in every case. We can only classify diseases as they more or less naturally are connected with certain typical forms. At one end of the scale we have purely traumatic dermatitis produced by a demonstrable external irritant, limited to its immediate effects and disappearing not to return when the cause is once removed; at the other end we have dermatitis appearing on parts of the skin which are not exposed to any known irritation, following a distribution which is independent of irritants, recurring without external cause after it has once disappeared, and only curable by measures other than those addressed to the local irritation. But in every case of dermatitis, however idiopathic, there is no doubt an *irritans*, if we could only recognise it, and in every case, however traumatic, there is an *irritabile* in the patient's tissues. Inflammation can never be truly "idiopathic," that is uncaused; for like every other event it depends upon antecedents. No heat of the sun, no activity of cantharides or of croton oil can produce a pustule or a bleb upon the skin of a corpse. All eczema is common superficial dermatitis, but every common superficial dermatitis has not the characters in its origin, its distribution, and its course; in fact, in its whole natural history, which entitles it to the name of eczema. In order practically to identify eczema we must, therefore, look for the clinical characters to be presently prescribed.

Eczema is dermatitis at the stage of *exudation*: it is well called "moist tetter." Cases of dry eczema no doubt occur, but they are either abortive or residual. When we use the term eczema we imply that the eruption is moist, or will be moist, or has been moist; or that at least it occurs in a person who has previously been, or will hereafter be, subject to another outbreak of the same thing, when exudation will be apparent.

Slight degrees of inflammation, when the result of irritants, fall under the minor degrees of superficial traumatic dermatitis. Slight degrees of idiopathic inflammation which do not reach the stage of exudation—hyperæmia, roseola, erythema of the skin—when not shown to be abortive eczema by their locality and course, belong to very different pathological groups. They may be, first, symptomatic rashes like those of measles and scarlatina which are true dermatitis with all the characters of inflammation, and followed by desquamation. Under the same head of exanthemata should be included, secondly, the roseolar, erythematous, or papular rashes

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of enterica, cholera, and syphilis. Again, there are superficial forms of dermatitis which never assume the characteristic exanthematous aspect, which differ entirely in locality, in the persons they are most prone to affect, in their local and general symptoms, and in their constantly subacute character. These superficial dermatites, of which erythema nodosum is perhaps the best type, are clinically and pathologically to be separated from eczema, and will be treated of in a separate chapter.

Histology.—The pathology of eczema is that of inflammation generally. Its signs are the four Galenical characters of pain, heat, redness, and swelling, to which we now add a fifth, pyrexia or febrile reaction. Of its cause we know no more than of inflammation in other parts. Traumatic inflammation follows injury or local death of a tissue; idiopathic inflammation we assume must have some corresponding lesion, but of its nature we are ignorant. The order of events is vaso-motor paralysis, dilatation of the small arteries and capillaries, stagnation of the blood-stream, diapedesis of leucocytes through the stomata of the capillaries, and exudation of the plasma or liquor sanguinis.

If a section of eczematous skin be made the cuticle is found unaffected, the Malpighian layer swollen, the papillæ œdematous with dilated blood-vessels, and multitudes of leucocytes clustered round them; the deep layer of the cutis and the subcutaneous tissues are unaffected. Looking at the living skin we see, so soon as a sense of slight irritation with some pain of a tingling or smarting character has drawn the patient's attention to the spot, that there is already an inflammatory blush. This usually has from the beginning a brighter, more arterial hue than the rose-coloured tint of true erythema. A more important distinction is that the erythematous blush is diffused and fades off at the edge. It is scarcely ever disposed in blotches, circumscribed or mottled patches or figures of definite outline. The swelling from œdema is very slight. On close inspection, particularly if a lens be used, one can see that the apparently uniform redness is produced by a number of isolated deeper-coloured points. In this and in other respects the early stage of eczema resembles scarlatina as true erythema resembles measles.

Before long, but never without a precedent stage of hyperæmia, there appear minute vesicles. Frequently, however, they are preceded by little red elevations, which for some time show no bright transparent spot of fluid, and such inflammatory papules may appear early and continue for a long time before becoming vesicular. Such papular forms of eczema must be regarded as abortive, and very seldom will a careful scrutiny fail to discover the evidence of liquid exudation at one period or another of the case. Soon after the vesicles have formed the remarkably thin roof of the cuticle ruptures and they run together, forming a raw weeping surface, eczema madidans, or they may previously have sunk somewhat deeper and acquired more or less purulent contents before their thicker roof bursts. Such pustular forms of eczema usually produce, not weeping surfaces, but more or less extensive scabs, though intermediate stages are very frequent. In the most typical form of eczema the weeping stage continues until a great abundance of clear watery exudation is poured out. It consists chiefly of serum, to which the salines give its irritating property and the albumen its characteristic effect in stiffening linen. On the raw weeping surface it is easy to distinguish more injected points which mark the seat of ruptured

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vesicles. This *état ponctué*, as the French writers call it, is very characteristic and may be sometimes seen before and even after the moist stage. The involution of eczema is accomplished by the exudation diminishing, and at last drying up, the weeping ceases, or scabs take the place of pustules. Finally, the cuticle again covers the abraded surface and a branny desquamation, formerly described as psoriasis diffusa and also as eczema squamosum, covers the lately inflamed parts. The itching still continues and is sometimes troublesome up to the very last.

In chronic eczema the skin becomes exceedingly thickened, a result which is readily appreciated on pinching up a fold. It is constantly covered with branny desquamation, acquires a deep red instead of a brilliant scarlet colour, and in certain parts is marked by deep fissures or rhagades, which often penetrate to the true skin and give rise to bleeding and excessive pain. This eczema rimosum is most frequent in the palms or the soles and in its hæmorrhagic form on the nipples and the lips.

Distribution.—One of the distinctions between eczema or idiopathic dermatitis and that which is traumatic in origin is that while the latter corresponds more or less exactly to the irritant, typical eczema has its own peculiar laws of local distribution. Speaking generally, it is a disease of the thinner parts of the skin, of the flexures, of the joints, and of the head and limbs rather than of the trunk.

Eczema, as above defined, is an extremely symmetrical disease, more so than any other affection of the skin excepting psoriasis.

The most characteristic locality for eczema is behind each *auricle*, not only because it is so frequently seen here when it affects other parts, but also because this spot is but little liable to other diseases. The *face* is more frequently affected with the pustular form of eczema (which we shall presently describe as impetigo) than any other part of the body, and this particularly applies to the lips, nostrils, and cheeks. In adults the face is less frequently the seat of ordinary eczema, coming next in frequency to the *limbs*. The same remarks apply still more strongly to the *scalp*, where impetigo is the commonest affection in childhood, but where ordinary eczema is comparatively rare except on the bald scalp of infancy or age. Eczema does not frequently affect the skin which is covered by the *beard*, and when it does is not usually remarkable for obstinacy, but sometimes the inflammation can be unmistakeably seen to penetrate the hair-sacs and there become a deep instead of a superficial dermatitis. Its clinical features and treatment are then so different that it is properly known as a spreading disease, sycosis. It may in like manner spread to the sacs of the *eyelashes* and become localised as what used to be called tinea tarsi.

The *neck* is very frequently the seat of eczema, especially the front and sides. On the *trunk* the shoulders, back, and loins are but rarely the seat of the disease; and the same statement applies to the gluteal region, which is so frequently the seat of isolated pustules, not only in scabies, but in the impetigo and ecthyma of children. The flanks, though covered with soft and delicate skin, are not often affected with eczema, which when present has usually spread from the axillæ or from the neck; and the same applies to the chest. In women, however, eczema of the *breast* is common either as eczema intertrigo beginning in the fold under the mammæ or as eczema of the nipple (*cf.* p. 784). The *abdomen* is more often the seat of eczema than other parts of the trunk, especially of that variety which begins at the

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navel. The *genital organs* by the thinness of the skin are readily disposed to dermatitis, but are certainly less often affected with ordinary eczema than either the face or the limbs. Either the inflammation begins as *intertrigo* (chafing) of the scrotum and thigh, especially common in infants, or it is an acute weeping eczema which extends to the abdomen, thighs, and other parts as well, or it is a chronic and extremely pruriginous eczema of the vulva or scrotum. The last form even more frequently affects the neighbourhood of the *anus*, particularly in elderly persons; and the cleft of the nates is extremely liable to eczema-intertrigo, particularly in fat persons, under the irritation of long walking and free perspiration. In many cases eczema of the anus, perinæum genital organs, and thighs forms a well-marked local variety, which must be carefully distinguished from the so-called "eczema marginatum" of the same regions, to be afterwards described as a form of tinea.

On the *arms*, eczema is scarcely ever seen over the deltoid, and though common in the axilla, and particularly its anterior fold, is much less so than at the elbow. The bend of the elbow is probably, next to the face and ears, the most frequent seat of ordinary eczema, and if we were to exclude cases occurring under puberty even that exception need not be made. The skin covering the biceps cubiti and the flexors of the front of the forearm usually participates in eczema of the elbow, and this local form of eczema is one of the most constantly and accurately symmetrical. The disease scarcely ever affects the olecranon, but there is a form of eczema which, though relatively uncommon, is seen quite often enough to deserve special mention. It is an ordinary weeping eczema occurring in adults and affecting the outer side of both forearms from an inch or more below the point of the elbow down to the wrist. It is extremely symmetrical, and often affects the skin of the upper arm which covers the triceps, though without spreading to either shoulder or elbow. The *wrist* and the back of the hand are comparatively seldom the seat of eczema, though the affection will sometimes spread from the arm as far as the knuckles, and this region is not unfrequently the seat of the dry chronic circumscribed dermatitis which will be described as "single patch eczema."

The *fingers*, especially their clefts, are often the seat of eczema, but in most cases this can be traced to a traumatic origin. The *palm* of the hand might from its thickness seem little adapted to eczematous inflammation, but it is very frequently the seat of a characteristic chronic dermatitis, painful disabling, symmetrical, and obstinate; which, from the absence of vesicles and the presence of deep fissures dependent upon the thickness of the epidermis, has received the name of eczema rimosum. This is, however, either of local traumatic origin, or at least is usually unassociated with eczema of other parts, and is curable by local applications alone. Eczema of the matrix of the *nails* is almost always part of eczema manuum. It proves a long period of dermatitis, and its presence is, therefore, a point of diagnosis in distinguishing eczema from scabies. The consequent malformation of the nail is generally marked by longitudinal grooves, and by less thickening than in the far more rare psoriasis unguium.

In the *lower extremities* eczema of the groin and inner part of the thigh is very common in adults, and is either associated with eczema of the arms or with eczema of the abdomen and genitals. The outer side of the thigh is not often the seat of ordinary eczema, and the patella, like the olecranon, is practically exempt, but the popliteal space is almost as favourite a seat of

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the disease as the bend of the elbow, and the inflammation spreads thence more or less extensively over the thighs and legs. Below the knee, however, eczema is on the whole less frequent than below the elbow, and most often appears in one of two forms. (1) Eczema of the calf and peroneal region of an ordinary weeping and irritable type frequently accompanies the corresponding affection just described of the outer side of the forearms, and, like it, is almost always symmetrical. (2) Varicose eczema, a local dermatitis often unconnected with eczema of other parts, is obviously the result of ordinary irritation acting upon a skin congested by varicose veins. It affects the inner side of the leg from the internal malleolus upward, that is to say from the point where, as Hilton showed, the least considerable anastomosis takes place, between the internal saphenous and the posterior tibial veins. This form of dermatitis is known by its purplish tint and frequent association with ulcers as well as by its locality.

The *foot* is less often affected with eczema than the hand, but follows its serial homologue very closely. When not of traumatic origin and not due to scabies or to intertrigo, that is to say, when dermatitis of the foot is true eczema, it either affects the dorsum in association with eczema of the outer side of the leg, or the soles as a chronic eczema rimosum much resembling that of the hand, or it is an eczema-intertrigo-digitorum sometimes leading to deep clefts between the toes. This last form is as common as eczema of the fingers, if not more so. It is rarely associated with the disease in other localities, and must be treated entirely by topical measures.

Chronic eczema of the soles sometimes assumes the characters of hypertrophic dermatitis, with accumulation of horny epithelium a quarter or even half an inch thick, very painful, and of a sickening caseous odour. This is quite distinct from the similar condition occasionally produced by syphilis.

Symptoms and natural history.—Eczema provokes extreme itching, more perhaps than any other cutaneous disease except prurigo and scabies. Indeed, although unlike these two there are many cases of eczema which are almost free from irritation, yet in others the pruritus seems to be at least as intense, constant, and obstinate a symptom as in the worst of any other disease. Itching is usually less in the weeping and acute than in the dry, papular, or chronic and scaly conditions, and it is very rarely marked in the pustular form of eczema. It is most intense in ordinary eczema of children, and in that of old persons, and of all local varieties is most constant and most severe in eczema ani et vulvæ.

Smarting, tingling, and some amount of local tenderness are common symptoms of the more acute and ordinary forms of eczema, and are associated with a peculiar sense of burning and tension. There is slight febrile movement at the onset of the disease and particularly when large surfaces are invaded at once, but even when the thermometer shows no appreciable elevation of temperature there is thirst, loss of appetite, and a slightly furred tongue. The mucous membranes are unaffected; there is no foundation for such names as eczematous gastritis, enteritis or bronchitis. The pathology of the digestive, pulmonary, and urinary mucous tracts is quite different from that of the skin. We have no right to assume an eczema of membranes which we cannot see when we cannot demonstrate eczema of those which we can. The writer has never seen eczema of the lips spread to the

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mouth or tongue, eczema of the anus to the rectum, eczema of the eyelids to the conjunctiva, or eczema of the penis to the urethra and bladder.

Except for anorexia produced by the slight pyrexia of the onset of eczema, the appetite and digestion are as a rule unaffected in this disease. Dyspepsia is so common that many persons suffering from eczema are also dyspeptic, but there is no reason to regard the latter condition as the result of the former, nor are the bowels either constipated or relaxed. One would expect beforehand the urine to be affected when large surfaces of the skin are inflamed, but there does not seem to be satisfactory evidence of any constant change. The fact that in the large proportion of cases of eczema the patients are not confined to bed, or even to the house, makes it the more difficult to obtain observations of the amount or average condition of the urine in this disease. Any deviation from health observed may be explained from causes unconnected with the eczema, so that, as far as we know at present, the urine must be stated to be unaffected in eczema.

Course.—Eczema is very rarely acute in origin, development, and recovery; even in what appear to be the most acute cases it will be found that the patient has been subject to previous attacks, and that in the intervals small patches of the disease linger behind the ear or on the face or hands or some other isolated part, and when the acuteness of the attack has passed off it is rare for the whole of the skin to return to its normal condition. Fresh smaller outbreaks occur again and again, so that with scarcely an exception, however acutely the first attack of eczema may sometimes begin, its course is a chronic one.

Another peculiarity also very characteristic of eczema is its strong tendency to *recurrence*. It is extremely rare for a person to suffer from a single attack in the course of his life. Again and again when the disease appears in a quiescent condition a fresh acute outbreak will occur, or one attack will scarcely have passed off when another supervenes. Happily the majority of cases are not lifelong in duration, but they usually extend over several years, and it is not unusual for recurrence to take place even after long intervals of complete freedom.

Ætiology.—We have already stated what appears to be the true relation between traumatic dermatitis and idiopathic eczema. When the inflammation directly follows an irritant, is not prolonged after its cessation, does not spread to other than the irritated region, does not recur without fresh irritation, and does not follow the local distribution of eczema, then it is best called common superficial dermatitis of traumatic origin. But some skins, whether by natural stability or by habit, are insensible to sun and friction and sweat, and the other irritants which in ordinary persons produce inflammation. In others a hot day, or bathing in sea-water, or an east wind, or a long walk, will produce eczema solare or eczema intertrigo or some other form of local traumatic dermatitis—which when once established becomes chronic, persists long after its original cause has ceased to act, and localises itself in the bends of the joints, and in the symmetrical positions which have been above described as characteristic of eczema. Admitting, therefore, local irritation of various kinds as an exciting cause of eczema, we must also admit a certain proneness of the skin to inflammation, and in more than half our cases this predisposition causes the disease without our being able to fix upon any probable irritant.

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It has been widely supposed that we are to seek the predisposing, and in most cases the efficient cause of eczema in a diathesis or disposition of the whole body, which can be recognised independently of the presence of the eczema, and which produces other recognisable diseases. This diathesis has been called dartrous, arthritic, and gouty, while the ever-ready explanation of a strumous disposition has been invoked when the other failed. The reader is referred to the works of Hardy and Bazin, of Gigot-Suard, and other French writers, for their exposition of the dartrous doctrine; while all that can be said for the more particularly gouty relations of eczema will he found in the writings of Erasmus Wilson, Hutchinson, and the late Dr Tilbury Fox. The present writer can only express unbelief in the whole doctrine of temperaments and diatheses, which appears to him to be the residuum of the exploded physiology of Galen, and seriously to impede the advance of medicine. It appears that although persons with gout are often subject to very irritable and obstinate eczema, in the vast majority of cases of eczema there is not the faintest reason for the belief that gout, that is, the deposit of uric acid in the joints, has been present in the patient or his immediate relatives; that there is no pathological connection between gout and true rheumatism, arthritis deformans, or gonorrhœal arthritis, and that none of the latter forms of multiple arthritis have any demonstrable connection with eczema; that eczema rarely co-exists with psoriasis, pemphigus, or other supposed manifestations of the dartrous diathesis; that no one can give an intelligible account of the characters by which this predisposition can be recognised; that there is no evidence that eczema has more than accidental connection with diseases of other parts of the body, or that it is anything but a common superficial dermatitis; and lastly, that the diathetic hypothesis is practically misleading in prognosis and treatment, no less than scientifically unsound.*

The efficient cause of eczema, apart from local irritation, is as much, and as little unknown, as the efficient cause of bronchitis or of cystitis. All we can say is that in some persons the skin is naturally sensitive, or delicate, or irritable. Such persons are, in other respects, like their neighbours, and the predisposition of their skin to inflammation can only be prophesied after the event.

General eczema is sometimes set up by an accidental and very local irritation, and this is probably the explanation of the undoubted though rare occurrence of vaccination leading to eczema: much more frequently their relation is purely accidental. It appears to be waste of time to discuss the vague speculations, at once unscientific and unpractical, which ascribe eczema to such common disorders as dyspepsia, or to such *idola theatri* as constitutional predisposition, assimilative debility, nervous debility, perverted innervation, renal inadequacy, strumous cachexia, scurvy in the blood, acidity of the *primæ viæ*, or *la diathèse dartreuse*.

There is no doubt that eczema is in some cases very decidedly *hereditary*, but it is certainly much less so than psoriasis, and in the great majority of cases there is no reason to admit hereditary predisposition. The exudation of eczema is not contagious so long as it is transparent. When purulent it probably shares in the infective characters more or less common to all pus, and occasionally the pustular eczema which we shall presently describe as impetigo capitis is most markedly contagious.

* The writer may be allowed to refer to papers on this subject in the 'British and Foreign Medical Review' for January, 1874, and in the 'Guy's Hospital Reports' for 1880.

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Eczema affects both *sexes* indifferently. It is common at all *ages*, but differs in its most frequent characters. In the infant it is of the ordinary papular and vesicular kind. The same form is seen in older children, but much more frequent in them is impetigo, or pustular eczema, which is comparatively rare before the first dentition and after puberty. In adults the commonest form is ordinary weeping eczema of the limbs and face. In old age, the dry, very chronic, and extremely pruriginous forms are the most characteristic.

Eczema appears to be universal over the globe. It is certainly not more frequent where gout is prevalent, as in London, than where it is almost unknown, as in Vienna and New York.

The traumatic forms of eczema naturally occur in those *occupations* where the hands are exposed to constant irritation. Hence, we have the eczema of the hands which has long been recognised as frequent in washerwomen, grocers, and other hand workers.

It is a popular opinion that skin diseases generally, and particularly eczema, as the most common of them, are most prone to occur at certain *seasons* —the spring and fall. Like most popular beliefs, this was not founded upon experience, but chiefly upon theory, and partly perhaps upon analogy. The period of change in the seasons seems "naturally" to be the most likely period for change in the human economy, and changes are proverbially dangerous. It is possible also that the insalubrity of southern Europe in the autumn, from the prevalence of malaria, led to a belief in the same result in northern countries, while even in England malaria was far from uncommon until quite recent times. However this may be, one meets with eczema of the ordinary irritative and inflammatory kind more often in the spring than at other times, and this may be fairly attributed to the dry east winds which then prevail.

Diagnosis.—Keeping to the definition of eczema as above stated, the only difficulty is on the one hand to distinguish between idiopathic and traumatic dermatitis, or rather to detect the decided and efficient prevalence of a traumatic cause, and secondly, to draw the line between eczema and certain other forms of superficial dermatitis, the distinctive characters of which, and the justification for their separation, will be considered under each head. The distinction between eczema and intertrigo, eczema and impetigo, eczema and some forms of lichen, and even eczema and scabies, depends ultimately, as all distinctions in medicine should, upon practical utility. What it appears to me we must recognise is that all superficial inflammations of the skin may be grouped around certain types, and that the most common and important of these, which we call eczema, is characterised by being *common*, that is to say, the same as is produced by ordinary mechanical or chemical irritants, *idiopathic*, that is to say, not directly co-extensive with irritation, *moist* from visible inflammatory exudation, *symmetrical*, *selecting* certain favourite parts of the skin, and *prone to recur* after disappearance.

It may, however, be well briefly to point out the following characters. From *scarlatina* and other rashes, eczema differs in being never truly universal in its moisture and in being unaccompanied by marked febrile symptoms. From *erysipelas* it is distinguished by the colour, the minute vesicles, the locality, and the absence of a defined margin, of œdema,* and, again, of fever. *Erythema* exudativum is more rosy in tint, and though it may form papules or even bullæ, never shows the small vesicles or the weeping surface

* An exception occasionally occurs when eczema affects the eyelids.

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of eczema: its distribution is different, and it is never chronic in its course. Eczema has no resemblance to *psoriasis* except in very old cases of the latter disease, when the scales have disappeared and the locality is obscured.

Prognosis.—Eczema is extremely amenable to treatment, that is to say, we scarcely ever see a case in which no improvement can be produced, and still more rarely one which finally resists all therapeutical measures. Moreover, it is scarcely ever dangerous to life. There are, however, exceptions to each of these statements. In the outbreak of acute vesicular and weeping eczema, whether primary, or, as far more often happens, occurring in a chronic or nearly cured attack, we can do little or nothing to stop its violence. Abortive treatment is unfortunately rarely successful in any acute disease. Again, in some cases eczema, though treated until very little remains, cannot be driven entirely away but remains in a quiescent state here and there, to burst forth again after a longer or shorter interval.

Lastly, though it is remarkable how little the general health is affected even by very extensive, troublesome, and long-continued eczema, yet occasionally, in infants, or in aged persons, broken rest and loss of appetite cause wasting and muscular weakness, which may at last end fatally. The only cases of the kind which have occurred in the writer's practice were, first, an infant which became emaciated, pale, and unable to take the breast, and secondly, an old gentleman considerably over seventy, who after being much relieved by constant tepid baths and other treatment from an almost universal and extremely irritable eczema, sank rapidly and died without any evidence of organic disease. On the other hand, everyone must have seen scores of little children who appeared worn to a skeleton and almost moribund through severe eczema, who nevertheless, by treatment or by time, completely recovered. Very general eczema in a person over seventy, especially if complicated with gout or with chronic Bright's disease, should, I think, always suggest a guarded prognosis.

Treatment.—In the first place, it is our duty to treat and if possible to cure every case of eczema as quickly, safely, and pleasantly as we can. The supposed danger of driving in eczematous eruptions upon internal organs appears to be without any foundation. It arose partly from theoretical views of the sympathy of organs, partly from the well-known fact that cutaneous hyperæmia diminishes or disappears during acute febrile affections, partly from observing the benefit of counter-irritation of the skin in synovitis or bronchitis, and possibly, as Hebra unkindly suggests, from the difficulty of curing some cases of eczema. In little children, however ill, one may again and again observe that as soon as cutaneous exudation is checked and the irritation subsides, their general health begins to mend. The only caution I would give is to be very careful to ascertain the condition of the heart, the arteries, and the kidneys in aged persons suffering from eczema, lest the treatment or cure of their cutaneous disease should be credited with the fatal result which is really the consequence of degenerated viscera.

Prophylaxis and general protective treatment.—The irritants which excite or keep up and renew eczema are chiefly mechanical, thermal, and contact with water. Of mechanical irritants the most important are rough clothing, friction against adjacent parts of skin (intertrigo), prolonged contact with decomposing sweat, and also with dirt and various chemical irritants which are incidental to certain trades. But the most difficult mechanical irritant

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of all to get rid of is that which is the result of the disease itself. Eczema always itches, and itching is sure to produce scratching. Hence our first attempt is to prevent this by persuasion of an adult patient, by muffling the hands of infants, and by such local applications as will at least relieve the intolerable irritation.

With the same object, we forbid cold bathing, we forbid the application of either cold or heat, the latter for its immediate, and the former for its consecutive, effects on the circulation of the part. Contact with air is, in many cases, a decided stimulant, and one important use of the various ointments with which the eczematous skin is smeared is to protect it from air. With weeping eczema, we obtain the same end by covering it with wetted rags, or, as is often more efficient, by dusting it with absorbent powders.

But even more important as a cause of irritation is moisture. It is not that mere contact with water is an irritant; probably a slight alkaline and weak saline solution with a little colloid material, such as gum or size, or the albuminous part of oatmeal, is the least irritating medium with which an inflamed and excoriated skin can come in contact. A continuous bath, even of ordinary water, is a most useful and perfectly safe means of treatment in some cases of very general eczema, with profuse exudation and great irritability. To Hebra is due the merit of proving that patients can be kept continuously in a bath of suitable temperature, not only for hours, but for days—indeed, for any indefinite period, without leaving it for any purpose whatever. The writer saw this plan carried out at Vienna, and has more than once adopted it himself. The practical difficulties are obvious, and it is fortunately not often that we need resort to it.

Though *continuous* contact with water is by no means irritating, bathing and washing mean *intermittent* wetting of the skin. The change from dry to wet, from higher to lower temperature, and the reverse change on withdrawing the eczematous surface from the bath, the necessary friction of the towel, the saline constituents of most waters, and most of all, the evaporation which even great care cannot entirely prevent, and which, after careless washing, goes on abundantly from the half-dried surface—these form altogether a most efficient series of irritations.

It is even possible that the frequent and systematic cleansing of the skin from dead epidermis and from sebaceous secretion, which is the result of the artificial condition of extreme cleanliness to which modern civilised society more and more tends, may itself render the skin more susceptible to slight irritants, and certainly with tender skins the use of soaps, of nail brushes, of rough towels, and of flesh gloves, may sometimes aid in exciting dermatitis—a small set off against the advantage to general health of mind and body to which a clean and active skin undoubtedly conduces.* Important rules of treatment in eczema, therefore, are that the inflamed parts must not be washed with soap, must not be washed with either hot or very cold water, must not be washed frequently, and must be very carefully dried after washing with soft, dry, and warm towels. In order to prevent the "chapping" of the hands which is so common in children during the winter, it is important to take care that they are thoroughly dried on towels which are not already damp. In large schools for poor children, a good plan is to make them dry their hands by dipping and rubbing them in a tub full of bran, instead of upon towels, which are sure to be wet for all but the

* See an amusing article by Hebra on the dangerous consequences of being overmuch clean, translated in the 'London Medical Record,' March 15th, 1877.

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first comers. They should also be given olive oil, or any other neutral fatty compound, to rub into the hands after washing, when there is the least appearance of dermatitis. With severe and chronic eczema rimosum of the hands it is necessary absolutely to forbid washing, and to protect from contact with air or moisture by ointment and a well-fitting kid glove. The best plan in chronic eczema is to advise that once a week, or more frequently as may be thought safe, a complete warm bath and thorough cleansing of the whole surface should be used, taking care to keep the skin immersed from the time of its being first wetted, and to dry it thoroughly when the washing is over. Washing, with unscented, pure yellow soap and lukewarm water, if done seldom and followed by careful drying and inunction, is less injurious to an eczematous skin than more frequent and careless ablution with no soap at all. For the exceedingly irritable skin of the face and hands, it is sometimes desirable, however, absolutely to forbid all contact with water, and cleansing can then be accomplished by friction with dry and stale bread crumbs. When there is eczema of the scalp the best cleansing agent is white of egg diluted with water.

Poultices are almost always injurious, and scarcely less so is the modified form which is the usual result of the application of water dressing, a piece of lint dipped in water or lotion, and closely covered with oiled silk or gutta percha. The impermeable covering soon raises the temperature. and the result is the combined warmth and moisture of a poultice, most valuable for relaxing tension, promoting suppuration, and relieving deep-seated inflammation, but most injurious in superficial dermatitis.

Eczema is common dermatitis and must be treated like other inflammations. Cold, however, is not often practically applicable, powerful as is its influence on inflammation ; the surface affected is too extensive, the difficulty of continuously applying adequate cold too great, and the ill-effects of considerable depression of the temperature of the surface too serious, for us to attempt treatment by ice or by cold baths. Moreover, intermittent application of cold by the reaction which ensues proves worse than useless. Eczema in the majority of cases which come before us has passed its acute or subacute stage, and irritation rather than heat is the common symptom. It is, however, always well for patients with eczema to avoid the heat of the sun or exposure to fires or to the heated atmosphere of crowded rooms. The affected parts should not be covered with thick woollen garments, and the patient should be lightly covered at night; the bedroom should be well ventilated, the temperature kept somewhat low, and much relief is experienced by keeping the feet or arms uncovered except with a thin rag dipped in lotion.

Local medicinal treatment.—We next come to the treatment of eczema by chemical applications. Our object is, first to diminish the hyperæmia and exudation by *astringents ;* secondly, to diminish irritability and to prevent scratching by *sedatives ;* thirdly, to substitute for a chronic and interminable process of inflammation a more directly traumatic, acute, and self-limiting process, or else, it may be, by less stimulus to produce an effect short of this but serving to quicken the natural process of physiological repair. Such agents have received the vague title of *alteratives.*

The most powerful chemical astringent which can conveniently be used is probably lead. (See a paper by Dr Payne in the 'St Thomas's Hospital Reports' for 1878.) Salts of copper, zinc, and iron, nitrate of silver, boracic acid and borax are also efficient astringents. So are galls, tannin, and similar

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vegetable preparations, though these are less applicable to the skin than to mucous membranes. As local sedatives we may use belladonna, opium, chloroform, hydrocyanic acid, but these are generally unsuitable to eczema on account of its extent and raw, denuded surface, both which characters make absorption too probable for these narcotics to be safe. More efficient as remedies against itching, and free from any but local action, are preparations of zinc, which combine antiphlogistic and antipruriginous qualities. Dilute solutions of carbolic acid, 2 per cent. in water, 1 in 20 in oil, are very useful. Weak tarry preparations are also efficacious, especially in the drier forms of eczema; as diluted oil of cade (juniper tar) or liquor carbonis detergens with vaseline in the proportion of two drachms to an ounce.

For chronic and no longer very irritable eczema more stimulant applications are necessary; sometimes stronger tarry preparations, unguentum picis liquidæ or pyrogallic acid. These are most useful in limited patches of scaly and very chronic dermatitis with much thickening of the skin; those, in fact, which approach most nearly in appearance and pathology to psoriasis. The colour and smell are objectionable, but the staining of both skin and clothes by chrysarobin ointment is much more unpleasant.

A still more energetic method, introduced by Dr Anderson, of Glasgow, is painting the eczematous surface with liquor potassæ. This must be done with much caution, for it gives rise to considerable pain, though in many cases this is less complained of than might be expected; but the writer can bear witness to the efficacy and safety of the treatment when applied tentatively on limited surfaces of old and obstinate eczema, especially of the dry kind. With moist secreting surfaces of unusual obstinacy, one finds more useful the application of a solution of nitrate of silver varying from a scruple to as much as a drachm to the ounce. It must be occasionally painted on, not kept in constant contact, and often proves most efficient as an astringent and a sedative, as well as an alterative.

But more often there is too much active inflammation for us to venture on such treatment, and more generally applicable alteratives are the various preparations of mercury, corrosive sublimate in solution, white precipitate ointment, red oxide ointment, and dilute nitrate of mercury ointment. Mercury in some form is particularly adapted to pustular forms of eczema, and is seldom suitable to those which profusely secrete serum.

Most often, however, the cases of eczema which come before us combine the characters of inflammation, itching, and chronicity, so that for perhaps the majority of cases, at least if we include those of impetigo of the scalp, there is no more useful preparation than such a combination of zinc, lead, and mercury as forms the unguentum metallorum of the Guy's Pharmacopœia.* This may be varied by substituting the red oxide for the nitrate of mercury ointment, and by varying the proportion of the three constituents; often again lead and zinc act better without mercury, or the carbonate better than the alkaline acetate of lead.

Whatever be the chemical application used it is important to decide whether the vehicle should be watery or oleaginous. An excellent general rule was that of the late Dr Hughes Bennett, of Edinburgh; for dry affections of the skin, use ointments; for moist, use lotions. If an ointment is applied to a profusely secreting eczema the drug and its vehicle are washed away by free exudation and never reach the subjacent skin. Lotions, on the

* ℞ Ung. zinci, Ung. plumbi acet., Ung. hyd. nitr. āā partes æquales: Misce.

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other hand, have but little power of penetrating the epidermis, and if carefully watched will be seen to run from the surface, which is greasy by its natural sebaceous secretions. With raw surfaces which do not secrete profusely, either lotions or ointments may be appropriately used. Practical considerations teach us that lotions are better suited to diseases of exposed parts like the face and hands, that they are readily applied to young children, that they are more efficiently used by persons confined to bed or by women living indoors than by those who are engaged in active work, that they are more cleanly and pleasant to most people, but that they also give more trouble and demand more time in their application, and lastly, that in the summer, when the skin is frequently covered with sweat, they are particularly grateful and efficient. We must remember that lotions should in most cases be used with exposed skin or with the surface only covered by a thin rag into which the lotion has soaked. If applied in the morning and covered up till night they speedily become water dressings, and probably in less than an hour mere applications of rag with no further therapeutical power. On the whole, therefore, notwithstanding the rule quoted above and the fact that eczema is pre-eminently a moist tetter, it will be found that with the majority of our out-patients, whether private or at the hospital, ointments are practically the more eligible vehicle. It is important to make sure that the lard or other oleaginous material is not in the least rancid, and that it is free from salt. The addition of benzoic acid as now ordered in the British Pharmacopœia makes as good a vehicle in most cases as can be wished. The mineral oils have the advantage of not decomposing, and for some reason ointments made up of vaseline suit certain cases of eczema better than those prepared with animal fats.

Unmedicated oily applications, vaseline, cold cream, olive oil, have in themselves the good effect of protecting from air and of softening rough, harsh skin, inspissated sebum and dried secretions. Lanolin is better fitted for psoriasis and other affections in which it is desired to rub the oily vehicle thoroughly into the skin. Glycerine, from its strong affinity with water, is well known to be a direct stimulus to a nerve-trunk. It is, except when dilute, a decided irritant in eczema, and has far from the same soothing effects as cold cream or zinc ointment, in cases of intertrigo, chilblains, and eczema solare. In very small quantities, however, it may be added to lotions with the view of securing some of the advantages of an oily preparation.

Weak alkaline lotions have often been recommended to relieve the burning pain and irritation of acute eczema, and they were extensively used by Professor Hardy at St Louis. But in the very cases of acute weeping eczema in children to which such treatment seems applicable, the parts are so excessively tender that even a 1 per cent. solution of bicarbonate of soda is ill borne, so that in such cases the lead lotion (liq. plumbi subacetatis dilutus of the British Pharmacopœia) is more useful. At all events, if soda is used at all it should be in quantities only just sufficient to react to test paper.

Quite apart from the ordinary use of a lotion, the whole object of which is to keep the part continually wet, is that of a solution which when painted on is allowed to dry. For this purpose nitrate of silver or other strong astringent solutions may be used.

Another useful method is to suspend insoluble powders like oxide of zinc, starch, or sulphate of lime or bismuth in water by help of a little mucilage

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or tragacanth, without, however, attempting to form a perfect emulsion. The milky liquid is applied freely with a large camel-hair brush or sponge, and is allowed to dry over the weeping surface, and in some cases of irritable and profusely secreting eczema, as also in pemphigus, this is found to be the most effective application. Care must be taken not to have too much of the colloid ingredient, or hard cakes are apt to form which crack and become painful. In fact, chalk or gypsum shaken up with water and applied like whitewash is sometimes the simplest and pleasantest method.

Lastly, we may apply our remedies directly as dry powders. In this way oxide of zinc, chalk, and other fine insoluble powders may be used, and such applications are usually better than starch. They dry up discharges, protect from the air, and are often the best applications in cases of intertrigo. On the other hand, they are unsuitable for pustular eczema, where they would form massive and troublesome crusts.

It must, however, be admitted that nothing but experience, insight, and previous knowledge of particular cases will guide one aright in the selection either of appropriate astringents, in the strength of the application, or in the kind of vehicle. Some patients assure one (and prove right again and again) that they cannot bear any kind of ointment. With others all lotions are apt to produce pustules, or even boils. Not infrequently, especially in the acute stage of eczema, an inert powder or unmedicated vaseline, according as the surface is moist or dry, will do more good than anything else. In all cases we should remember that the ointments are not to be rubbed in, but gently smeared on the skin, and afterwards kept in continual contact by well-adjusted soft linen bandages; that the lotions should never be allowed to get hot, and must be frequently renewed; and that the strength of our applications should be small in the acuter stages, and greater as the case becomes inveterate.

The writer, in accordance with Hebra's teaching, and contrary to that of most English writers, believes that the majority of cases of eczema can be cured by well-directed local measures of the kind above indicated; but it must be admitted that in the great reforms he established Hebra undervalued the treatment by internal measures, which undoubtedly holds an important though a secondary place.

Diet.—In the acuter stages of eczema the patient should be put upon almost fever diet, but should be encouraged to drink freely of any cooling beverage. He should take no stimulants or meat, and eat sparingly, chiefly of bread, milky dishes, green vegetables, and ripe or stewed fruit.

In ordinary chronic eczema no such strict diet is necessary. It is, however, usual to forbid certain articles of food, and I think that the experience of patients shows that, at least in some persons, one or all of these really aggravate the disease, chiefly perhaps by producing thirst, increased irritation of the skin, and scratching. The kinds of food referred to are salt meats of all kinds, including ham and cured fish, cheese, pepper, spices, and other hot condiments. The stronger wines and malt liquors are also usually forbidden, but although in the necessarily generalised treatment of hospital out-patients this is doubtless good advice, there does not appear to be any evidence that the moderate use of malt liquors or wine (with food) does harm in eczema or any other affection of the skin—except in cases where, independently of dermatitis, even moderate stimulants provoke dyspepsia with flushing of the face or symptoms of gout. In many patients, especially those in middle and later life, wine or beer with the principal meal of the day helps digestion,

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and certainly does no harm to the eczema, while a little spirit and water at bedtime will help sleep, and in that respect prove a useful adjunct to other treatment. Sometimes, however, even weak whisky and water produces heat and discomfort after retiring to bed, and must then of course be interdicted. In almost all cases a somewhat free supply of unstimulating diluents should be taken between meals, and a glass of water while dressing of a morning, and again the last thing at night, is almost always useful.

Watering places.—In chronic and obstinate diseases like eczema, patients frequently ask whether change of air would do them good. They are usually recommended to go into the country if they live in town, or go to the seaside if they live in the country, or, if they can afford it, to go to Scotland or Switzerland, or some other attractive place of resort. The only point on which one may speak with confidence as to the effects of air and climate upon eczema is that, just as it is aggravated by the east winds of an English spring, so it is more difficult to cure in the eastern counties of England and Scotland, and is often favourably influenced by removal to the moist and soft air of the western Highlands, of Devonshire, or of Ireland. As one sees, however, in so many other diseases, it is the change which does the good, and this is most apparent when the change is from an unfavourable climate. Secondly, there is no doubt that in many cases of the more irritable forms of eczema sea air proves a decided irritant. It is only now and then, in chronic and non-pruriginous eczema or in the impetigo of childhood, that sea air and even sea bathing do good instead of harm.

With respect to baths generally we have already sufficiently insisted upon the evil effects of frequent contact with water, but there is no doubt that in the very chronic and intractable forms of eczema, saline and sulphurous baths act beneficially, probably like the stimulant and alterative applications above described. When the period has arrived for their use it is difficult to say, and each case must be judged by the tact and experience of the physician. A single bath may bring back in all its virulence an eczema which had nearly disappeared. Long-standing dryness, thickening of the skin, and absence of excessive irritability are the features which should generally weigh with us in advising or permitting this mode of treatment. The baths best adapted for the purpose are perhaps those of Harrogate.

Internal treatment.—Lastly, we come to the treatment of eczema by drugs, which I regard as less important than that by external applications and by what may be generally called the hygienic treatment of the skin. Still there is no doubt that while we could better dispense with this group of remedies than with the others we should often fail for want of them, or the success of our treatment would at least be less rapid and complete.

In the acute stage of eczema with profuse exudation and much irritation, it is the practice of the French school to purge freely, and most English physicians adopt the same plan, though perhaps less systematically. Saline laxatives are, perhaps, the most useful in these cases. The old-fashioned white mixture of sulphate and carbonate of magnesia taken three times a day, or the pleasanter combination of Epsom salts with carbonate of soda in peppermint or cinnamon water, are useful and popular medicines. A seidlitz powder or a dose of Rochelle salts or Carlsbad salts every morning is suitable for less acute or less extensive cases. Often it is sufficient for the patient to take a draught of Püllna, Friedrichshall, or Hungarian bitter water. Of these three Friedrichshall is, perhaps, most often suitable, particularly when the eczema occurs in a gouty subject. Sometimes, however, it is less efficient

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than a seidlitz powder, and occasionally it produces much griping without satisfactory result. In such cases it may be changed for the Hunyadi Janos with advantage. Some find that this Hungarian bitter water agrees better with women than with men. Whichever form of laxative is selected it should be taken with a large draught of warm water early in the morning and on an empty stomach. Such a dose should give one or two loose motions after breakfast without griping or subsequent irritability, whereas even larger doses, if undiluted with water or taken with the stomach already full, are more slowly absorbed and produce more frequent and less effectual irritation. In cases of eczema in which the patient has other independent evidence of gout, it is well to combine with moderate laxatives the exhibition of a pill containing colchicum and aloes or rhubarb every or every other night. In persons who have lived freely and who are subject to hepatic dyspepsia, beside restricted diet both in food and drink and moderate laxatives, it is important to prescribe small doses of mercury, either a single grain of blue pill with a little nux vomica and rhubarb before dinner, or two, three, or four grains with an equal quantity of the compound rhubarb pill every other night or twice a week.

In many cases, especially in women affected with eczema, there is considerable anæmia, and then steel must be added to the laxative medicine, There is no better combination for this purpose than that of sulphate of iron, given in doses gradually increased from two to five or even ten grains, with half a drachm or more of sulphate of magnesia, five or ten drops of dilute sulphuric acid in peppermint, cinnamon, or chloroform water. Along with laxatives it is usual in cases of chronic eczema to prescribe acetate of potash and other diuretics. Their action is somewhat uncertain, more so than digitalis, squill, or the resin of copaiba; but salines, and especially those of potash, have other actions beside that upon the kidneys, and in ordinary cases of eczema with much secretion and extensive inflammation, citrate of potash or acetate of potash is often found more beneficial as well as more agreeable than the alkaline carbonates.

We have seen above how important a point it is to relieve the itching of eczema, not only for the comfort of the patient, but to secure the physiological rest which the night should bring to all inflammatory processes, and also to save him from the serious aggravation of his disease which scratching and rubbing the eczematous parts infallibly cause. Moreover, it is at night (even during sleep) that the irritation, increased by the warmth of the bed, reaches its maximum, and that the self-control of the patient is weakened or abolished. Beside the various measures above mentioned for securing coolness, protection from the air, and such help as local sedatives can give, it is often necessary to call in the aid of internal narcotics. Of these opium and its preparations should generally be avoided. Unless given in large doses they are apt to increase rather than to quell the irritation of the surface; they also check secretion and bind up the bowels. It is therefore better to prescribe chloral hydrate or bromide of potassium, or the two together.

Chloral should be avoided with old people, and with patients who may have disease of the heart or atheromatous arteries. On the other hand, it is extremely well adapted to young children, and I have found a dose of syrup of chloral the most harmless and useful sedative in the case of infantile eczema. The safest plan is to give a moderate dose when the child is put to bed, and repeat it towards midnight, and, if necessary, again towards morning. Fifteen or twenty drops (about two or three grains) may be given with

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perfect safety to a child of six months old; half a drachm twice or even thrice repeated in the night may if necessary be given to a child of twelve or eighteen months; and after infancy, say from two to five or six years old, half a drachm, or for older children a drachm of the syrup may be given at bedtime with safety. Again and again this treatment has been followed by the best results. In the first place the child gets rest, and in the morning is ready for food, and all its organs have profited by the natural refreshment of the night. Next, the skin has been free from fresh irritation, and instead of being marked with the little sufferer's nails, is paler and less angry than the night before. All the processes of repair have had opportunity to go on; the habit of pruritus is broken for the time, and the nervous apparatus concerned has escaped from a vicious circle of inflammation, itching, scratching, and increased irritation.

The bromides are unsuitable to infants from their bulk and disagreeable saline taste, but with older children five or ten grains of the bromide of potassium may be sometimes added with advantage to the chloral draught if suitably covered with syrup of lemon or orange. With adults nocturnal irritation is not usually so severe as with children, and a draught of bromide of potassium or ammonium, with or without the addition of chloral hydrate, is usually sufficient when a sedative is required. Fifteen or twenty grains of ammonium bromide, with ten of the potassium salt, and twenty drops of aromatic spirits of ammonia in an ounce of camphor water, forms an effectual and not unpleasant sleeping draught.

In some cases, especially in old persons, neither bromide, nor chloral, nor a combination of them acts well. Henbane may then be prescribed with advantage, but in doses of not less than a drachm of the tincture, either alone or with a little compound tincture of chloroform in camphor water, or the tincture of hyoscyamus may be combined with that of hop. Indeed, in old persons where the irritation is not severe, and the want of sleep is rather dependent on general conditions of their age than upon pain or pruritus, two to four drachms of tinctura lupuli is a pleasant form of sedative, and may take the place of the whisky and water which the ascetic habits of the patient may render distasteful, or which habits of the opposite kind may render too agreeable. In the happily rare cases of severe and intractable pruriginous eczema in aged persons we are sometimes compelled to resort to continual doses of opium as the only means of obtaining rest.

The exhibition of antimony has been recommended in the acuter forms of eczema, and in the writer's experience has sometimes proved useful by subduing vascular excitement and hastening the passage of the acute into the chronic stage of the disease.

In many cases of eczema, especially in children, the patient is thin and pale, with a poor appetite, and a frequent and feeble pulse. It is in these cases that a little wine or malt liquor is not only admissible but often extremely useful; and here it is that the exhibition of iron finds its proper place. In the case of anæmic women with constipation the best combination is that of sulphates of magnesia and iron. For children the syrup of the phosphate, the saccharine carbonate, or the citrate of iron and quinine, are all valuable remedies. For infants steel wine is also a popular and valuable remedy, but after eight or ten years old effectual doses are too large to be convenient. In cases of great anæmia in children, especially where there is diarrhœa, no preparation of iron is so useful as the tinct. ferri perchlor., guarded, if necessary, by twice the number of drops of glycerine, or its taste concealed by a

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little syrup. It would almost seem as if the astringent quality of the drug had an effect upon the profuse secretion of the eczema. The result, at all events, is often striking as well as beneficial.

With the exception of iron the group of so-called tonics are not generally indicated in the treatment of eczema. Quinine, however, has a very distinct effect, particularly in the case of infants, children, and persons below adult age, in preventing itching. Half a grain of sulphate of quinine may be given for this purpose to a child a year old an hour before bedtime, a grain if a year older, and as much as five grains to a boy or girl of fifteen. This effect of quinine was well known to the lamented author of this work, and it has been independently and strongly recommended by Dr Eustace Smith in his work on 'Disease in Children.' This writer also recommends guaiacum in the treatment of eczema, especially where there is reason to suspect a disposition to gout.

There remains a drug which, in England especially, has been very largely and often far too indiscriminately used in the treatment of eczema, as of all other diseases of the skin, namely, arsenic. It is undoubtedly a therapeutical agent of the utmost value in psoriasis, in pemphigus, and in certain other cutaneous diseases to be afterwards described, and no one of experience can doubt its efficacy in certain cases of chronic deforming arthritis, neuralgia, idiopathic anæmia, leuchæmia, and anæmia lymphatica; but, like all powerful medicines, it is powerful for evil as well as for good. In the acute stages of eczema, in most cases where there is extensive and active inflammation, and in most cases accompanied by severe pruritus, arsenic is decidedly injurious. In other cases, however, its success is so marked that in spite of its frequent failures it has never lost a certain reputation in the treatment of eczema.

The first indication for the exhibition of arsenic is that the eczema must be in a chronic condition—the greatest benefit is obtained in cases which have persisted for years. Secondly, the more dry and scaly the surface, the more infiltrated and indurated the skin, the less there is of active inflammation and the less disturbance of the stomach and intestines, the more likely is arsenic to be beneficial. As a rule, children with eczema do not need it, but some of the most striking instances of its value are in very obstinate and long-continued cases in young patients. One was a boy of fourteen, who from five years old had been the subject of what by his own and his mother's testimony was really uninterrupted eczema, spreading from time to time with excessive violence from its favourite seats over almost the whole body, but never absent from the scalp, the ears, and the limbs. When he was taken into the hospital there was dry scaly eczema of the head, face, and neck, and the hair was very thin. There was eczema rimosum of the ears and axillæ, papular dermatitis of the arms and back, and eczema rubrum madidans of the abdomen, genitals, perinæum, nates, and thighs. The only parts of the whole surface free from the disease were the palms, the soles, and the shoulder. He was thin, worn, and miserable, and the whole skin was so deeply pigmented that he looked like a mulatto, but the urine was perfectly healthy, and he had no other disease than this severe dermatitis. He was carefully treated with zinc and lead ointment and unguentum metallorum as he had been before while an out-patient, but he was also given arsenic in steadily increasing doses, from three drops up to fifteen three times a day. Under this treatment the inflammation gradually subsided, and at the end of five weeks it was reduced to a little ordinary

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eczema of the arms. This also gradually disappeared. Meantime he had become a stout, healthy-looking lad. He has from time to time appeared again with slight return of eczema, chiefly in the scalp and arms, but it has never in the least approached its former severity, and the skin generally, instead of being thick, rough, hard, and infiltrated, with almost entire absence of subcutaneous fat, is now smooth, soft, plump, and elastic, while his head is covered with a thick growth of hair.

In prescribing arsenic the following rules will be found useful:—To begin with a small dose, and gradually but steadily increase it until either obvious benefit results, or the physiological action of the drug is shown by itching of the eyes or slight nausea. When these occur the arsenic should be at once stopped, and then resumed in somewhat smaller proportion, and if necessary again cautiously increased. Secondly, it should always be given either with, or immediately after, food and sufficiently diluted with water. There appears to be no advantage in any other form of the drug over Fowler's solution. The arseniate of soda may be given in somewhat larger doses, but is probably converted into the same form during digestion. The liq. arsenici hydrochloricus is useful if we wish to combine it with perchloride of iron. The "Asiatic pills" of Vienna are in every respect less eligible.

Troublesome and difficult to treat as many cases of eczema are, sometimes rebellious to the very treatment which in apparently similar cases has proved effectual, and always liable to relapses which are most trying both to patient and physician, it is nevertheless very rare for us to fail in at least relieving the miseries of an attack, and in a great majority of cases we may be fairly said to cure a disease which, without skilled treatment, would linger on almost indefinitely. Hebra concludes one of the most masterly and original chapters in his great work by saying that he who having once decided upon his plan of treatment, follows it out with patience and determination, will attain his object sooner than he who often changes the measures that he uses. We may venture to add that while keeping steadily in view the broad principles of treatment based upon rational pathology and tested by experience, the most successful practitioner will be he who knows how to vary their application in accordance with the perpetually varying needs of each individual patient.

There remain certain practical points in the treatment of local varieties of eczema which must be briefly mentioned.

Eczema of the ears is one of the commonest local forms of eczema and is sometimes extremely troublesome. Ointments will be found almost always to suit better than lotions—lead, zinc, or equal parts of the two, or in some cases weak carbolic oil, 1 in 40. When extremely moist, powders suspended in thin gum are better than dry powders, which are almost sure to form thick crusts and produce bleeding.

Chronic *eczema of the meatus* may cause deafness by swelling or the accumulation of its products. This must be treated by syringing with soap and water and, if necessary, application of an alkaline wash followed by unguentum plumbi or unguentum metallorum made soft by an equal part of carbolic oil.

Eczema of the scalp is complicated by the presence of hair and of sebaceous secretion and is apt to become more or less pustular. The hair should always be kept short, but shaving is unnecessary. Unguentum metallorum is commonly a good application. In the drier form of eczema

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of the scalp with scarcely any exudation, which is often combined with seborrhœa sicca under the name of pityriasis capitis, tarry applications are most efficient, and none is better than liquor carbonis detergens, either diluted to form a lotion, or, as I have found better, with vaseline in the proportion of a drachm or half a drachm to the ounce. Impetigo capitis will be presently considered separately.

Eczema of the eyelids and adjacent parts is apt to cause considerable inflammatory œdema which resembles erysipelas, but the colour, undefined edge, and the absence of marked febrile symptoms, together with the almost certain presence of ordinary eczema, in other parts, distinguish the two.

Eczema of the lips is sometimes confined to that part and has then a peculiar aspect, there being very little serous or purulent secretion, great swelling, deep cracks, thin scabs, and considerable hæmorrhage. When chronic, large thin scales, partly epithelial and partly dry secretion, are formed which have led to its being called psoriasis labialis. The difficulty is to keep the parts from movement. Very mild ointments, vaseline with zinc, yellow oxide of mercury, or honey and borax will be found useful. Deep and painful fissures should be touched with nitrate of silver either in strong solution or (what is less painful) with a pointed pencil.

Eczema of the palms is usually bilateral and confined to these parts, or it may persist here after it has disappeared from the rest of the body. As above mentioned, it is often directly dependent upon irritants. Having made sure that the case is not one of syphilis, the first and essential point of treatment is to protect the hand from contact with all other irritants, and especially with soap and water. For this purpose scabs, scales, and crusts should be carefully removed with sweet oil, or if necessary by poulticing. The cleansed surface should then be anointed with unguentum metallorum, and thin rags covered with the same ointment should be closely applied to each affected part. A well-fitting thin kid glove should then be worn over the whole, and the dressings should be changed night and morning only. At the end of a week the improvement will generally be striking, or if not it will be due to some neglect of the patient in uncovering his hands or in washing them. If the parts are very irritable it is better to use diluted white precipitate or yellow oxide ointment, or occasionally unmedicated vaseline will be most effectual of all. In chronic indolent cases, on the other hand, a little of the red oxide ointment will often stimulate most usefully. If there is great accumulation of epidermis it must be removed with soft soap or Hebra's diachylon ointment. Deep and painful fissures should be touched at once with lunar caustic.

Chronic *eczema of the sole* is not nearly so common. It must be carefully distinguished from a syphiloderm of those parts by the greater pain, deeper fissures, and more exclusive range. Sometimes it is accompanied by enormous hypertrophy of the epidermis, with deep bleeding cracks and horrible odour. Such cases may be cured by the application of salicylic acid in an ointment (ʒss ad ℥j) until the horny masses are removed, and then assiduous treatment with the *empl. plumbi.*

Eczema of the matrix of the nail is still more local than eczema of the palms. It is comparatively rare, as a complication of ordinary eczema, and a precisely similar inflammation of the matrix of several nails is sometimes seen where there is no other evidence of its eczematous character. The ill-formed nail may be scraped, but its removal is unnecessary and

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painful. The grooves around it should be carefully anointed with some form of mercurial ointment.

Eczema of the mamma is not infrequent as a local variety of intertrigo (p. 767). It begins in the lower part of the breast, when it comes in contact with the adjacent skin, and is most common in stout women with pendulous breasts. The surface is raw, weeping, and irritable. The treatment suitable is extreme cleanliness and the application of drying powders, such as fullers' earth, bismuth or zinc, with separation of the inflamed surfaces by a bandage supporting the breast.

Eczema of the nipple.—Most cases which were formerly described under this title were probably examples of what is now generally known as "Paget's disease of the nipple." Its characters were graphically delineated by Sir James Paget in the 'St Bartholomew's Hospital Reports' for 1874. In his original fifteen cases, cancer developed as a sequela within two years, marked by retracted nipple and its other symptoms.

The appearance of the affected nipples is at first that of superficial dermatitis, with an intensely red, raw surface, minutely granular, and pouring out "a copious, clear, yellowish, viscid exudation." The border is more sharply defined than is usual with eczema, and it is said to be thickened from the first, feeling, as Mr Henry Morris puts it, like a penny in a fold of cloth. In any case this induration of the edge is present in the later stages of the malady, and is the precursor of invasion of the ducts and the whole glandular tissue by ordinary alveolar carcinoma. The supervention of cancer has sometimes been delayed until more than two—six or even ten—years have elapsed after the eczema appeared; so that it is doubtful whether the cancerous growth should be regarded as more than a frequent result of the chronic cutaneous irritation.

A parallel case has been reported by Dr Crocker, where a patch of eczema of the scrotum in a man of sixty ended in the development of cancerous nodules ('Path. Trans.,' 1890); and the process is familiar to surgeons in the case of cancer of the glans penis, of the lower lip, and of the tongue, following chronic and apparently innocent inflammations of the same parts.

The anatomy of the disease has been investigated by Butlin, Thin, Bowlby, and other histologists ('Med.-Ch. Proc.,' May 12, 1891), and many cases have been published on the Continent and in the United States (see 'American Journal of Med. Sc.,' July, 1884).

M. Darier brought before the Congress of Dermatology which met at Paris in 1889, remarkable microscopical observations of the presence of psorosperms (*coccidia*) in the epithelial cells of this disease, before as well as after cancerous changes begin (cf. p. 405). The amœba-like parasite is described by Dr Louis Wickham in his *Thèse de Paris* (1890) as recognisable in an early stage by appropriate staining in the substance of the cells, and as subsequently becoming encysted, glistening, and readily seen.

The patients are usually women who have arrived at, or passed, the climacteric age. There is at first no special pain, and but little irritation.

It is most important to treat every case of eczema of the nipple as early and sedulously as possible, even when it does not offer the non-characteristic features above described; but when the raised and indurated edge is present, the best plan is probably complete destruction of the diseased surface by caustic.

Eczema of the anus, perinæum, and genitals is sometimes confined to the immediate neighbourhood of the rectum. This eczema ani, prurigo podicis

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of Willan, lichen podicis of Hardy is, as these names imply, most frequently dry and papular and is apt to be intolerably itching; the irritation is sometimes most severe, especially while in bed, while the disturbance of the rest, and the remarkable effect of mental depression which is common to most of the disorders of this region, make it sometimes a truly miserable complaint. French writers describe it as sometimes associated with a profuse and almost paroxysmal discharge of mucus from the rectum. This form of eczema is most common in elderly persons and is often associated with portal congestion, hæmorrhoids, and the hepatic dyspepsia described in a previous chapter. In children it most commonly depends upon the presence of thread-worms. Occasionally it is started by fissure of the anus, and disappears when this has been cured by division of the sphincter.

The scrotum and penis are frequently the seat of eczema, most often of the weeping form: eczema vulvæ closely resembles eczema ani in its symptoms and, like it, most frequently affects persons beyond middle life. It is sometimes associated with, and probably dependent upon, diabetes, and sometimes appears clearly due to inflammation, new growths, or degenerative changes in the uterus or bladder. Eczema of the anus, perinæum, or genitals often proves very rebellious and leads to great thickening and induration of the parts affected. Borax lotion or lead ointment, according to the degree of moisture, relieve, perhaps, more frequently than other applications, but this is one of the forms in which one must be content with tentative measures in each patient. In some obstinate cases a drying lotion of nitrate of silver proves effectual when other means fail.

For the *intertrigo* of infants, finely powdered starch and oxide of zinc or chalk is the best application. When it affects the fold of the nates in adults it is better treated by extreme cleanliness and the application of vaseline or diluted white precipitate ointment. Glycerine to most skins proves an irritant rather than a healer.

Eczema of *the legs* due to *varicose veins* must be treated like varicose ulcers, by elevation and bandaging. An old-fashioned flannel bandage often proves a cheap and efficient method. Martin's elastic bandage often produces the most valuable results, but in wearing it or an elastic stocking care should be taken that the pressure is not too great.

So-called *eczema marginatum* of the thighs is essentially a form of ringworm, and will be described under parasitic diseases.

PUSTULAR DERMATITIS.—*Impetigo capitis, Pustular eczema of the scalp*—is one of the most frequent diseases in children. It was known to our forefathers as "scald-head," but has happily become far less common than it was when children's heads were more neglected than at present, and especially when the bad habit prevailed of covering the scalp with caps and linen hoods, indoors as well as out, by night as well as by day. Perhaps the majority of cases are due to the irritation of pediculi capitis, but there remain a large number where no such cause can be found, and where a similar eruption upon the face or other parts establishes its independent character. There is no doubt that we are right pathologically in counting these forms of dermatitis as belonging to eczema. They are superficial and never leave scars, they are often associated with ordinary characteristic eczema of the ears, the limbs or the trunk, and the same child may be affected at one time with what will be called impetigo of the face or scalp, and at another with eczema of the same parts; or in an infant with ordinary

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eczema of the scalp the dermatitis will be seen to become more pustular as the hair grows thicker over the head until it has assumed all the characters of the porrigo favosa of older writers. Nor does the fact that this dermatitis of the scalp is often dependent upon dirt, lice, and other irritants, prevent our regarding it as true eczema, if the principles above laid down (p. 763) are correct. That something beside a traumatic cause, an *irritabile* as well as an *irritans*, is necessary for the production of impetigo, is proved by the fact that some children and most adults may have pediculi capitis for many years, and may even suffer from the irritation and yet be free from impetigo.

We have already mentioned the best treatment for impetigo of the scalp when associated with ordinary eczema. The children who are the subjects of it are often rosy, plump, and in every way healthy, though here as in other cases it is necessary to judge by the trunk and limbs as well as by the face. If, notwithstanding fat cheeks and ruddy complexion, the child is found to have flat shoulders and nates, thin arms and thighs, apparently disproportioned knees, and ill-developed pectoral muscles, his impetigo should be treated not only with equal parts of ung. zinci and ung. plumb. acet. or ung. metallorum, but also by careful attention to diet, by Gregory's powder, with or without a little grey powder, and when the digestive disorder is corrected, by cod-liver oil.

Impetigo affecting the scalp or face alone without ordinary eczema, and in a healthy child, is happily not difficult of cure. Indeed, apart from the purely pustular secretion, from the eruption being discrete and with a defined margin, and from the absence of severe itching, these typical cases of impetigo are separated from eczema by the fact that they are not prone to recur. Zinc ointment is the popular remedy for the eruption, but its efficacy is much increased by the addition of equal parts of white precipitate ointment or by the substitution of unguentum metallorum. The hair should be cut short, but there is no need to shave it, and the parents may be assured that it will grow all the better afterwards. It is only in extremely rare cases after the inflammation has penetrated to the hair-sacs, owing to a deeper suppuration of the scalp from too strong local irritants, that the hair-sacs are destroyed, and a bald cicatricial patch results. Such an event is more often seen from impetigo of the scalp in an adult than in the far commoner cases in children. When, as is usually the case, the scabs are thick and massive they should be removed first by poulticing. In circumscribed cases the bread and water poultice may be used, but where the whole scalp is covered it should be anointed with linseed oil and a large linseed poultice be then applied. For circumscribed and strongly adherent crusts, soft soap or even liquor potassæ may be necessary. Great patience and gentleness should be used in removing the scabs, or the child will suffer considerable pain, and the cure will be retarded.

Impetigo a pediculis.—In all cases of pustular inflammation of the scalp the hair should be carefully searched for pediculi. Equally decisive of the cause is the discovery of the nits, which consist of small triangular cases containing the eggs made of hard material, in colour and consistence like dried size, adhering to the hairs by one side of the triangle, and visible to the naked eye. The impetigo which results from their presence is produced more by the scratching of the patient than by the irritation of the lice. It affects the back of the scalp chiefly or exclusively, and is attended with great consecutive swelling of the posterior cervical lymph-glands. Indeed,

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occipital impetigo is almost synonymous with impetigo a pediculis. The treatment is decisive and efficient. In bad cases, the whole of the long, tangled, and filthy elf-locks should be cut off, and the head washed with soap and water; but in slighter cases it is not necessary to cut the hair at all. The noxious insects are readily destroyed by mercurial washes, but an equally efficient and harmless remedy is the stavesacre ointment (ʒij ad ℥j). Common petroleum oil is also a cheap and efficient parasiticide, or if the hair is cut short, as is much the best plan in hospital practice, the white precipitate ointment which cures the disease will also kill the vermin. The egg cases are less easily attacked, and might be sources of future trouble. They must be either combed off, or removed with spirits of wine, or cut off, hair and all. The impetigo which results from pediculi will sometimes heal spontaneously as soon as they are removed, but usually unguentum metallorum, or white precipitate ointment, diluted to 1 in 3, hastens recovery.

Contagious porrigo.—This form of impetigo of the scalp has been separately described, and some authors have laid much stress on its distinction both from pustular eczema and from impetigo from pediculi, but no sharp line can be drawn. All impetigo is more or less traumatic and more or less eczematous, and all impetigo is more or less contagious. Nor is it in the scalp alone that contagious pus is secreted. The most virulent of all is the pus of a gonorrhœa; but no one can doubt that even it is of varying degrees of activity, when we consider the frequency of the urethral inflammation compared with the comparative rarity of gonorrhœal ophthalmia. Leucorrhœa is supposed as a rule to be non-contagious, and no doubt with justice, but the most experienced surgeons admit the possibility of infection from an idiopathic and apparently innocent discharge. Again, the pus of boils is extremely contagious, and is often the source of what is called ecthyma. The pus of scabies, too, is contagious. So also a whitlow on a child's finger may cause by contagion impetigo of the hand, of the nates, and sometimes of the scalp. Impetigo *a pediculis* often secretes pus of a most actively contagious kind, the proof being not only in the outbreak of similar pustules on other parts of the child's body, especially the fingers and the buttocks, but also in the spread of the disease to other children of the same household. Most cases of impetigo are only slightly if at all infectious, whereas in others a whole family or a whole street may be infected from a single case. It is said that the most contagious forms of impetigo are characterised by thick yellow scabs, by a sharp line of demarcation, and by readiness of cure by local means, as well as by absence of itching, by restriction to the scalp and face, and by being practically confined to children; but these are only the characters of impetigo generally, as distinct from ordinary eczema.

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THE ITCH*

"Occupet extremum scabies."—HORACE.

Importance of the disease—Its nature—The acarus—The superficial dermatitis it produces—The distribution of the parasite and of the inflammation—Diagnosis—Treatment.

THE last variety of common superficial dermatitis is one which may be called traumatic or parasitic eczema, for it is pathologically identical with eczema and impetigo as described in the last chapter. But it depends directly and exclusively on the presence of an irritant, namely the invasion of the skin by a parasitic insect (or rather mite). Hence its distribution, ætiology, prognosis, and treatment, are different from those of idiopathic or true eczema as above defined; and on clinical grounds—which should always decide classification—we accordingly place it apart.

This curious disorder is extremely common, almost as common as phthiriasis over the whole globe, and has been well-known from the earliest times, though its origin has only recently been discovered. It was called Scabies by the Romans, though no doubt the term was applied to many itching disorders of the skin in men and animals which were not parasitic.

Though once scarcely accounted worthy of a place in nosology, and though without the interest of danger, scabies is really one of the most important diseases from a scientific point of view; for, if this were the place to enter fully into its history, we should find that it illustrates the whole progress of scientific medicine,—the ancient method which still survives of inventing explanations instead of investigating circumstances, the fallacy of ascribing results to dyscrasiæ of which the existence has never been proved, the survival of doctrines in pathology which have long been exploded in physiology, the value of apparently useless knowledge, the bearing of pure sciences like zoology upon practical therapeutics, the nature of inflammation and the relation between an irritant and an irritable tissue, the radiation of sensations, the pathology of pruritus, and the importance of a patient's nails in the production of cutaneous lesions. Finally, scabies is the typical example of a disease which is now as fully known as it is, perhaps, possible for us to know any disease—of which we know the pathology and the cause, of which we can explain the symptoms, which we can diagnose with certainty, in which the hypothetical *vis medicatrix naturæ* is utterly powerless, but which we can cure by definite, simple, and rational means, quickly, safely, and completely.

Scabies, like the affections which have hitherto occupied us, is a superficial dermatitis: in the character of its lesions it may even be called a common superficial dermatitis, for they do not essentially differ from those

* *Synonyms.—Greek,* ψώρα.—*Lat.* Scabies, psora.—*Fr.* La gale.—*Germ.* Die Krätze.

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which will be produced by any common mechanical or chemical irritant of sufficient energy, and are exactly comparable in their anatomy to the vesicles of eczema, the papules of lichen or prurigo, the bullæ of pemphigus, and the pustules of impetigo. Hebra, therefore, since as we have seen he called all common superficial dermatitis of traumatic origin "eczema," logically describes scabies as a form of eczema. But, as explained on a previous page (p. 763), eczema is not a mere traumatic dermatitis, and scabies must be separated from all other diseases because its cause, its prognosis, and, above all, its treatment, are totally different. We may define it as a superficial dermatitis of various degrees of severity, but always accompanied with intense pruritus, which results from the invasion of the skin by a parasitic acarus and from the scratching which ensues.

The itch-mite.—The living cause of this disease is the female itch-mite now known as the *Sarcoptes hominis*, formerly as the *Acarus scabiei*, belonging to the acarine division of the class Arachnida. It has four pairs of legs (which at once distinguish it from parasitic insects) and is clothed in a chitinous integument furnished with abundant bristles.* The male acari, which are much the smaller in size and fewer in numbers, live upon the surface of the body, but do not burrow. The female after impregnation digs her way into the integument, forming a straight, curved or sinuous *cuniculus* (mite-burrow, *Milbengang*, *sillon* or "run") which is visible to the naked eye as a slightly raised ridge, with a dark depression at one end (the entrance clogged with dirt) and a papule or small vesicle at the other, where the parasite lies.† A lens of low power shows these characters more clearly, but it is comparatively rare to see the runs perfectly well developed, for they are injured by the inflammation set up, by the patient's scratching, by friction and by dirt. When fresh they are best seen in the soft skin between the fingers and on the ulnar and palmar side of the wrist, still better when present in the skin of the prepuce and penis, or in that of the mammary gland in women. In children their locality is less certain, and they are much less easily found. With quick eyesight and a little dexterity the burrow may be laid open with a needle from the entrance to its blind extremity, and the acarus, a minute white grain just visible to the naked eye, extracted. It generally clings to the point of the needle, but a microscopic slide with a drop of water, glycerine, or liquor potassæ should be ready to receive it. The needle should be sharp, stout, and not too long or elastic; some prefer the broader needle of the oculist. Another plan is to excise the parasite, burrow and all, by means of a sharp pair of scissors

* It is possible that the Greeks were acquainted with the acarus, for Aristotle describes φθείρες (*i. e.* pediculi) as coming out of little pimples which contain no pus ('Hist. Anim.,' v, 138). He applies the special term ἀκαρί to the cheese-mite (*ibid.* v, 144).

The acarus scabiei was recognised in the sixteenth century. Thus Rabelais (1483—1553): "*Mais d'ou me vient ce ciron icy entre ces deux doigtz?*" Ingrassias (1570—1580) described "lice which burrowed under the skin;" and Hebra quotes a passage from the famous French surgeon Ambrose Paré, living at the same time (1570—1590): "*Les cirons sont petits animaux toujours cachez sous le cuir, sous lequel ils se trainent, rampent et le rongent petit à petit, excitant une fascheuse démangeaison et gratelle.*" In Thos. Moufet's 'Insectorum Theatrum' (Lond., 1634) he writes: "Latine *pediculi*, gallice *des cirons* . . . Anglice *mites* in caseo, foliis, ligno arido, atque cera; sed in homine *whealewormes* dicuntur. Sunt pediculi, subter manuum crurumque et pedum cutem serpentes, et pustulas ibidem excitantes aqua plenas: tam parva animalia ut vix visu perspicaci discerni valeant." This account seems to have been taken from an Arabian writer, Abenzoar (Ebn Zohr).

† Compare the figures given by Dr Bristowe in his 'Practice of Medicine' with those of M. Hardy in his recent 'Traité des Maladies de la Peau.' The writer's experience is that short straight runs are more frequent than would appear from either of these figures.

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curved on the flat. The winding passage can then be demonstrated, with the black granular fæces of its inhabitant, and often with a row of oval eggs in chitinous cells, which are laid one by one as the acarus bores deeper into the skin. Sometimes the scissors fails to secure the parasite, but proves its presence by that of one or more of its ova.

The dermatitis.—The presence of the acarus produces irritation which in most cases is intense, equal to that of the most irritable eczema or the worst kinds of prurigo; but often it is comparatively slight, only annoying the patient after he is warm in bed, when the skin is more vascular, the papillæ more sensitive, and possibly the acarus more lively, while the patient has nothing to divert his attention from his own sensations. The degree of inflammation also varies extremely, and cannot always be explained by the more or less severe scratching of the patient. As above stated, there is usually a small vesicle formed at the end of each run; but beside these, large vesicles, bullæ, and pustules frequently follow, first on the hands and then (probably through transfer of pus and serum by the patient's fingers) on various other parts of the body. Small acuminate papules are also very characteristic, and not less so are the scratch marks, often accompanied, especially in children, by wheals like those of urticaria. In severe cases of scabies the dermatitis may be intense, both hands and arms swelling as if with phlegmonous erysipelas; or arms, hands, legs, and feet may be the seat of weeping, raw surfaces like those of eczema madidans; the lymph-glands of the axillæ and the groin become swollen and painful, and the excessive itching is at last replaced by the smarting and tingling of acute dermatitis. More often, especially in children, the pustules resemble impetigo or ecthyma, and form as they dry up thick scabs and crusts. In chronic cases—for unhappily we often see scabies which has lasted for weeks and months without detection, and has been therefore ineffectually treated—the skin becomes thickened, indurated, hard, scaly, and fissured, resembling the condition of the more chronic forms of dry eczema. Bullæ as large as those of pemphigus are less frequent lesions of scabies, but are not uncommon in children. A case was figured in the 'Guy's Hospital Reports' for 1877. In fact, we may say that any of the inflammatory lesions of eczema, erythema, urticaria, pemphigus, impetigo, ecthyma, and lichen, may be more or less perfectly represented. The large, flat, and discrete papules of prurigo, the imbricated scales of psoriasis, and the thin, dry abundant squames of pityriasis rubra are never simulated by scabies.

Localisation.—The acarus itself infests the thin skin of the hands between the fingers, the flexure of the wrist, particularly its ulnar side, the flexor surface of the front of the forearm less frequently, the foot and ankle occasionally, the axilla and the groin, the genital organs, the inner part of the thigh, and the fold of the nates. But the lesions indirectly caused by its presence have a far more extensive though perfectly definite range; in fact the local distribution of scabies is so well marked that in a majority of cases a glance is sufficient to identify it. The inflammatory lesions are always present on the hands, except occasionally, when the patients are engaged in some handicraft which leads to the constant immersion of their hands in oily or strong-smelling substances or in metallic solutions, and forbids the development of the parasite. The same result is often seen in private patients, where the hands escape owing to the frequent use of soap and nail-brush. With these exceptions which are important for diagnosis, the fingers and ulnar side of the wrist may be said to be the favourite seats of scabies as

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they are of the acarus. Some lesions will almost always be found upon the prepuce, and the inflammation usually affects not only the thin skin of the genitals, but that of the lower part of the abdomen, at least as high as the umbilicus. The whole of the forearms is very liable to be affected, and the eruption is more general than is the case with eczema. The axillæ seldom escape altogether. The buttocks are almost always more or less affected, and in children nearly constantly, especially at the gluteal fold, but the perinæum and the sacral region usually escape. The toes, feet, and ankles, especially the inner ankle where the skin is thinnest, are very frequent seats of the dermatitis of scabies ; less often the knees ; the whole of the inner side of the leg and thigh may share in the inflammation. The back, shoulders, and chest are but little affected, the thick or hairy skin being apparently less favourable to the parasite. The neck, face, ears, and scalp almost invariably escape, in striking contrast to the frequency of eczema and impetigo in these parts ; and the only exceptions are in children. Why the skin of the face is shunned is hard to say. It is not more exposed to the air than the hands, it is as thin and delicate and vascular as that of the abdomen, but for some cause it is shunned by the acarus. May it be that the large sebaceous sacs and thick cutis with thin cuticle which are characteristic of the face, as of the shoulders, the chest, and the scalp, furnish a fatty secretion which repels the invader ?

It has often been remarked that a line drawn across the waist and elbow of a man standing in "the first position" will have below it the regions of scabies acarorum, and above it those of prurigo pedicularis.

In children, localisation of scabies is much less strict than in adults, as we found to be the case with eczema and psoriasis, and probably from the same reason (p. 759). Only in children is the face affected ; the hands frequently escape, and runs are found as well or better on the ankle or in the skin of the sole ; the trunk, and particularly the nates, are often more affected than the limbs.

Diagnosis.—This depends on a recognition, first, of the characters of the dermatitis, next of its very constant localisation, and thirdly of the cuniculi, the ova, or the acarus. The general facies of the disease is so characteristic that nine out of ten cases will be recognised in a moment when the patient is stripped, but in private practice anyone may be thrown off his guard who is accustomed to diagnose by probabilities rather than by facts. As Sir William Gull said, there are three diseases which we all sometimes overlook, —phthisis and syphilis and itch.

Where the inflammation has completely obscured all trace of acari, the existence of the itch-mite may be proved by removing the crusts, boiling them in solution of potash or soda, and allowing the dissolved mixture to stand in a conical glass. On decanting and removing the deepest layer with a pipette, fragments of the chitinous skeleton may be recognised.

It need not be said that scabies is always *contagious*, and its occurrence in an entire household often leads to its recognition. It is remarkable, however, how cases may remain isolated, and we must remember that impetigo and prurigo (pedicularis), not to mention variola and varicella, may also be contagious. The mode of transference is not always easy to follow ; direct contact of hands is probably one method ; often the ova are conveyed by clothes or other articles of constant use. Bedfellows seem particularly liable to infection. There is no doubt that scabies is frequently a venereal disease, the acarus having first invaded the genital organs.

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Treatment.—Experience long ago discovered that sulphur is good for the itch; it is an effectual poison to the acarus, and all we need is the best method of applying it.

The general practice is inunction of unguentum sulphuris into the affected parts, especially those which are the chief seat of the acarus. The colour of the application may be disguised, but its smell is always unpleasant. Sulphur lotions or sulphur fumigations may be substituted, but neither are so effectual. The best method is for the patient to rub the ointment well in every night, to lie in merino clothing all night, and next morning to wash with hot soap and water, and apply a little dilute ointment to the most irritable parts.

A rapid cure may be effected by first rubbing the skin with soft soap so as to remove crusts and epidermis, and then using thorough sulphurous inunction. In this way patients are cured in a few hours at St Louis on a large scale, and their clothes meanwhile are baked and washed. This last precaution is important, since otherwise the patient may readily reinfect himself from his own clothing. With private patients the disease rarely gains such extension by neglect as to be severe, and its cure is usually quick and easy.

It may, however, happen that the sulphur ointment is itself too irritating; so that, although it kills the acarus, it perpetuates or sets up a fresh and even more severe dermatitis. One often sees these cured but over-treated cases of scabies, and all that is necessary is to recognise their nature.

With children, diluted ointment, two to one, or equal parts,—in infants with much dermatitis one to two,—are the best proportions, the dilution being made with benzoated lard or with zinc ointment.

In slight cases, especially in children, balsam of Peru is a pleasant and generally an efficient parasiticide.

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PAPULAR FORMS OF CHRONIC SUPERFICIAL DERMATITIS

(*PIMPLY TETTERS*)

"Ardentes papulæ atque immundus olentia sudor
Membra sequabatur." VIRGIL.

Definition—Relation to common dermatitis and to eczema.
LICHEN.—*Its traditional species—Strophulus—Lichen scrofulosus—Lichen planus.*
PRURIGO.—*Pruritus—Summer and winter prurigo—Prurigo senilis a pediculis—Idiopathic Prurigo: Hebra's and milder forms—characters and treatment.*

THE group of diseases to be now described is far from being natural or well defined. They agree with eczema in being inflammatory, in beginning as papules, in affecting only the epidermis and the papillary layer of the cutis, so that they never leave scars, in their essentially chronic course, in the itching rather than pain that they produce, and in their persistence and liability to recur.

But they differ in the following important points:—(1) The inflammation never goes on to the stage of exudation either of serum or of pus; they are all "dry tetters." Even when, as the result of scratching, common inflammatory exudation follows, the pustules or raws thus produced are limited by the cause and only assume the form of eczema or impetigo for a time. (2) They are much less symmetrical, and rather avoid than choose the favourite places of eczema. Their locality may be said to be undefined, and widely diffused, but they affect rather the trunk and the outer side of the limbs than their flexures.

At present it is convenient to deal with all the traditional "papular diseases" together, different as they are in pathology. Of the whole group only two can be said to be natural and well-defined morbid types, with a characteristic anatomy, course, and habitus: these are *Lichen planus* and *Prurigo senilis.*

Willan, who first strictly defined the term papulæ, admitted three genera of papular diseases: Strophulus, Lichen, and Prurigo, and of these names the latter two are still in general use.

LICHEN.*—Many dermatologists express by this term a papular dermatitis which by subsequent more or less free exudation of moisture, or by its symmetry, or by its association with previous or later attacks of ordinary eczema,

* The word Lichen (λειχήν, translated by the Latin word *impetigo*) was applied in Greek to any moss or "lichen" that grows on stones or trees, and also to any more or less similar rough "efflorescence" on animals or man. So Æschylus (Choëphori v, 281).

σαρκῶν ἐπαμβατῆρας ἀγρίαις γνάθοις
λειχῆνας, ἐξέσθοντας ἀρχαίαν φύσιν.

The word is very commonly used in the Hippocratic and Galenical writings and by Celsus and later writers, but without stricter definition than the following: *Lichen* est summæ cutis vitium ut *psora et lepra,* cum asperitate et levi pruritu; deterius quidem Pruritu, Psora autem et Lepra levius.

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proves itself to be more properly termed papular or *abortive eczema*. Its pathology, natural history, prognosis, and principles of treatment are precisely those of the drier forms of eczema. It most often affects the arms and legs, and the extensor rather than the flexor aspect.

Willan's definition is: "An extensive eruption of *papulæ*, affecting adults, connected with internal disorder, usually terminating in scurf, recurrent, not contagious." Bazin divided lichen, according to its origin, into parasitic, dartrous, herpetic, scrofulous, and syphilitic—a good example of his classification and pathology.

*Lichen circumscriptus** was the name given by Willan and Bateman to a peculiar and characteristic disorder. It occurs upon the trunk, usually between the shoulders, but may spread over great part of the back, or may affect the chest or abdomen. The papules are small, red, and arranged in patches with somewhat well-defined margins. It is not very irritable, and rarely, if ever, ends in ordinary moist eczema.

Many cases which have been described under this head are probably nothing but papular dermatitis, or local eczema, depending upon the irritation of decomposing sweat. The locality between the scapulæ and on the front of the chest is just where sweat accumulates; the eruption is most common in summer, and in persons who sweat freely; moreover, with the papules true vesicular sudamina may often be detected. But apart from this, it must be admitted that there is a distinct, though somewhat rare, circumscribed papular dermatitis, which, from the shape of its patches and from their spreading at the edge while the centre returns to its natural condition, reminds one of spots of tinea. Parasitic fungi are apt to occur in the locality and under the conditions named, as in tinea versicolor, but the affection under consideration is certainly distinct from tinea versicolor.

Dr Payne has pointed out that it is usually associated with wearing thick woollen vests, often night as well as day, and calls it flannel-rash.†

The areæ of the circles present a yellowish tint (*Eczema flavum*), and are sometimes covered by branny desquamation. When several circles combine they form irregular lines, and the eruption thus formed was called *Lichen gyratus* by Biett and Cazenave. Two of Mr Towne's models in the Guy's Hospital museum (Nos. 267, 268) show this affection perfectly.

This affection may be frequently seen in young men or young women, never in children or persons over fifty. The patients are usually cleanly, and, in fact, appear almost as often in private practice as at the hospital. Since the eruption generally does not itch much, and is most marked where it is not seen, one often comes upon it accidentally when examining

* *Synonyms.*—Lichen annulatus (Wilson)—L. marginatus (Liveing)—L. circinatus—Lichen acnéique—Eczema flavum—Seborrhœa of the trunk (Duhring).

† The following are Dr Payne's observations:—"In Dr Colcott Fox's interesting remarks (Jan. 8th) on a peculiar form of lichen, called L. annulatus by Erasmus Wilson, it is not stated that this is really the disease called Lichen circumscriptus by Willan and Bateman, and figured by the former in 1808, so that his name clearly has the priority. It has also been called L. circinatus and L. marginatus, and, as such, is described by Dr Liveing. For so unimportant a disease, the number of synonyms it has received is remarkable; it being also known as lichen acnéique, eczema flavum, &c.

"Which of the three names—lichen, eczema, seborrhœa—should be retained? I have described several cases in the 'St Thomas's Hospital Reports,' 1884 and 1885, and have suggested that, as the disease is not really either lichen or eczema, a neutral name, such as 'circinaria,' which does not beg the question, would be preferable" ('Brit. Med. Journ.,' January 22nd, 1887).

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the chest. In every case the patient has been wearing a thick woollen jersey next the skin, and almost always another or even the same has been worn at night.

Duhring and other American dermatologists regard Willan's Lichen circumscriptus as the result of irritation of the sebaceous glands, and call it Seborrhœa corporis; and seborrhœa capitis is present in most cases that the writer has seen. On this point Dr Payne remarks: "(1) that this affection is certainly often associated with seborrhœa of the scalp; (2) that minute examination undoubtedly shows that the starting-point of each so-called papule is a sebaceous gland; (3) but that it is not accurately described as merely seborrhœa or excessive secretion. The bright red colour of the papules or margins of the patches, which strikes every observer, depends not only on hyperæmia but on dilatation and elongation of the capillary vessels, which project above the skin level, as in psoriasis. Hence it is that slight scratching causes hæmorrhage. This is something more than over-secretion. A few cases, in which the eruption existed on the limbs as well as on the body, I believe to belong to a different disease, apparently identical with *Pityriasis rosea* of Gibert."

The aspect of L. circumscriptus is like that caused by a parasite; but, though often looked for, none has yet been found that is at all distinctive.

After shifting the underclothing, soap and water and a little liq. carbonis detergens as a lotion (1 in 10), or an ointment (ʒij—℥j), is speedily effectual in curing the disorder.

The form of lichen described by E. Wilson, and also by Hardy (*lichen circonscrit*), affecting the extensor aspect of the forearm and the back of the hands, and running an acute course, should probably be regarded as eczema papulatum.

*Lichen tropicus.**—The writer has seen only three or four cases of this curious affection, so well known in the East and West Indies under the name of *prickly heat*. It occurs also in Australia and on the West Coast of Africa. Its characters are the sudden appearance of the eruption, its almost universal distribution, and the intense irritation it produces. It is said to affect blonde more than swarthy persons, and whites more than blacks. After once attacking a patient, it is apt to return with each hot season until the patient is acclimatised, and, though usually cured by a temperate climate, it sometimes comes before us in England. In the cases referred to the eruption has been entirely papular, with no other lesion but scratch-marks or occasional wheals. The parts most affected were the abdomen, buttocks, and thighs. The face and scalp, the hands and feet, and the genital organs seem to be usually free. One must speak doubtfully about a disease of which the personal experience of an English physician is small; but from its acute character the absence of moisture, and its un-eczematous distribution, it is at present best classed as a form of papular dermatitis. The small red papules are frequently associated with sudamina.

It was once supposed, of prickly heat as of other eruptions of the skin, that driving it in by a cold bath was extremely dangerous; but more than fifty years ago Dr James Johnson, who gave a graphic account of it in his own person, justly ridiculed this superstition.

The late Dr Tilbury Fox regarded this disease as essentially an *adenitis*

* *Synonyms.—Greek*, ἴδρωα.—*Lat.* Papillæ sudoris.—*Arab.* Essera (in part).—*Angl.* Prickly heat.—*Dutch*, Rootvont.

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of the sweat-glands, the direct result of excessive heat and perspiration, and Dr Duhring, of Philadelphia, in his excellent text-book of dermatology, calls it *miliaria papulosa* on the same theory. Whatever its pathology, it has no affinity to any acknowledged form of lichen.

Lichen pilaris was a name applied by Willan to a familiar condition which is, however, not a dermatitis at all. The hair-sacs of the affected part of the skin become filled up with horny cuticle, which forms rough papular projections, hard, pointed, and very characteristic, both in appearance and feeling. They do not occur in places where the hair is long, but are almost exclusively confined to the outer side of the limbs, over the vastus externus most often, but not uncommonly more or less developed on the outside of both arms and legs, on the buttocks, and the shoulders. The condition is most common in the brawny skin of muscular working men, and may be readily removed by soap and water and friction. Occasionally it may be seen on the limbs of delicate children, in girls of only seven or eight years old. This affection was described by Devergie under the equally inappropriate term of *Pityriasis pilaris*. It may be better called *Keratosis pilaris*. Dr Fagge proposed for it the name *Rhinoderma* (from ῥίνη, a file, not ῥίς, a nose); but as he himself says, this term has such obvious disadvantages that he prefers Devergie's title.

Occasionally keratosis pilaris is complicated by local inflammation and large flat red papules, or pustules may result, each surrounding a minute hair. These cases have been called sycosis of the trunk or limbs. Several examples were shown at the Dermatological Society in 1885–86.*

Willan's *Lichen lividus* is purpura, the petechial spot being perforated by a minute hair.

The *Lichen agrius* of Willan is clearly, from its acute course and the presence of small vesicles filled with a straw-coloured fluid, a form of eczema.

Lichen urticatus (Bateman) seems to be nothing but papular erythema combined with urticaria. It corresponds to much of what is now called prurigo infantilis and prurigo æstivalis.

Lichen hypertrophicus is a name given by French writers to what is probably in most cases identical with the rare affection to be described under the name of *Mycosis fungoides*.

The term *Strophulus*,† applied by Willan and Bateman to certain papular eruptions in infants, is now deservedly abandoned. Willan defines lichen as a papular eruption occurring in adults, so that the original distinction between the two diseases was merely one of age.

Green remarks that "strophulus differs from lichen in no essential particular, a circumstance that might warrant us in discussing the two diseases under one and the same head" ('Compendium of Diseases of the Skin,' 1836, p. 174). This author points out the difference in age of the

* Such cases have probably been often recorded as examples of the rare form of disease, to be presently described, *Lichen ruber* of Hebra: indeed, Hans v. Hebra is convinced that some of his father's typical cases were really inflamed keratosis pilaris.

† Strophulus.—This name, derived from στρόφος, a swaddling band, was apparently first used to describe any skin-eruption occurring in an infant. A popular English name is *red* or *white gum*, or *tooth-rash*. These names point to the popular explanation of all cutaneous rashes, and most other affections which occur during teething: but it is probable that originally "red gum" was only a corruption of another vernacular title—"red gown," a not inapt description of a child covered with general erythema; and this word *gown*, though in English meaningless without the prefix, is only a translation of *Strophulus*.

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patients, the more frequent intermissions of strophulus, and its milder character. Rayer regarded strophulus as infantile lichen, but Wilson described them separately. Most authors admit that the papules so closely resemble those of lichen as to appear identical with that disease. They are, indeed, only modified by the age of the subject.

Strophulus albidus is not dermatitis, but *milium*, a variety of comedo which will be mentioned among affections of the sebaceous glands.

Strophulus intertinctus and *S. confertus* may be called *infantile lichen* by those who keep to this name. They are papular dermatitis of more or less acute form, and in most cases may fairly be termed eczema.

Strophulus volaticus, with its acute course and slight maculæ following the patches, is a typical form of *erythema papulatum*. Bazin and Hardy are unable to class these papular eruptions of infants among the chronic inflammations which they ascribe to the dartrous diathesis. The former writer places them under it among the scrofulides, the latter among what he ingeniously calls "Maladies cutanées accidentelles." The *Strophulus prurigineux* of these authors is identical with infantile prurigo.

Lichen scrofulosorum, or, as it is more conveniently called, *Lichen scrofulosus*, is a somewhat rare form of eruption, which was first accurately described by Hebra. He describes it as consisting of papules arranged in groups with some amount of pigment and slight desquamation, not itching, and lasting for a long time without change. It is almost always confined to the trunk, and in forty-five out of fifty of Hebra's original cases the patients had swollen lymph-glands, or chronic disease of the bones, or scrofulous ulcers, or were supposed, from a fulness of the abdomen, to be subjects of tabes mesenterica. On the other hand, in none of these cases was there evidence of phthisis. All Hebra's cases occurred in young men, the youngest patient being fifteen, and the eldest twenty-five. His description has been followed by subsequent German writers, who have added little to the account which he gives. The writer saw two of his cases in Vienna, and can testify that they were not, as has been naturally supposed by some writers, cases of pityriasis scrofulosorum sive tabescentium, the xerodermia or dry rough scaly condition of the skin not uncommon in phthisis and other wasting diseases.

Hans von Hebra calls it *scrofuloderma papulosum*. It does not appear under any form of Hardy's scrofulides. It might, however, be well included under Bazin's large group of scrofulides bénignes.

Kaposi has made sections of the affected integuments, and describes the sebaceous glands as blocked by epidermic plugs, and surrounded by a copious infiltration of leucocytes, so that according to this excellent observer, the disease would be a chronic inflammation of the corium surrounding the sebaceous glands. The late Dr Tilbury Fox transcribed Hebra's account without comment, only stating that the condition is "of infinitely rare occurrence in England." He afterwards published six cases in the 'Clinical Transactions,' vol. xij, with a plate. Dr Liveing has met with a few typical cases among poor out-patients, and thinks that the inconspicuous colour of the papules and the absence of itching lead to its being overlooked. Several well-marked cases have been lately shown before the Dermatological Society; one was a patient of Dr Payne's, a girl aged seven, pale, and with swollen glands.

From English experience, Lichen scrofulosus is more common in children than in adults, and is as common in one sex as in the other. Its locality,

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the circumscribed patches, the pale colour of the papules, and the yellowish pigmentation, together with entire absence of itching, are sufficient characters for diagnosis and justify its recognition as a distinct variety of chronic papular dermatitis.

The treatment consists in the internal administration of cod-liver oil, and is said to be uniformly successful.

*Lichen planus.**—There is a form of chronic superficial dermatitis which is so distinct from all others that it is well entitled to a separate name. Of all forms of papular dermatitis it recedes furthest from typical eczema, and approaches nearest to the dry tetter or psoriasis which will be described in the next chapter. It does not appear to have attracted the attention of the older dermatologists, and is indeed a somewhat rare disease. It was first described by Hebra under the title *lichen ruber*, and shortly after from a different point of view by Erasmus Wilson, under the more distinctive name which has been generally adopted in this country. In Germany the two forms are distinguished as *lichen ruber acuminatus* and *lichen ruber planus.*†

Hebra gives an elaborate table of the differences between lichen ruber and lichen scrofulosus, psoriasis, eczema, and pityriasis rubra. Some dermatologists question whether the disease described in Vienna is really the same as lichen planus, but in pathology and the essential points of their natural history the affections named by Hebra and by Wilson are one, although they represent two varieties which may be recognised.

Dr Liveing, Dr Crocker, and Dr Duhring in America, regard Hebra's and Wilson's disease as unquestionably the same. Wilson himself suggested that the cases described by him were varied examples of the lichen ruber of Hebra, but Hebra himself considered the two affections to be distinct, and this was also the opinion of Dr Fagge. It is held in America by Dr A. R. Robinson and Dr R. W. Taylor, of New York, where Hebra's form of the disease seems to be more common than Wilson's. Hans von Hebra describes two forms; one very rare, more acute, with greater formation of scales, more itching, and more generally diffused dermatitis, and also followed by more severe affection of the general health; the second common, more chronic, never spreading over the entire surface, with only slight irritation, and with no injurious effects on the health ('Kr. Ver. d. Haut,' p. 376, and 'Brit. Journ. of Derm.,' March, 1890). It appears to the writer that we must recognise two types of the disease: one more discrete, more widely spread over the surface, and with more marked general symptoms; the other more confluent, more strictly localized, and with less irritation and general disturbance. But that they are varieties of the same malady seems shown (1) by the fact that one passes into the other by intermediate cases and even in the same patient, so that we have seen what Hebra would have called "exquisite Lichen ruber" of the trunk and legs, with equally typical Lichen planus of the arms; (2) by the identity of the elementary lesion in appearance and histology; and (3) by the course and the therapeutics of the disease ('Guy's Hospital Reports,' vol. xxv, p. 254, and vol. xlvi, p. 391).‡

* *Syn.*—Lichen ruber—Lichen invétéré (Hardy) including Lichen plan corné (Vidal).

† Dr Unna, of Hamburg, proposes to add a third variety, *lichen ruber obtusus.*

‡ See an account of the discussion on the mutual relation of *Lichen planus* and *L. ruber* which took place at the Dermatological Congress at Paris in 1889, reported in the 'British Journal of Dermatology' for October, 1889.

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Anatomy.—No one who has seen a well-marked example of Lichen planus can doubt the accuracy of Wilson's description; the raised flat patches, their dull glistening surface, deep purple-red colour, slight desquamation, chronic course, and resulting pigmentation are together most characteristic. Hebra insisted upon the genuine papular origin of the affection, on the deep red colour of the papules, and their not increasing in size when once formed. Fresh papules appear, and they become confluent, so as to form the raised flat patch which struck Wilson's attention (see model 260 in the Guy's museum).

Hillier in 1866, and Neumann more fully in 1869, described the histology of the disease. The hair-sacs and adjacent sebaceous glands are the chief and apparently earliest seat of infiltration. The opening of the hair-sacs is wide and funnel-shaped, a fact noted by Hebra in his original account. The cells of the rete mucosum contain granules of dark-brown pigment, the natural papillæ are enlarged, the sweat-glands are unaffected, the sebaceous glands atrophied. There is after a time considerable thickening and induration of the skin, as in other forms of chronic dermatitis.

Distribution.—Lichen planus may occur upon the extremities or trunk, but has never been observed upon the face or head. Hebra describes it as sometimes affecting the palms and soles, and this statement is confirmed by Wilson and by Hutchinson. The patches are apt to be most marked in parts subject to friction, as the waist and the circle of skin pressed on by a garter. Its favourite positions are on the limbs, especially the forearm and wrist (front as well as back), and the leg below the knee; also the thigh, particularly the skin over the internal condyle and the hollow over the great trochanter. In the lower limbs the colour is more deeply purple than elsewhere.

It is often symmetrical, but less decidedly so than psoriasis or eczema.

In cases which agree more nearly with Hebra's lichen ruber the papules are of a brighter colour, and more generally distributed over the limbs and trunk. The papules and raised patches are not arranged in groups, as in most forms of lichen. In extensive cases the nails may be affected, but this complication is less common than in chronic eczema.

Mucous lesions.—Lichen planus is undoubtedly often associated with the white patches on the tongue and cheeks, which have been described under the varied titles of ichthyosis linguæ, psoriasis linguæ, tylosis, keratosis, and leucoplakia. The association was noticed in two of his cases by Mr Hutchinson ('Lectures on Clinical Surgery,' vol. i, pp. 211, 213), and we have seen it repeatedly at Guy's Hospital and elsewhere.

Natural History.—Hebra's patients were almost all men. In England it has been more often seen in women, but in 27 consecutive cases of the writer's there were 15 men and 12 women. It seldom or never attacks young children, but the writer has seen a typical case in a girl of thirteen and another in a boy of nine. Observers differ as to the existence of itching. In the cases on which the present account is founded it has once or twice been absent, sometimes troublesome, but never severe, that is, not comparable to the irritation of eczema, scabies, or prurigo.

Lichen planus is chronic in its development and course. Hebra describes lichen ruber as leading to marasmus and death. The late Dr Fox says that in both forms of the disease, the more general and severe of Hebra and the more local of Wilson, the patient is ill. Mr Hutchinson says that the large majority of patients believed themselves to be in their usual health when it began, but that if it persists long the general health may fail.

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All the cases seen by the writer were in persons in average condition, some of them in robust health. No internal organ is affected, nor are there symptoms of general disturbance, at least in ordinary cases.

Lichen planus does not tend to cure; it continues indefinitely, may spread extensively, and, as above stated, may in certain severe and protracted cases seriously affect the general health. After being cured it is liable to recur.

Diagnosis.—This affection is distinguished from eczema by its never forming either vesicular or raw surfaces, by its avoidance of the face and ears, its general distribution, and the comparatively slight amount of itching. In some cases it undoubtedly approaches very closely to psoriasis, especially to inveterate cases of the latter disease which have become generally diffused and have lost much of their characteristic appearance. Mr Hutchinson would recognise transitional cases, and indeed proposes to name lichen planus "lichen-psoriasis." But difficult as the diagnosis occasionally is, the distribution, the character of the scales, and the persistence of the papules sufficiently distinguish lichen planus from psoriasis.

Lichen ruber accuminatus must be carefully distinguished from what Devergie called L. ruber pilaris, which, as above stated (p. 796), is an inflammation of blocked hair-follicles (keratosis pilaris).

A more important distinction is between lichen planus and syphilis, for which it is often mistaken. The colour, the frequent absence of itching, and the somewhat irregular distribution, lead to this error, which is apt to be confirmed if white patches are found on the tongue or cheeks. This leucoplakia, however, is no proof of syphilis. The colour of lichen ruber is more purple and less brown than that of a syphilide. The freedom of the face and scalp, the absolute uniformity of all the lesions, and their persistence unchanged during long periods of time, usually ensure a correct diagnosis; but some cases which conform to Hebra's type (*L. ruber*) rather than to Wilson's (*L. planus*) are so much like a secondary scaly eruption that the present writer must confess that he has more than once mistaken the one for the other.

Treatment.—The treatment adopted, both in Germany and England, is the administration of arsenic. Most writers speak of this as specific and certain in its effects, but some of considerable experience find it occasionally fail, and lichen planus is certainly slower in yielding to the remedy than average cases of psoriasis. Locally, tar ointments or some of the milder preparations, which will be described under psoriasis as substitutes for tar, are important aids in treatment. In obstinate cases an ointment of pyrogallic acid should be applied. Unna recommends one composed of hydr. perchlor. gr. ½, ac. carbol. gr. xx, and ung. zinci ʒj. Dr Heitzmann, of New York, who tried this treatment, found a 3 per cent. solution of carbolic acid much more efficient ('Journ. Amer. Derm. Soc.,' Sept., 1889, and *ibid*, pp. 52 and 64).

PRURIGO.—Prurigo, "the disease attended with pruritus or itching," was a term formerly very loosely applied and is still somewhat difficult to define. Willan described it as a papular eruption in which the papules are of the same colour as the skin and accompanied by itching. His "species" were *P. mitis* and *P. formicans*, which are merely more or less severe cases of the same affection, and *P. senilis*, characterised by the age of the patients and the difficulty of cure. Bateman thinks that pediculi are not unfrequently generated when prurigo senilis is present, thus putting the cause

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for the effect, since it is now well ascertained that most if not all cases of prurigo senilis are directly caused by pediculi corporis.

Willan and Bateman also mention *Prurigo pubis*, which they rightly ascribe to the presence of pediculi, and *P. præputii* and *urethralis*, which are both sympathetic pruritus. Lastly, their species, *prurigo podicis* and *P. pudendi*, correspond to the drier and more papular and indurated forms of the irritable local dermatitis which was described in the last chapter as eczema ani and eczema genitalium.

1. *Pruritus.*—Prurigo, a papular inflammation of the skin, must first be distinguished from pruritus or subjective sensation of itching without any local lesion. Pruritus accompanies not only prurigo but also eczema and the desquamative stage of many exanthems. It is the constant effect of pediculi and of the acarus; it may be produced by jaundice, and may be the result of the various atrophic changes which take place in the senile skin. These, which have been well described by Neumann, include the wasting and ultimate disappearance of the papillæ, and it is probable that the process gives rise to senile pruritus.

Prurigo, or irritable papules, may be produced by primary pruritus. This occurs in hot weather among children, most often from the irritation of sweat or of vermin (the *Lichen urticalus* of Bateman). The papules are large, flat, and discrete; but there is no pigmentation, no thickening of the skin, and the distribution is irregular. Mr Hutchinson has observed this summer prurigo affecting the face and arms of adolescents and relapsing every year (*Pr. æstivalis*). According to this author another form of prurigo is common as a sequel of varicella.

Some persons, usually young adults, are liable in cold, frosty weather to great irritation of the skin, which becomes dry, harsh, and pale. This affects the covered parts most, and is often supposed to be due to flannel underclothing. It has been called *winter prurigo* by Hutchinson and *pruritus hiemalis* by Duhring, but in England is more common during the east winds of spring than in midwinter. The scratching of the patient produces a crop of papules. Dr Payne has published several cases of this disorder ('Rare Diseases of the Skin,' chap. iv). He recommends unmedicated glycerine or vaseline locally, and chloral as a sedative at night.

Many cases formerly described as prurigo should be called papular erythema, or urticaria, or papular dermatitis—arising from the irritation of bugs or pediculi (which in infants do not cause the characteristic appearance of prurigo pedicularis), or from the friction of flannel next the skin. Lichen circumscriptus, papular eczema, and even congenital syphilis have been mistaken for prurigo.

2. *Prurigo senilis a pediculis.*—This is a well-characterised and common disease known as prurigo senilis, phthiriasis, or prurigo pedicularis. It is a papular dermatitis of definite clinical characters dependent on the irritation of body-lice, and is only seen in elderly persons. It is a good example of the combination of two conditions—the excitant and the predisposing cause, the *irritans* and *irritable*—to form a constant clinical result.

Phthiriasis is not a sufficient title, for children may be swarming with vermin, and may suffer from urticaria or ecthyma as the result, but are never affected with this form of prurigo; nor is "prurigo senilis" enough, unless we recognise the exciting cause of the disease.

The papules are separate, not spreading over wide surfaces as in eczema, nor collected in more or less rounded patches as in lichen circumscriptus,

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nor coalescing as in lichen planus. Moreover, they are much larger than in eczematous dermatitis, flat rather than pointed, less red, and more persistent. But what is most characteristic is that before long each of them is capped by a little black crust of dried-up blood, the result of scratching.

Beside these papules, the disease is marked by an extensive series of scratch-marks following the curves which are described by the right or left hand respectively, working from the shoulder. The irritation of scratching not only causes excoriation and hæmorrhage, but sometimes produces wheals like those of urticaria, and raw surfaces which may be properly called traumatic eczema. Both these effects may be absent, but prurigo senilis never lasts long without the whole surface between the papules becoming more or less deeply pigmented, until in some cases the affected parts are as dark as the skin of a mulatto.

The *distribution* of prurigo senilis is as characteristic as its anatomy. It occupies the shoulders, back, and loins, the papules usually stopping abruptly at the waist or the sacral region, and sometimes not spreading below the scapulæ. They may appear over the upper arms, but rarely below the elbow and never on the hand. They are numerous on the flanks, and in severe cases may cover the whole chest and abdomen. The thighs may share in the disease; but even in the most extensive cases it is generally found that the outlying parts are rather the seat of ordinary dermatitis produced by scratching than of the true papules of the disease. Prurigo senilis never affects the face.

The itching is most severe; and, like all pruritus, it is worst at night and when the patient is warm. The absence of pain and tenderness leads to more reckless scratching than in any other disease. It is the consequence, however, and not the cause of the papules, for we can distinguish the latter from the traumatic dermatitis set up by the former.

The exciting cause of the disease can be found when carefully looked for, especially in the plaits of the underlinen about the neck and waist. It is important to remember that pediculi corporis may exist in old men and women of apparently scrupulous cleanliness.

The whole facies of the disease is so well marked that it can scarcely escape recognition. It affects both sexes. Typical cases are very rare in persons as young as fifty.

The *treatment* is simple and effectual when the disease is once recognised. The most effectual parasiticide is the white precipitate ointment, and if only applied to the shoulders no harm will ensue; when used more freely and extensively it may cause salivation. Inunction, as used with grey ointment in cases of syphilis, is quite unnecessary; it is enough for the parts to be smeared over. Carbolic acid lotion (5 p. c.) or diluted creasote ointment relieves the itching. The clothes must be scalded or fumigated.

3. *Prurigo*—in the restricted sense.—There remain certain forms of disease which are quite independent of pediculi, but agree with prurigo senilis in the anatomical character of the papules and in the excessive itching to which they give rise. One form is the prurigo of Hebra, a striking description of which is given in the Sydenham Society's translation of his work (vol. ii, p. 258). He admits milder cases which correspond to the prurigo mitis and formicans of Willan, but would separate them broadly from the severe form which is congenital and incurable. The writer saw at Vienna cases of this "Hebra's prurigo," as it has been called, and ventures to think that their characters were somewhat over-described, if not exaggerated. At all events, cases have been described, both in America

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and in England, which agree with Hebra's cases in all essential particulars, and which would make an uninterrupted series connecting the worst of those in Vienna with the slightest forms of infantile prurigo.* We may therefore fairly include these affections under a common name, using such adjectives as *mitis*, *gravis*, *agria*, *congenitalis*, *infantilis*, *inveterata*, to denote the varieties which we find in practice.

The papules of prurigo are at first scarcely distinguishable in colour, and, as Hebra says, are felt rather than seen. They are not closely set, and do not appear in patches; they produce great itching, which causes black spots and scratch-marks as in prurigo pedicularis. The skin between is more or less pigmented, and is generally covered with a fine branny desquamation. In course of time it becomes thick and indurated, and in many cases there is traumatic eczema, often of a pustular kind. In severe cases, inflammatory enlargement of the lymph-glands occurs both in the groin and the axillæ.

The distribution of prurigo is over the trunk and limbs. The face is almost always free, and also the flexures of the joints, palms, and soles. It is generally most severe on the back, chest, and abdomen, on the buttocks, the shoulders, and upper arms; and it is generally worse on the lower than on the upper extremities, and worst of all below the knee.

Prurigo begins in early life, and either disappears during childhood, or if present in an adult, has persisted from that period. It is generally worse in winter. In one exceptional case it began in a lad at the age of fourteen. It first appeared on his legs, and affected the whole surface except the head, palms, soles, and flexures. There were a few spots on the cheeks and neck, on the hands and feet, and on the penis; the trunk was moderately affected, the buttocks and thighs more so, and the arms and legs most of all. There were severe buboes, and he was thin and wasted. He improved greatly under treatment, but the disease returned from time to time.

In twenty consecutive cases observed, the ages of the patients when first seen by the writer were—under twelve months two, between two and five years eight, between five and ten one, between ten and fifteen five, between twenty and forty-five three. It is more common in men and boys than in females. In long-standing cases the skin is often much thickened as well as pigmented.

The *treatment* of prurigo, even in its most typical and severe form, is far from being as hopeless as Hebra supposed. Frequent warm baths and assiduous inunction, together with arsenic internally in steadily increasing doses, with cod-liver oil and good feeding, will often restore even inveterate cases to health and comfort. It is, however, almost certain to return, probably more than once, and must be kept at bay for years before it finally disappears.

The slighter forms of true prurigo in infants and children are very much aggravated by scratching, and the first point is to prevent this by hydrocyanic lotion or other local anodyne, and by sedatives at night, as described under eczema (pp. 779, 780). In some cases quinine appears to be almost a specific, both for the irritation and the disease.

Locally ointments are usually more effectual than lotions in allaying the itching. *Ung. hyd. ammoniati* or *Ung. hyd. ox. rubri* is particularly useful for circumscribed regions, as in pruritus ani. *Liq. carbonis detergens* (ʒj—ij ad ℥j) is often serviceable in addition. Of local anodynes cucain is more generally efficient than opiates.

* See, on this point, a paper by Mr Morrant Baker, 'Internat. Med. Congr., 1881,' vol. iii, p. 177, and the discussion which followed.

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PSORIASIS

(*DRY OR SCALY TETTER*)

"Durateque cuti squamas increscere sentit."
OVID.

Frequency—Name—Anatomy and histology—Course—Symptoms—Distribution—Ætiology—Varieties: guttate form, inveterate form—Relation to pityriasis rubra, to eczema, and to syphilis—Prognosis—Treatment.

EXCLUDING scabies and syphilis, by far the most common cutaneous disease is eczema; next comes acne, and then psoriasis. Like the affections hitherto described, it is a chronic superficial dermatitis, and like them has been described as a dartrous or herpetic affection. It stands, however, at the opposite extreme from typical idiopathic vesicular eczema, with which it offers points of contrast rather than of resemblance.

An old and good name for psoriasis was "dry tetter." The Greek term signifies the condition of *psora* or itching, and has no bearing on the present signification of the term. Certain forms of psoriasis were formerly known as *lepra*, a term which from its etymology, "the scaly disease," would be more appropriate, but the confusion with leprosy is decisive against the word. The Greek term *alphos*, referring to its white scales, was revived by Erasmus Wilson, but without general acceptance.

Anatomy.—Psoriasis is an extremely well-marked and characteristic form of disease. It begins as papules, which rapidly increase in size, and form flat patches. From the beginning white scales can be seen upon the papules, and by the time they are as large as a pea the scales form conspicuous shiny spots (*Ps. guttata*). They are large, perfectly dry, strongly coherent, and not easily separable from the skin; they have also a characteristic white silvery lustre, due to the abundance of air which is included between the layers of horny epidermis. When the scales are removed, the surface on which they rest is seen to be red, shining, and dry, but the injection is not that of acute hyperæmia, and either stops at the edge of the scaly patch or only extends very slightly beyond it.

Histology.—The earlier dermatologists of the present century, Gustav Simon, and even Hebra, were unable to prove what they recognised as probable, that psoriasis is essentially a form of dermatitis. As with eczema and most other cutaneous affections, the characteristic appearance is lost after death. By the better methods of modern histology Neumann established the existence of abundant cellular infiltration of the papillæ of the corium, extending along the tracks of blood-vessels in its deeper layers. The papillæ are enlarged to ten or twelve times their natural size, and this papillary hypertrophy is present from the first, not only, as with eczema, in the later stages. The scales of psoriasis, like those of pityriasis rubra, consist almost entirely of keratin—unmixed with fibrin and leu-

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cocytes, as in chronic eczema and syphilis, or with sebum, as in pityriasis capitis.

Local evolution.—Psoriasis is no less characteristic in the regions it affects than in its anatomical lesion. Its favourite spots are over the olecranon process of the ulna and over the patella, ligamentum patellæ, and tubercle of the tibia. In fact, it is remarkable how very rarely these spots are found free even in the most chronic and varied forms of the disease. Here it begins and here it almost always remains. From these points it spreads downwards over the extensor surface of the forearm and on the shin and calf, not, however, by a continuous advance, as is the case with eczema, but by the development of separate discs with well-defined margins (*Ps. nummulata*), which as they increase in size become confluent with the originally diseased surface. The whole upper and lower extremities may be covered with such patches, which increase by coalescence; but there will always be found more or less extensive islands of healthy skin between the diseased parts, and these will have a concave, while the scaly patches have a constantly convex, outline. On the back or chest the same process is seen on a larger scale.

As the raised, red, and scaly edge of the eruption advances, the inner parts, which were first affected, lose their scales and return, more or less incompletely, to a healthy condition; so that by this progressive spread and involution of the disease the scattered scaly patches in which it began gradually give place to extensive surfaces of almost normal appearance, bounded by sinuous lines of red and scaly skin, made up by the intersecting segments of many circles. *Psoriasis gyrata* was the technical term applied to this stage.

After psoriasis has lasted for some time, its colour begins to acquire a deeper and brownish tint. It no longer disappears completely upon pressure—that is to say, pigmentation has been added to hyperæmia. In inveterate cases this becomes very characteristic, the colour being of a deep brown, sometimes almost mulatto tint. When the disease has been cured, when the scales are removed, the hyperæmia has subsided, and the finger cannot feel anything but healthy skin, dark pigment-blotches remain to attest the nature of the recent malady. They always disappear in time, but, especially in old persons, their disappearance is slow. It may be said that, next to syphilis, psoriasis produces pigmentation more quickly than any other form of dermatitis, and the depth of pigment may be as great as in the most chronic cases of eczema or of prurigo senilis. In this, as in other respects, psoriasis resembles lichen planus, and differs from pityriasis rubra.

Course.—Psoriasis is never acute. Even when it develops rapidly it is unaccompanied by the ordinary symptoms of inflammation, and never causes constitutional disturbance. Often a patch on each elbow, or on the elbows and knees, may appear and remain for years before it shows signs of spreading. When it has become extremely diffused and passed through the centrifugal process above described, it will, if untreated, enter upon a very chronic and almost interminable course, the skin being habitually thick, harsh, and dry, and the general aspect resembling that of some of the forms of dry scaly chronic eczema in old persons.

Of all skin affections psoriasis is most prone to *recur*, more so even than eczema. It is very rare for a single outbreak to occur. Sometimes, when the eruption has only just disappeared under treatment, a fresh attack comes

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on, and the very means which will almost infallibly cure it when developed are often powerless to prevent its return.

Notwithstanding its etymology, itching is comparatively unimportant as a symptom of psoriasis; it is much less severe than in eczema, scabies, or prurigo. In many instances there is no irritation at all; in most it is slight, but in a few it is sufficiently troublesome to demand special treatment. It is still more rare for the affected parts to smart or to feel hot and tender. Though pathologically it is an inflammation, it is the most chronic, cold, and uninflammatory of all kinds of superficial dermatitis.

It produces no constitutional effects, and persons subject to it are entirely free from special liability to any other disease. The digestive, urinary, and other functions are carried on as usual, unaffected by the condition of the skin.

Distribution.—Psoriasis is of all diseases the most completely and constantly symmetrical; not more so, it is true, than typical forms of eczema, but its range is so much more restricted, and its varieties so unimportant, that while typical eczema does not include three fourths of the whole number of eczematous cases, we seldom meet with one of psoriasis which deviates from the characteristic type. As above stated, its favourite or practically its constant seat is upon the two elbows and the two knees; next it is common over the whole extensor surfaces of the extremities, specially the forearm, the front and outside of the thigh, and the peroneal side of the leg. Even when most extensive it shuns the bend of the elbow and the popliteal space. It not unfrequently extends from the forearm to the back of the hand, and from the leg to the dorsum of the foot, and occasionally may cover the fingers, and even affect the nails. *Psoriasis unguium* is known by the excessive and unsightly thickening of the nail, and by the absence of soreness and suppuration of the matrix. It sometimes occurs when the rest of the fingers or toes is free from the disease, but almost always spots of psoriasis will be found on the elbows or knees, or the patient has previously suffered from the disease. The only other affections of the nails which at all resemble it are eczema unguium, above described, and onychomycosis, to be mentioned under ringworm.

Psoriasis very rarely affects the palms or the soles, and never unless other parts of the body are previously the seat of the disease. What used to be described as primary psoriasis palmaris was probably either eczema squamosum or scaly syphilis. When present, the scales of palmar psoriasis are comparatively small, but the patches keep their well-marked edge. There is little or no disposition to form cracks, and the soreness and irritation of eczema of the palms or soles is absent.

Next to the extensor surfaces of the limbs, the trunk is the most common seat of psoriasis. The shoulders, back, and loins, the sacral and gluteal regions, are very frequently its seat, the chest and abdomen somewhat less so. Indeed, we never see psoriasis of the abdomen which does not also affect other parts of the trunk, and it is very rare to find psoriasis of the trunk when the limbs are completely free. The genital organs are occasionally the seat of psoriasis, which has usually spread thither from the abdomen or the thighs; but this is far less frequent than eczema of the same parts, and what used to be called *psoriasis scroti* is really *syphilis squamosa*. The face and head are less frequently attacked by psoriasis than the trunk, far less frequently than the limbs; but with so common a disease cases often occur

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in which the red scaly patches appear upon the neck, the cheeks, the forehead, and the scalp. The scales are usually smaller upon the face, the whole aspect less characteristic, and apt to be further confused by slight ordinary dermatitis, but the presence of unmistakable psoriasis on the limbs or trunk prevents any mistake in diagnosis. On the scalp the closeness of the hairs prevents the formation of large scales, and the sebaceous secretion gives them a greasy consistence and a yellowish tint. *Psoriasis capillitii* is a not infrequent affection, and must be carefully distinguished from eczema and impetigo capitis, from syphilodermia and seborrhœa sicca or pityriasis capitis. It is always dry, the scales are coherent, the hair does not fall out, and it is coincident with existing or previous psoriasis of other parts.

The affection called *psoriasis labiorum* has been described already as a form of eczema. It is doubtful whether psoriasis affects the mucous membranes. *Psoriasis linguæ* is *leukoplakia*, white patches on the tongue, distinct from syphilis and often antecedent to epithelial cancer; when it is coincident with an affection of the skin, that affection is usually lichen planus. The occasional coexistence of these patches with ordinary psoriasis must be admitted; but the question is whether the coincidence is accidental or not.

Ætiology.—The cause or causes of psoriasis are absolutely unknown. By French writers it is generally ascribed to a dartrous diathesis, and by those who go still further into speculation a *dartrous* is distinguished from an *arthritic* psoriasis. In England it is very commonly regarded as *gouty*, while by some authors it is considered, especially in children, as a *scrofulous* disease; and there is about the same evidence for the one hypothesis as the other. Dr Yandell regards a great deal of psoriasis as well as of eczema as *malarial* in origin; and in the last century psoriasis was by most physicians considered undoubtedly *scorbutic*. Some writers have speculated on the possible connection of psoriasis with *leprosy*, and would have us regard it as the expiring and gradually mitigated manifestation in modern times of the scaly leprosy, "white as snow," which is described in the Old Testament. The remarkable centrifugal progress above described naturally suggests the idea of a parasitic vegetable growth; but we may confidently assert that no fungus is present, and although it has been asserted that *bacteria* may be found in the affected skin when the scales have been removed, their presence, which is certainly not universal, must be regarded as purely accidental, and would neither explain the course and spread of the disease nor help in its treatment. So unwelcome is it to admit a disease without a cause, that some dermatologists refuse the title to psoriasis, and regard it as a condition of health.

With respect to gout it must be remembered first that the word, in Germany and even in England, is often applied upon very insufficient evidence. In fact, those who use it would sometimes not even imply the presence of urate of soda in the joints. Moreover, the diagnosis of gout is always acceptable to an Englishman of the middle class. On the other hand, it is true that few families of rank in this country are free from unmistakable gout in some of their members. But psoriasis is comparatively rare in private practice as compared with that of hospitals. Very few of those who have unmistakable podagra are liable to psoriasis; and psoriasis is as common in Scotland, Germany, and America, where gout is rare, as in England, where it is frequent. If we determine that every disease must have for cause some condition already known, it will be easy to find one in the list given above for every case of psoriasis; but such a practice

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hinders the progress of knowledge of the real causes of disease, interferes with rational and successful treatment, and leads to an acquiescence in unsupported statements and fallacious arguments which is fatal to medicine either as a science or an art.

Psoriasis occurs equally in both sexes and at all ages above infancy; it becomes more common from the age of six or seven up to puberty, and the first attack usually falls in childhood or early adult life. It may, however, begin after fifty and even in old age.

The present writer published in the 'Guy's Hospital Reports' (vols. xxv, p. 243, and xlvi, p. 419) a tabulated statement of 267 cases of psoriasis observed by him. Of these patients, 145 were men, and 122 women. Their ages varied from four to seventy-two, but in many cases the disease has begun long before the patient appeared. There were 22 patients under ten years old, 35 between ten and sixteen, 32 between sixteen and twenty, 66 between twenty and thirty, 23 between thirty and forty, 25 between forty and fifty, 23 between fifty and sixty, 22 between sixty and seventy, and 3 over seventy.

Varieties and relations.—As above stated, psoriasis compared with eczema is singularly uniform in its anatomy and natural history. The description above given applies to ninety-nine out of a hundred cases, of course with individual variations, but less than those of even such typical diseases as typhus and variola. There is, in fact, only one variety which demands notice: in *children* the ordinary form is frequently seen, but more commonly the early spots of *Psoriasis guttata* do not grow into the nummular stage, and the large patches of gyrate psoriasis are decidedly rare under puberty. This would be scarcely worth mentioning in itself, but the spots are also remarkable for having little or no red border. They produce no irritation, and—as is common with other diseases affecting children—the local distribution is less rigidly marked than in adults (p. 759). The guttæ are frequently seen on the face, and they are perhaps more abundant on the trunk than on the limbs. Dr Liveing thinks, moreover, that guttate psoriasis occurs particularly in children who are scrofulous, *i. e.* pale and thin. It is, however, often seen in those who are robust, and certainly in most cases there is no chronic enlargement of the lymph-glands, no caries, no chronic synovitis, and no evidence of tubercle.

When psoriasis has lasted for many years and has spread over the greater part of the surface, it loses much of its characteristic appearance, the scales are less abundantly formed, the margins are less definite, and the whole skin becomes thickened and indurated, so that it often requires careful investigation and a knowledge of the earlier stages of the affection to distinguish this *psoriasis inveterata* from the dry and chronic eczema described in a former chapter.

There is no question that psoriasis may pass into, or be supplanted by, the dry, scaly and universal dermatitis to be described in the next chapter as pityriasis rubra. As is there stated, the late Dr Baxter thought that any dermatitis—eczema, psoriasis, lichen, or pemphigus—might, if sufficiently extensive, assume the characters of that remarkable disease. In a paper in the 'Guy's Hospital Reports' (vol. xxv, p. 266), reasons are given against accepting this hypothesis. The writer had a remarkable case in which a woman, who had been in St Thomas's Hospital under Dr Payne with ordinary psoriasis of the elbows and knees, and whose daughter was a patient of his own, also with psoriasis, came under his care with marked and typical

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pityriasis rubra. It is very probable that some at least of the cases of general psoriasis, described by Hardy as very rare, would have been recognised by Devergie as pityriasis rubra.

One may admit that eczema and psoriasis, which in so many ways are allied by points of contrast, have connecting links on the one hand with pityriasis rubra, or universal exfoliative dermatitis, and on the other with lichen planus, which, as we saw, sometimes so closely resembles psoriasis, while by its relation to ordinary forms of lichen it has affinities with papular eczema. We draw lines as nearly as we can in accordance with the broad demarcations of pathology and natural history, but here as in other departments of medicine it would be pedantry to deny that there are transitional forms which it is difficult or perhaps impossible to classify.

The important question, however, of the diagnosis between psoriasis and the scaly forms of syphilis is one which rests on the absolute distinction of cause, and is, therefore, of the utmost practical importance. The locality and symmetry, the character of the scales, the colour, the presence of itching, the uniformity of the lesion, and the absence of other signs of syphilitic disease are the points to be attended to. The last, however, may deceive, for a man with psoriasis may acquire syphilis, as he may scabies; and the writer has notes of four or five cases in which true psoriasis and a secondary syphilitic eruption existed in the same patient, ran independent courses, and were cured by different treatment.

Prognosis.—Psoriasis, if left to itself, lasts for an indefinite time, though almost always getting better or worse at intervals. It never interferes with the health or affects other organs than the skin. After being cured it is of all cutaneous diseases most apt to return.

Treatment.—The external treatment of psoriasis consists in inunction of some preparation of tar. Nothing is so effectual as the *unguentum picis liquidæ* of the Pharmacopœia well rubbed in at night, and allowed to stay on while the patient sleeps in a special suit of underclothing; it may then be washed off in the morning, to be reapplied at night. When the scales are very thick and indurated, it may be well to precede this application by the use of hot baths and soft soap. When the smell and colour of tar are objected to, useful though less efficient substitutes may be found in the liquor carbonis detergens, made into an ointment with lanoline (one, one and a half, or two drachms to the ounce), or in the *huile de Cade*, ung. creasoti, oleum rusci, &c. Another plan of obtaining the same result is to apply a spirituous solution of tar or the liquor carbonis detergens diluted with water. Goa powder, and the chrysophanic acid or chrysarobin which it contains, are powerful cutaneous stimulants, and have been often used with success in the treatment of psoriasis. They occasion, however, with many patients, considerable pain, and stain the skin and linen unpleasantly, as well as the hands of the person applying them.* In cases where tar is inadmissible, a better substitute is pyrogallic acid, gr. xv—xxx to an ounce of benzoated lard or lanoline, the strength being increased with caution. For rapidity

* Dr Crocker writes ('Brit. Med. Journ.,' November 19th, 1887): "The stains in linen are quite indelible without injuring the fabric, but they may be avoided by using the Anspitz method. A gutta-percha varnish, called traumaticin, is made by dissolving ʒj of pure gutta percha in ʒx of chloroform, ʒj of chrysarobin is added, making an emulsion, which is painted on with a stiff brush after removing the scales every day, until a thick coat is formed; it is then allowed to peel off and is renewed. It acts effectually and does not stain. Besnier modified this by brushing in ʒj of chrysarobin in ʒx of chloroform, and then varnishing with traumaticin. Both plans are equally good."

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of cure, with freedom from unpleasant smell, this is perhaps the most eligible of all applications.

Beside the local treatment, it is almost always desirable, after the scales have been thus removed, to put the patient upon a course of *arsenic*. It is usual to prescribe it in a bitter infusion, but it will be found to agree quite as well, and to be more constantly taken, if merely diluted with water or flavoured with syrup or peppermint. It should always be taken at or immediately after a meal. Three, four, or five drops in an ounce of water three times a day is the dose to begin with, and it may be increased to ten or beyond. If properly diluted, and taken with food, even full doses very rarely cause pain, sickness, or diarrhœa. The first sign of the physiological limit being reached is usually irritation and slight injection of the conjunctiva. As soon as the patient feels his eyes begin to itch, he should be instructed to leave off his medicine for a couple of days, and then resume it in slightly smaller doses. He has then reached what is for him the full therapeutical benefit.

Psoriasis may often be cured by arsenic without any external application whatever, or by local treatment, without internal medication; but in most cases the cure will be hastened by the application of tar, and will be rendered more permanent by the administration of arsenic.

In the cases of anæmic persons it is desirable to give steel. We may then combine the liq. arsen. hydrochlor. with the liq. ferri perchlor. In other cases Fowler's solution acts better alone. It is the fashion to administer Pearson's solution (the arseniate of soda) to children, but there is little doubt that it is absorbed in exactly the same form. When arsenic disagrees it should not be hastily given up, but the dose should be diminished until unpleasant effects no longer follow, or we may sometimes prevent them by adding a few drops of laudanum or a little compound tincture of camphor to the dose. When the patient suffers from gastritis and sore eyes, four drops of Fowler's solution—three, or even two—will probably be sufficient to cure the psoriasis; when a patient can take ten without discomfort, it may be that fifteen will be needful.

Children take arsenic very well. When they are pale and thin and ill-nourished, cod-liver oil is often a useful coadjutant. In the guttate form of the disease, most common under puberty, local treatment is often scarcely required.

Purgatives and diuretics are quite unnecessary; and colchicum is not needed, as it undoubtedly is in the treatment of certain irritable and probably gouty forms of eczema.

In some cases psoriasis is very obstinate, not only returning again and again after being cured, which is common, but yielding very little to the most careful local measures and to the most persevering use of arsenic. In these cases liquor potassæ in half-drachm doses sometimes succeeds, sometimes iodide of potassium* and other diuretic remedies, and sometimes prolonged maceration with soft soap and water. But we occasionally meet with cases which seem to be quite incurable.

* This has been advocated by Dr Cæsar Boeck and other physicians in Norway and Denmark. Dr Haslund gives enormous doses without harm (see 'Brit. Med. Journ.,' 1888, vol. i, p. 27).

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PITYRIASIS RUBRA

(*GENERAL EXFOLIATIVE DERMATITIS*)

> "Which, like a searching tetter uncorrected,
> Left no part of his body unaffected."
>
> QUARLES, *Job militant.*

Name and traditional species of pityriasis—Pityriasis rosea vel circinata. Pityriasis rubra—History—Accounts by Devergie, Hebra, Wilson, Hutchinson, Baxter—Course and symptoms—Histology—Prognosis—Diagnosis—Treatment.

THE word "Pityriasis," meaning, as its etymology implies, a branny or furfuraceous desquamation of the skin (πίτυρα), is conveniently used to describe that pathological condition, but no one *disease* is entitled to the name.

The species defined by Bateman as *Pityriasis capitis* is in most cases *Seborrhœa sicca*, an affection of the sebaceous glands of the scalp; or it may be slight local dermatitis (*Eczema capitis*), due, as he remarks, to want of cleanliness and removable by soap and water, but apt, if neglected, to degenerate into "Porrigo," that is to become pustular eczema or impetigo of the scalp. Or, again, pityriasis capitis may be a slight form of psoriasis capillitii.

Pityriasis versicolor, now known as tinea versicolor, is a parasitical disease, and will be described in a subsequent chapter.

Pityriasis nigra, described by Willan as occurring in children born in India, was not identified by Bateman, nor probably by anyone else. A case of Alibert's, which Devergie calls "pityriasis nigra with prurigo," was apparently *Prurigo pedicularis* with pigmentation and leucodermia.

Bateman's fourth species, *Pityriasis rubra*, "resembling psoriasis diffusa," is a stage in the involution of eczema.

Pityriasis rosea is the name given by Gibert to a trivial affection of the skin which he regarded as erythema. It has been named by other French writers *Pityriasis rubra maculata*, or *P. maculata et circinata*. Hebra and some other authorities have regarded it as a form of ringworm of the body (cf. p. 867), but for this opinion there is no adequate evidence. Dr Liveing calls it Roseola circinata.

At present it may find a place in this chapter, as a form of superficial dermatitis, with a centrifugal mode of spreading, a branny desquamation, and a somewhat characteristic colour, localisation, and course.

It begins as bright rose-coloured patches very slightly raised above the surface, which rapidly extend so as to form fresh discs, then circles, and, lastly, gyrate bands. In this respect it resembles psoriasis as well as ringworm. As the border advances, the central part fades into a yellowish stain (*P. maculata*) and the branny desquamation disappears.

The favourite locality of this eruption is the chest or abdomen; but it

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sometimes spreads to the neck and face, the buttocks, and the proximal part of the limbs.

It usually passes away of itself after a few weeks, and needs little or no treatment, for the irritation is but slight.

It has a certain resemblance to Lichen circumscriptus vel circinatus, to Tinea versicolor, to T. corporis, and to an early syphilitic rash.

PITYRIASIS RUBRA—*Exfoliative dermatitis.*—The term "pityriasis rubra" was unluckily applied by Devergie in 1854 to a severe and remarkable form of superficial dermatitis which certainly deserves a special name. It is probably identical with Alibert's "Herpes squamosus."* Hebra in 1860 thought himself bound to follow Devergie's nomenclature, and his authority has led to the term P. rubra being generally accepted. Wilson's proposed names of "Pityriasis foliacea rubra" and "Eczema foliaceum" (1876), or the better title, "Exfoliative dermatitis" (1870), have not displaced the original term. *Universal exfoliative* (or *desquamative*) *dermatitis* is perhaps the title that would most clearly express its characters.

History of the disease.—Devergie ('Traité Pratique des Maladies de la Peau,' p. 263) describes the disease as beginning with an erythematous redness, usually on the chest or flexor surface of the limbs, and spreading rapidly, with a well-defined margin, deep colour, abundant scales, and more or less thin serous discharge. It covers the whole body, is very obstinate, lasting for months, and occasionally proves fatal by exhaustion and diarrhœa. As a rule, however, patients slowly recover. Relapses are frequent. Devergie admits the difficulty of distinguishing this new disease from eczema, and bases the diagnosis on the following points:—It is of a deep red colour, it has sharply marked borders, it may affect the whole surface; the skin, and even the subcutaneous fascia, are thickened; it is less itching, more burning than eczema; its secretion is thin, and does not stiffen linen; the scales are abundant, readily detached; and from the first no red moist points (*état ponctué*) are seen when the scales are removed.

In the 'Glasgow Medical Journal' for January, 1858, p. 421, Dr McGhie recorded a case of "pityriasis rubra acuta" which he rightly regarded as one of Devergie's disease. This seems to be the first published in this country, and preceded Hebra's cases. The same patient's condition was described by Professor Gairdner ('British Medical Journal,' March 13th, 1875, p. 359) seventeen years later. Among the early cases may be mentioned one by Dr Wilks in the 'Guy's Hospital Reports' for 1861, which he called "general dermatitis;" the eruption was universal, red, and dry, with abundant desquamation. Another was recorded by the late Dr Hillier ('Handbook of Skin Diseases,' p. 101) in 1864, and another by Dr Fagge in the 'Guy's Hospital Reports' for 1876, vol. xiii.

Some authors regard pityriasis rubra as essentially Eczema squamosum, and one of Mr Wilson's titles is Eczema exfoliatum. Dr Liveing agrees with Wilson and Fagge in looking on it as only a peculiar form of eczema. Mr Hutchinson ('Lectures on Clinical Surgery,' part i) would separate pityriasis rubra from eczema, and regard it as the type of a group of affections which differ in anatomy, but agree in being universal, in resisting treatment, and in often proving fatal. This would include *Pemphigus foliaceus* with certain cases of psoriasis and lichen.

* The case to which the name pityriasis rubra was first applied by Cazenave seems to have been *Tinea versicolor*, with more irritation than usual.

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The late Dr Baxter had previously published a valuable paper on this disease under the title of "General exfoliative dermatitis" ('British Medical Journal,' July 19th, 1879). He considered the affection as the result of a universal inflammation, and as arising by the general diffusion of either eczema, psoriasis, lichen, or pemphigus. The objections to this view are that eczema may be nearly if not completely universal, and for long periods together, without losing its characteristic features and without endangering the health. The same appears to be true of *Lichen planus*, if we accept Hebra's descriptions of universal chronic *Lichen ruber*; for this he carefully distinguishes from pityriasis rubra. *Pemphigus foliaceus* is seldom if ever universal, and differs markedly, as will be seen hereafter, from pityriasis rubra. That the whole skin may be occupied by a scaly eruption without interference with health is proved by many cases of ichthyosis.

Auspitz, who is followed by Hans von Hebra, separates pityriasis rubra from the inflammatory diseases, and places it among affections of the epidermis (keratonoses) as *keratolysis*, a somewhat arbitrary decision.

Origin, course, and characters.—Pityriasis rubra may undoubtedly arise from eczema or psoriasis, and probably from any other form of superficial dermatitis, including erythema, impetigo, and traumatic dermatitis; but it most often begins without previous cutaneous lesion.

It rapidly spreads over the trunk and limbs, but in an irregular fashion, unlike the gradual and methodical extension of eczema or psoriasis.

Finally, it affects the whole of the cutaneous surface, including the scalp, the palms, and the soles. The skin is of a full deep red colour, not thickened and indurated as in chronic eczema (the exact reverse of Devergie's statement), covered with profuse and abundant scales, which are large, thin, and easily detached, unlike those of psoriasis, or of syphilis, or the branny desquamation which follows eczema and the exanthems. They are apt to form successive undulating ridges, which Wilson compared to those of the "ribbed sea sand;" and they are so abundant that the patient's bed is filled with them by the peck.

In most cases the surface is absolutely dry; occasionally there may be a slight inflammatory exudation, especially in the flexures where the inflamed skin is apt to crack. This exudation has not the stiffening property which marks that of eczema, and it is clearly an accidental, not an essential character of the disorder.

There is more or less pyrexia and general disturbance of health, especially at the onset. If, as is most frequently the case, the disease becomes chronic and inveterate, albuminuria is occasionally observed, and the appetite and health begin to fail. The irritation varies in different cases; sometimes it is very slight, but more often it is considerable, and occasionally almost as intense as in eczema, so as seriously to interfere with sleep.

Histology.—In a case of a year's standing examined after death by Hans von Hebra the whole of the cutis was filled with leucocytes; in another case, which had lasted several years, all signs of active inflammation had disappeared; the Malpighian layer was thin and its cells shrunken, the papillæ atrophied, and the deep layer of the cutis transformed into thick bundles of elastic fibres with abundant pigment; the glands had also suffered atrophy. In chronic cases the hair may be lost.

Ætiology.—General exfoliative dermatitis is common to both sexes and

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to all ages. Though more frequent in the latter periods of life, it is not unknown in children. A certain number of cases, as above stated, begin in eczema or psoriasis, but more have no such origin. Its true causes are entirely unknown.

Diagnosis.—Pityriasis rubra is distinguished from *eczema* by its abundant scales, by the absence of visible moisture, and by its not showing predilection for the ears, face, and flexures of joints; from *psoriasis* by the thin, loose scales, and by its not specially affecting the elbows and knees; from *pemphigus foliaceus* by the scales not being preceded by bullæ; from all these forms of superficial dermatitis by its being universal and uniform in distribution, and by the severe symptoms which usually accompany it.

Prognosis.—This is much graver than that of ordinary dermatitis, eczema, psoriasis, lichen, prurigo, or pemphigus. For not only is it difficult to cure, but it sometimes ends in death, especially in elderly people. The presence of albumen is a bad sign, though not a fatal one. Emaciation is still more serious, depending, as it usually does, on loss of appetite or sleeplessness or diarrhœa. Yet the disease is not, as Hebra supposed, incurable, nor is it by any means constantly fatal. Since his book was written, cases of recovery have occurred at Vienna. In forty cases collected by the writer, from various sources ('Guy's Hospital Reports,' series 3, vol. xxv), recovery ensued in fifteen, improvement in several more, and death only in eight. In these cases the fatal event was caused by bedsores and exhaustion, by lobular pneumonia, by acute pneumonia, or by bronchitis. In other instances marasmus ensues, and diarrhœa ends the disease. *P. rubra* often persists for an indefinite period almost uninfluenced by treatment.

Treatment.—Locally the best applications are those which have been recommended in the drier forms of eczema—weak carbolic oil, lead and zinc ointment, or liquor carbonis detergens with vaseline (ʒj ad ℥j) freely and frequently applied. Tepid baths are not counter-indicated, and usually give relief, but if too warm they lead to irritation afterwards, and the effect on the pulse must be carefully watched. Arsenic has not the power it possesses with psoriasis and with chronic eczema. It is best given in small doses combined with steel. Bark and mineral acids are often useful. Milk and farinaceous diet appears to suit best, and cod-liver oil should be taken if it does not interfere with other food. Good red wine, or sometimes porter, is in certain cases decidedly beneficial. In one obstinate case the patient, an otherwise healthy old gentleman, completely recovered after six weeks' sojourn at Strathpeffer, in Ross-shire.

In another well-marked and very severe case, dry, scaly, universal, and pruriginous, in a clergyman about sixty, long perseverance with soothing ointments and increasing doses of arsenic internally, restored him, after several months, to almost complete freedom from the disease.

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PEMPHIGUS

(*BLADDER-TETTER*)

"A most instant tetter barked about,
Most lazar-like, with vile and loathsome crust
All my smooth body."—*Hamlet.*

Names and definition—Anatomy—Histology—Local distribution—Age and sex—Question of an acute form of pemphigus—Diagnosis—Prognosis—Pemphigus malignus—P. foliaceus—P. serpiginosus—Hutchinson's cases—Chiropompholyx—Hydroa of Bazin—Frequency of pemphigus—Treatment.

We now come to a form of superficial dermatitis which is decidedly rare compared with eczema or psoriasis. Although not less remarkable than these in its anatomical characters, its course and natural history are far less characteristic, its pathology more obscure, and its origin entirely unknown. It has been called by two names, *pemphigus* and *pompholyx*;* but of these terms, which, like "lepra" and "psoriasis," were made separate genera by Willan and his disciples, there is no need to retain more than one.

The name *pomphus* seems to have been originally applied to what we now call a wheal; *pemphix* meant a bulla, and *pemphigus* was originally applied to a supposed "febris bullosa" of doubtful nature. Bateman practically admits only one bullous disease, a chronic superficial dermatitis, characterised by blebs.

It was by Willan associated with erysipelas, a striking example of the ill result of following an anatomical, or any exclusive, basis of classification for so complex conditions as diseases. He had previously united it with vesicular diseases, but distinguished the two orders in consequence of the criticism of Tilesius, of Leipsic ("über die flechtenartigen Ausschläge," in Martin's 'Paradoxien,' 1801).

Anatomy.—The bullæ of pemphigus begin with a scarcely demonstrable papular stage. The first lesion seen is usually a small transparent vesicle which rapidly increases to the size of a pea or larger. These bullæ are sometimes seated on perfectly natural skin; sometimes they are surrounded by a rose-coloured injected ring, but this is narrow, and they are never found upon an actively inflamed or swollen surface. They may burst when not bigger than a pea or a marble, but, on the other hand, will sometimes increase to the size of a billiard-ball, or more. They are usually tense and hemispherical, occasionally oval. There may be either a single bleb or several of various sizes, irregularly scattered over the same region, and when in such groups the intervening skin is often injected. Each bulla, however, forms separately, and it is very rare for two to run together. The contained liquid is transparent, and gives the bulla a pearly appearance. When removed by pricking the bleb, it is thin, watery, colourless, usually not coagulating, but

* Πομφόλυξ means a bubble, almost synonymous with φύσαλις, and is applied by Hippocrates to the froth which forms on urine ('Aphor.,' vii, § 34). Πίμφιξ means a blister.

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becoming opalescent or turbid on heating, and showing a few leucocytes under the microscope. After a time, however, it often becomes turbid from increase of the inflammatory corpuscles, and before the bulla bursts the contents may be opaque and yellow—in fact, almost purulent. They do not, however, acquire the thick creamy character of pure pus, and always begin as serous and not purulent cavities. Threads of fibrin also appear, not unfrequently, before the rupture of the vesicle. Still more common is an admixture of blood, which gives a pinkish aspect to the bulla. After it has burst, fresh secretion soon ceases, the ruptured cuticle is either torn off or adheres to the exudation, and the lymph, whether serous, puriform, or coagulated, dries up into a thin yellow crust, which may be more or less stained by hæmoglobin. This soon falls off and leaves a smooth, healthy surface, with scarcely any desquamation; but some passive injection remains, and with this may be mingled more or less pigmentation, so that the circular patches, of sizes varying from a sixpence to a florin, remain for some time as characteristic evidence of pemphigus.

Histology.—The inflammatory exudation of a bulla produced by such an irritant as cantharides takes place in the deepest part of the Malpighian layer of the epidermis. The cells of this layer are first drawn out into bands by the accumulating serum, so that in the early stage each vesicle consists of a series of loculi, as in the case of a burn, first described by Biesiadecki; this stage is long and well marked in the case of traumatic bullæ and in the vesicles of smallpox.

The bullæ of pemphigus, however, as shown by Leloir and Auspitz, consist of an exudation of serum between the horny layer of epidermis and the deeper Malpighian layer, or between the granular layer and those beneath it, without the cells themselves being much affected. There is no vacuolation, the cavity is at no stage multilocular, and the formation of the bleb is far more rapid than that of an inflammatory vesicle or pustule of half its size.

No scars are left after pemphigus, but there is often some degree of pigmentation, and in long-standing cases this may become deep and extensive.

Distribution.—Pemphigus differs from eczema and from psoriasis in being *unsymmetrical*, and having no definite local predilection. The bullæ appear sometimes singly (*P. solitarius*), or succeed one another indefinitely upon distant parts of the body (*pompholyx diutina*); more often two or three up to half a dozen form an irregular patch; and isolated bullæ, or one or two other patches follow on other parts of the surface. Occasionally the trunk and limbs are so covered that scarcely any region can be said to be entirely free, yet even then the lesions show no preference for one part over another.

There is scarcely any part of the surface on which pemphigus may not be seen. On the trunk and limbs it is most frequent; the abdomen and thighs, the genital organs, the ears, the hands and the feet, even the palms and soles and the matrix of the nails may occasionally be the seat of pemphigus; the hairy scalp is least frequently affected. Bullæ have been observed in the mouth and on the conjunctiva, and in one case of the writer's the latter complication was present.

Age and sex.—Pemphigus, though belonging to the rarer diseases of the skin, may be seen in patients of almost any age. It is commonest in children, decidedly infrequent in adults, but may sometimes be observed in elderly patients, when it is apt to assume its more severe characters. Among

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38 consecutive cases of the writer's, 10 occurred in males, and 28 in females; 7 between one and five years of age, 15 between six and ten years, 11 between nineteen and fifty, and one in an old woman of sixty-eight.

Diagnosis.—The bullæ of this disease are so characteristic that it cannot be overlooked, and cannot be mistaken for eczema, lichen, psoriasis, or any other of the forms of superficial dermatitis already described. But all bullous eruptions are not pemphigus.

1. Blisters may be produced designedly or accidentally by local irritants, especially by scalding water, or by cantharides. The traumatic bullæ which follow extreme heat are of two kinds—true inflammatory products containing serum or pus, and bladders filled with gas, which have been formed by the lymph of the living skin being turned into vapour and expanded by heat. The latter condition was long ago described by Hilton as the result of burns and scalds; it is of rare occurrence. Its purely physical nature he proved by the fact which the writer has himself verified, that it is possible to produce it in the skin after death. If a hot iron be held close to the surface, the cuticle rises in a blister like that produced by the sun on a painted board, and on pricking it, no liquid is found within. This is strikingly seen in a negro's skin, when the white cuticle is raised from the dark rete mucosum beneath.

Factitious inflammatory bullæ are usually seen on the arms, and in doubtful cases the glistening scales of the elytra of the Spanish fly may often be distinguished by a lens.

2. Scabies is sometimes accompanied by bullæ, especially in children. An example of this was figured by the writer in the face of a child whose appearance closely simulated that of pemphigus ('Guy's Hospital Reports,' 1877, pl. i), and another was recorded by Dr Fagge (*ibid.*, 1870, p. 333).

3. Syphilitic eruptions in the later stage of the disease are often bullous. Usually the exudation becomes purulent and the resulting crusts are massive, dark from blood-pigment, and more or less conical, forming the condition described as "rupia," and leaving a superficial ulcer when they fall off, with considerable pigmentation and final cicatrisation. In cases of congenital syphilis, however, bullæ exactly like those of pemphigus may be observed; so-called *pemphigus neonatorum* is probably always syphilitic. Besides other signs of the congenital disease, the appearance of the bullæ upon the palms and soles is a character which is diagnostic.

4. More difficult of distinction from true pemphigus are the bullæ of certain forms of erythema, to be presently described as herpes iris and erythema bullosum. Their locality and symmetry, their multiformity, and their acute or subacute course, are the chief marks which distinguish these erythematous bullæ from true pemphigus.

5. Iodide of potassium occasionally produces blebs, along with other lesions, which have been mistaken for those of pemphigus.

Ætiology.—The cause of pemphigus is absolutely unknown, although, as in other cases, teething, gastric irritation, excess in diet, irritability of the system, mental affections, anxiety, fatigue, amenorrhœa, exposure to cold, and residence in damp situations have been confidently stated as each a cause of the disease. According to Alibert, the "lymphatic temperament" predisposes to pemphigus, which perhaps meant what is true, that it is more common in children than in adults.

It is now well established that pemphigus is never contagious. Hebra relates one remarkable case of heredity.

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Some cases appear by their distribution to depend on peripheral neuritis, and such tropho-neurotic cases would connect pemphigus with zona (so Schwimmer, of Buda-Pesth, and some other authorities). In other cases micrococci have been found in the contents of the bullæ, but they have often been sought for in vain, and there is no reason to believe that they are specific.

Prognosis.—In children pemphigus is rarely fatal (excluding so-called *syphilitic pemphigus*), and under suitable internal treatment it is in most cases quickly curable. But in old persons it is apt to spread very widely; sleeplessness and loss of appetite follow, and death may result. This is most to be feared when there is chronic renal disease present, but it may occur independently of this complication.

Such cases have been made a distinct variety, *pemphigus malignus vel cachecticus.* The bullæ are very numerous, are never tightly filled with serum, but look flaccid and rupture early. There is little effort at healing, and extensive raw patches cause much pain and distress, combined sometimes with more or less itching. The exudation is frequently hæmorrhagic and sometimes fibrinous, or, as German writers call it, "croupous." As in other severe and extensive forms of dermatitis there is sometimes albumen in the urine, independently of previous Bright's disease.

These serious cases, though rare except in old persons, may occur at any age; in children, bullæ after bursting are sometimes succeeded by gangrene, and this *Pemphigus gangrænosus* has also been separately described. It has no doubt been frequently confused with what used to be called "rupia escharotica" and "pemphigus neonatorum," that is to say with a bullous syphilitic eruption. But there is no question that true gangrenous pemphigus does occur. In a little boy aged four, who died of it in Guy's Hospital in 1882, we found *post mortem* all the viscera perfectly normal, and there was no reason to suppose the presence of congenital lues. In one case it occurred after varicella.

Acute pemphigus.—Hebra discusses the existence of acute or febrile pemphigus (*febris pemphigodes*), and, like Bateman before him, concludes that when urticaria and herpes iris, erysipelas and rupia, with other forms of syphilis are excluded, there is no such disease. The late Dr Sparks, however, in 'Quain's Dictionary,' rightly stated that the existence of such cases is now certain. Dr Southey has recorded a case ('Clin. Soc. Trans.,' viii, 1875), Dr Payne another, with a table of temperature ('St Thos. Hosp. Rep.,' vol. xii), and Sir D. Duckworth a third ('St Barth. Hosp. Reports,' vol. xx). The last occurred in a man of fifty-four, suffering from Bright's disease, and he died on the ninth day; but the event was probably not due to the eruption on the skin, and if not cut short by death this might have proved chronic. Moreover, it is possible that this as well as other cases might be interpreted as bullous erythema, though it would doubtless be difficult to maintain the distinction in every instance.

Pemphigus foliaceus.—Cazenave described under this title a remarkable form of cutaneous disease which has since been recognised by Hebra and other dermatologists. It is rare, and the writer has only seen two well-marked cases, one at Vienna, and the other in Guy's Hospital, which was modelled for the museum ('Catalogue,' p. 94).

The patients are usually adult women. The blebs appear at first like

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those of ordinary pemphigus, but they never become tense and pearly in appearance. They rupture early, and form thin, dirty-white laminæ, which continue to exude a scanty secretion. The aspect of the affected skin has been likened to that of flaky pie-crust, to birch-bark, and to dead leaves—whence the specific name.

Beside the anatomy, the distribution of this form of pemphigus is remarkable, in being more or less universal. On this ground and its malignancy the late Dr Baxter associated it with pityriasis rubra.

Its course is very slow, and there is no disposition to recovery. Indeed, it is doubtful whether any genuine case of pemphigus foliaceus has ended favourably. Drugs have little or no influence upon it, and after a protracted illness the patients die emaciated, or are carried off by some intercurrent disease.

Serpiginous pemphigus is a rare form, which arises only in chronic cases. The bullæ, which are small, are seen on the red advancing border of a considerable space of skin, formerly the seat of others that have disappeared. When first seen in this latter stage it might well puzzle an observer. A well-marked case of pemphigus serpiginosus, which began as an ordinary case, recurred, and each time was cured by arsenic. It furnished two of Mr Towne's models in the museum of Guy's Hospital ('Catalogue,' p. 92).

Pemphigus vegetans.—Neumann has described under this title a bullous disorder in which the bursting of the bullæ is followed by the appearance of granulation masses (*i. e.* ulcers covered with framboesiform papillary growths). The cases were not benefited by arsenic, and ended fatally. Eleven cases have now been recorded in Germany, one by Dr Crocker ('Med.-Ch. Tr.,' March 12, 1889), one by Dr Mapother, of Dublin (*ibid.*), and another by Mr T. P. Lowe, of Bath ('Lancet,' 1891, vol. i, p. 1046).

Hutchinson's bullous disease of hands and feet.—A curious form of bullous eruption, which may be provisionally called pemphigus vegetans, was shown by Mr Hutchinson at the Pathological Society, as "hand-foot-and-mouth disease." Beside the bullæ on the trunk and limbs there was severe inflammation of the hands with loss of nails, and also inflamed mucous membrane of the mouth and tongue. A case was exhibited at the Dermatological Society in 1885, which was recognised by Mr Hutchinson as of the same character. There was unmistakable pemphigus here, and both loss of nails and sore mouth in pemphigus have been described by Hebra, so that no doubt he would have included under that title the curious cases described. Moreover, the same combination was described by Rayer as complicating pemphigus.

Chiro-pompholyx.—This affection also was described by Mr Hutchinson. It is chiefly confined to the hands and feet, is symmetrical, affects the nails, is recurrent, and the bullæ are small without dermatitis around them. Dr Robinson, of New York, Dr Liveing, and other writers have described similar cases. It affects the palm and sides of the fingers, as well as the dorsum of the hand. Before rupture the small bullæ, or large vesicles under the thick skin of the fingers, are described as like sago grains. This disease is probably distinct from the affection of the sweat-glands, described by the late Dr Fox as dysidrosis (cf. *infra*, p. 858). It more nearly resembles erythema bullosum.

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Hydroa.—The group of eruptions named hydroa by Bazin is not a natural one, either clinically or pathologically. Of the three species described by him, the first, or "vesicular hydroa," would clearly seem by its localisation on the back of the hands and wrists, and on the front of the knees, as well as by its acute but sometimes recurrent course, to be erythema. Other cases are identical with the curious affection long known as herpes iris, which is itself a form of erythema. "Bullous hydroa" probably includes *pemphigus pruriginosus*, or herpes gestationis (*infra*, p. 829), and erythema multiforme with blebs. Some cases, again, which have been described as hydroa, have turned out to be iodide rashes.*

Frequency of pemphigus generally.—Hebra, writing of twenty years' experience in the General Hospital of Vienna, as well as in his large private practice, could reckon only about 200 cases. He estimated that, excluding infants, one case of pemphigus occurred in 10,000 cases of illness generally. We must remember that the hospital statistics apply to all medical (as well as surgical) diseases, whereas his own practice was exclusively dermatological. He found in thirty years' statistics at the General Hospital that there were ten cases of pemphigus in men for rather more than three in women, excluding pemphigus foliaceus. In a report to the American Dermatological Association in 1881 only twenty cases occur in 11,000; Erasmus Wilson reported only nineteen cases among 10,000 private patients; and Kaposi, in 1890, had met with only 210 cases among more than 40,000 cases.

Treatment.—In Joseph Frank's work on diseases of the skin he stated that the best treatment of pemphigus is to leave it alone. Hebra proved the uselessness of diuretics and purgatives, tonics and quinine, mineral acids and Carlsbad waters. Formerly English physicians recommended venesection or leeches with antiphlogistic regimen, but with a caution to pursue the plan guardedly, which probably meant not to pursue it at all. They also recommended acids and bark. In the first edition of Wilson's work (p. 142) he writes: "When there is reason to believe that the eruption is an effort on the part of nature to determine to the surface a morbid disposition, I should strongly recommend the employment of mustard baths to the entire surface of the skin, or a stimulating liniment of some kind, such as that of croton oil, in the proportion of a drachm to an ounce of olive oil, to be well rubbed into the sound parts of the skin." Hardy says: "Le traitement général du pemphigus est encore à trouver." German authorities speak doubtfully of the prognosis in this disease, and depend chiefly upon local applications for its treatment. The late Dr Tilbury Fox recommended chlorate of potash, good food, and above all quinine, which he preferred to arsenic.

At the present time, however, most English physicians are agreed that *arsenic* is as much a specific remedy for pemphigus as for psoriasis. No doubt it occasionally fails, even in ordinary cases; but this can also be said of mercury in syphilis, and no one pretends that arsenic will cure gangrenous

* On the subject of Hydroa, see an elaborate paper by the late Dr T. Fox in the 'Philadelphia Archives of Dermatology' for 1880, p. 16, and one by Dr Crocker in the 'Lancet' for May 22nd, 1886.

The meaning of the word Hydroa is uncertain. Dr Crocker gives its derivation from ὕδωρ; but surely it is only a mistake for Hidroa (ἵδρωα), the regular term in Greek medicine for what the Latins called *sudamina*.

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pemphigus, or pemphigus foliaceus, or bad cases of extensive pemphigus in aged patients; but in nine tenths of the cases of pemphigus occurring in children and young adults the statement holds good that arsenic may be esteemed a sure remedy, even in severe cases. The late Dr Fagge, as well as Dr Habershon, Dr Hillier, Mr Hutchinson, Dr Gee, and many others, supports the same opinion. The drug should be administered on the same principles and with the same determination as recommended in psoriasis.*

Occasionally, however, we meet with cases in which, after perseverance with varied doses and varied forms of administration, we are obliged to abandon arsenic, not because of its disagreeing (that can always be met by diminishing the dose), but because the disease is unchecked. The best remedy then is tincture of steel. Sometimes quinine, guaiacum, or cod-liver oil succeeds when other drugs have failed.

In true gangrenous pemphigus of children, excluding syphilis, brandy and strong broths, or raw meat, with chlorate of potash internally, is the best treatment, and commonly proves successful. But even in infants one-minim drops of Fowler's solution should be administered.

In bad cases, especially in aged patients, attended with restlessness and distress, *opium* is a most valuable remedy, but unfortunately its use is forbidden or much circumscribed by the not infrequent presence of albuminuria. When this is absent it has a most valuable effect. In *P. vegetans* and "hand and mouth" cases opium is particularly indicated.

So far as is known, neither arsenic nor any other drug is of service in cases of pemphigus foliaceus.

Locally, no applications are very useful; whatever is most soothing is best, either zinc ointment, or oxide of zinc in powder, or what is often more pleasant and effectual, oxide of zinc with finely powdered chalk or gypsum suspended in water, and applied with a large soft brush. In very extensive and severe cases continuous baths have proved useful.

* See 'Med. Times,' Feb., 1854. Also the fourth of Mr Hutchinson's 'Lectures on Clinical Surgery:' "Can Arsenic cure Pemphigus?" and his recent lecture on the uses of arsenic ('B. M. J.,' 1891, vol. i, p. 1213).

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ERYTHEMA

AND ITS ALLIES

"Τὸν ὑπὸ βλεφάροις
φοίνικ', ἐρύθημα προσώπου."—EURIPIDES.

Definition of the group—Its characters—the anatomical lesions—course—locality—symptoms—ætiology—Symptomatic and traumatic erythema—Varieties: (1) *Erythema multiforme*—(2) *E. bullosum—Herpes—E. iris—Herpes gestationis—Dermatitis herpetiformis—Hydroa*—(3) *E. nodosum*—(4) *Urticaria—Urticaria pigmentosa—Treatment of erythematous affections. Erythematous rashes produced by drugs—The rash of copaiba—Bromides and iodides—Belladonna—Opium—Quinine—Salicylates—Arsenic—Mercury.*

WE have hitherto considered diseases of the skin which, though differing from one another in many particulars, are all examples of superficial dermatitis; they never leave scars, are chronic in course, apt to return, and are accompanied with more or less decided irritation.

We now pass to disorders which also form a natural group, though the line is perhaps more difficult to draw. They also are *superficial inflammations* of the skin, and therefore leave no scars, but they are *acute or subacute* in their course. Moreover, their lesions are usually slight and evanescent; and although occasionally a bleb may be produced, yet this is quite exceptional, and none of them is attended with pustules or crusts. Perhaps their most conspicuous anatomical character is the presence of inflammatory *œdema*, and this is often accompanied with slight *hæmorrhage*. The sensations accompanying them are usually *smarting* rather than itching. They have no relation to cutaneous irritants, but may often be traced to gastric or other internal causes.

Pemphigus forms a natural link between the true tetters—eczema, lichen, and psoriasis, on the one hand, and the erythematous affections on the other. Indeed, while Hardy classes it decidedly with the dartres, Auspitz and Hans v. Hebra place it in close relation to erythema multiforme.

The previous group of diseases, eczema and lichen, scabies and psoriasis, are usually chronic, and even when their onset is acute they run an indefinite course afterwards. They are more or less closely related to local irritation. The present group has an acute or subacute, self-limited course, often recurrent but seldom or never chronic.

ERYTHEMA is the name which has been given to some affections belonging to this group, and it may be conveniently extended to the whole.*

Willan classed erythema with roseola, urticaria, scarlatina, rubeola, and purpura under the title "exanthemata," the general character of the order

* This word, denoting "redness" of the skin, is applied in classical Greek, either as ἐρύθημα προσώπου or alone, to a blush, and by Thucydides to the redness of the eyes seen in those suffering from the plague at Athens. It was also used by medical writers as almost, if not quite, synonymous with ἐρυσίπελας.

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being hyperæmia of the skin without further lesion. Subsequent writers have called a mere hyperæmia "rose-rash" or roseola; while the word erythema, or, as Hebra calls it, *erythema exudativum*, has been confined to a rose-rash with palpable inflammatory exudation, diffuse or forming pimples. Hebra included also the so-called tubercular and nodose species of Willan's genus erythema, and invented a convenient term, *erythema multiforme*.

But in truth we have no need of a special title for mere hyperæmia, that is, dilatation of the blood-vessels without inflammatory exudation, such as follows division of a vaso-motor trunk in an animal. A transitory blush is always a physiological phenomenon. Clinically, persistent, "active," or arterial hyperæmia is found to be due to inflammation. Even the erythematous eruption of scarlatina, of measles, or of enteric fever can be proved by its course and sequelæ to be in each case true dermatitis. Bateman himself remarked that the efflorescence to which Willan appropriated the title of roseola is of little importance practically, and quotes the dictum of Fuller in his 'Exanthematologia' that it is "rather a ludicrous spectacle than an ill symptom."

We must, however, recognise two meanings of the word "erythema," just as we are obliged to recognise two of the word "eczema." We saw that eczema is, as Hebra proved, a common superficial dermatitis, which has reached the stage of visible and usually serous exudation; but we saw also that the most important peculiarity of the disease eczema is that it is not traumatic, not called forth by ordinary irritants, and not limited by their action. An artificial or traumatic eczema is therefore for practical purposes better refused the name. Accordingly we added to the definition of eczema as a disease the character of being *idiopathic*, with its own peculiar distribution and course.

In the same way "erythema" may be defined, and has been used by Hebra and other authorities to denote the slightest form of dermatitis in which the classical signs of redness, heat, and pain are accompanied by little or no perceptible swelling. The irritation of a mustard plaster, for instance, will in most persons produce such a typical "erythema;" the scorching of the sun does the same; and if the skin be more than usually delicate, and the mustard or sun more than usually strong, what was an "erythema" becomes an "eczema." It would be better if the term "superficial traumatic dermatitis" were used for both stages of the inflammation; or we might speak of the earlier as an erythematous and of the latter as an eczematous or weeping dermatitis.

But apart from this use of "erythematous" as indicative of a slight degree of inflammation with hyperæmia, we are only following Hebra's initiative by taking the best marked forms of disease to which the name Erythema is attached (*e. g. E. nodosum*), and grouping with them under the same title others which have the same pathology and clinical features, so as to form a natural group.

Just as we define the disease eczema by its clinical and pathological features apart from its mere anatomy, so we can define this erythematous group of affections. They are distinct from the erythematous stage of common dermatitis, and though often strictly "erythematous" in the symptomatic sense of the word, they sometimes exhibit other lesions, to which Hebra's adjective "multiform" applies.

1. The characteristic *anatomical lesion* is a rose-rash, resembling the first degree of traumatic dermatitis, that is to say *injection* of the surface;

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sometimes with obvious general *œdema*, sometimes with circumscribed œdema, forming *wheals*, and sometimes with *papules*—which are distinguised from those of eczema by not developing into vesicles, from those of lichen by their bright colour and transitory duration, from those of impetigo and prurigo by never becoming pustular, and from those of psoriasis by never becoming scaly. In certain rare forms of erythema separate *bullæ* are formed which may simulate those of pemphigus. The rash is usually followed by a slight branny *desquamation*. It will be seen that, after all, the multiformity of the lesions of erythema is less than that of the lesions of eczema.

2. Whatever the nature of the lesion, the *exudation* is of a watery rather than a corpuscular nature, so that œdema, diffused or circumscribed, is its characteristic, in contradistinction to the sero-purulent or purulent vesicles and pustules of eczema and scabies. Moreover, along with the hyperæmia and œdema, there is very apt to be a certain amount of escape of blood-corpuscles, an event which never occurs in eczema except as the result of direct injury, as by scratching. The result of this hæmorrhage is sometimes so marked as to give the title "purpura" to the eruption. Willan and Bateman rightly included *purpura urticans* with erythema among the exanthemata, although other kinds of purpura are altogether distinct from any form of dermatitis, and are only parts of a general hæmorrhagic condition. The result of the hæmorrhage is to leave bruise-like pigmentation behind, so that this when present is very characteristic of true erythema.

In these characters, as in some others, and especially in the fact of the occasional occurrence of bullæ, the erythematous group bears a closer relation to pemphigus than to any other of the chronic forms of dermatitis belonging to the so-called dartrous group, but if one attempts to include pemphigus as an erythema its course and treatment forbid the conjunction. Dr Hans von Hebra, however, whose classification differs widely from that of his father, unites under the title "Angioneurotic affections of the skin," the erythematous rashes of infectious diseases, the rashes produced by drugs and poisons, and, thirdly, the essential erythemata, together with pemphigus and acne rosacea.

3. The *course* of erythema is subacute, that is to say it begins quickly, sometimes with slight febrile symptoms, and does not last indefinitely. Even when its course is comparatively chronic it will be found that the protracted disease is really made up of a series of outbreaks, which may sometimes run into one another, but always preserve a recurrent or intermittent character. No erythematous disease ever acquires the chronic, stable, and inveterate stamp of eczema, lichen planus, psoriasis, or pityriasis rubra.

Erythema is, however, often recurrent, and, when frequently so, may produce chronic induration and hypertrophy. Of this, Gutta rosea is a capital instance (*infra*, p. 878).

4. The *locality* of erythema is much less definitely marked than that of psoriasis or of eczema. On the whole it is symmetrical, sometimes accurately and exclusively so, but there are frequent exceptions to the rule.

The favourite localities are, first, the extensor surface of the forearms and legs, especially the back of the hand, wrist, and ulnar side of the forearm, the dorsum of the foot, and tibial side of the shin;* secondly, the face,

* Although the true homology of the tibia is undoubtedly with the radius and not with the ulna, yet the tibial aspect of the shin, from its having no underlying muscles, agrees pathologically with the skin covering the subcutaneous surface of the ulna, just as pathologically the patella answers to the olecranon, and the second metacarpo-phalangeal to the first metatarso-phalangeal joint.

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cheeks, and neck; thirdly, the chest and abdomen. The back of the trunk, the buttocks, thighs, and upper arms are much less frequently affected; while the scalp, the flexures of the joints, the palms, and the soles are scarcely ever attacked by any form of true erythema.

5. As a rule, smarting and tingling are the *symptoms* which accompany erythematous eruptions, while severe pain and itching are rare. Local tenderness is more marked than in eczema. Sometimes, and especially when wheals are present, the irritation is considerable, though never comparable to that of chronic eczema, scabies, or prurigo.

6. The erythematous rashes do not spread. They appear simultaneously at different spots, and fresh patches appear which may occasionally unite, but we never see the affected part of skin gradually enlarge its borders—the characteristic course of eczema and psoriasis.

7. Of the *ætiology* of erythema we in most cases know nothing. The lesion can, as above explained, be produced by moderate irritation, the diffused forms by heat or friction, those with wheals by a lash, or by the poison of the stinging-nettle, the hairs of certain caterpillars, and the thread-cells of certain anthozoa. But in the non-traumatic, idiopathic, or "true" cases of erythema, the eruption can, in striking contrast to those of eczema and psoriasis, be in most cases traced to some *internal* disorder. In other words, erythema is usually *symptomatic*. The most striking instance of this is the erythematous rash produced by copaiba and by certain articles of food. Many other cases are dependent upon *dyspepsia*; others, again, upon *rheumatic fever*. This is particularly true of E. nodosum, urticaria, and the hæmorrhagic form of erythema known as *Peliosis rheumatica*.

Moreover, one may fairly adduce in this connection the fact that the symptomatic early rash of syphilis, the exanthema of scarlatina and of measles, and the occasional prodromic roseola of smallpox, enteric fever, and cholera, all belong to the erythematous type.

8. Erythema occurs most commonly in children and young adults; it is comparatively rare after forty. Among persons past their prime it is less uncommon in women than in men. In these, as in so many other points, we observe a marked contrast to eczema and psoriasis, and a resemblance to pemphigus and also to rheumatism.

Traumatic dermatitis of slight degree is still often called erythema. Thus *intertrigo*, mentioned above under eczema (p. 767), was classed under erythema by Willan, Hebra, and Neumann. Erythema leve is another example.

Roseola furfuracea (roseola maculata et circinata) is a slight inflammation approaching some forms of erythema, which has been described above as Pityriasis rosea (p. 811).

The chronic congestive disorders, of which chilblains are the type, are chronic in course and venous or cyanotic in aspect and pathology. They have no claim to be ranked under erythema, as above defined, and will find their place in a subsequent chapter, along with Gutta rosea.

The erythemata which are symptomatic of measles, scarlatina, enterica, rubeola, as well as choleraic roseola, and that which sometimes precedes the characteristic rash of smallpox,—all these have been described in the first volume of this work.

The erythematous rashes which follow the administration of drugs will for convenience be considered together at the end of this chapter.

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There remains a group of skin affections which agree in the general characters of anatomy, course, and natural history described above, which are not traumatic, nor secondary to diseases of the vascular system, and which are not symptomatic, either of febrile diseases or of what Continental writers call "intoxication" with drugs or poisons. These we may style idiopathic, primary, or essential erythema. Their common characters have been already sufficiently expounded. It remains to point out the principal varieties which they present.

1. *Erythema multiforme.*—Simple or ordinary erythema, erythema papulatum, erythema exudativum.

The commonest kind of erythema is that which consists in general hyperæmia with œdema of the skin, a diffuse dermatitis which may either spread over a large surface with indefinite edges, or, as is more frequently and characteristically the case, occur in patches with defined edge.

On careful examination, small papules may be often distinguished scarcely rising above the level of the skin, as in the eruption of measles; sometimes these are well marked enough to deserve the title *erythema papulatum*, but this is comparatively rare, and most lesions of the skin which receive this name are probably either traumatic dermatitis or an early stage of papular eczema. Large, firm, and persistent papules such as occur in prurigo are never seen in true erythema.

The inflamed patches have usually a very short duration; they may disappear in a few hours (*Erythema fugax* of Willan) and be succeeded by others, but if they persist for a day or two they may form rings which have been specially described as *Erythema annulatum* (*E. circinatum* of Willan), or *Roseola annulata.* When closely set, several of these rings unite, and a sinuous reddish band is produced which has been named *Erythema marginatum* or *E. gyratum.* Finally, the redness fades, the œdema subsides, and may leave no trace behind. If there is desquamation it is very slight and furfuraceous; more frequently a slight amount of pigment marks the seat of the eruption.

The favourite localities are the back of the wrists and forearms, and the legs; less often the face and neck; sometimes the trunk is affected, but very rarely the thick parts of the skin or those covered by hair.

Erythema annulatum is often observed in association with rheumatic fever, either in the course of an attack or preceding it. It is more common in children and young adults than in older persons.

The inflammation of the skin called *Erythema leve* is a common dermatitis which is apt to appear upon the tense skin of dropsical parts, and may go on to deep dermatitis and sloughing. It is not uncommon as the result of acupuncture or of tapping, and is allied to traumatic erysipelas.

2. *Vesicular and bullous erythema.*—The exudation of erythema, instead of being a somewhat deep diffused œdema, sometimes appears in superficial collections of serum. These when small are called vesicular erythema or "herpes;" when large, "erythema bullosum."

*Herpes,** the common Latin term for an eruption of the trunk, in contra-

* The word ἕρπης is derived from ἕρπειν. "Herpes dicitur eo quod videtur ἕρπειν, quod est serpere per summam cutem, modo hanc ejus partem modo proximam occupans." From the same creeping progress the disease was, according to Bateman, called *formica* by the Arabs.

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distinction to porrigo or an eruption of the head, was limited by Willan to vesicular eruptions which he distinguished from the vesicles of smallpox and chicken-pox, from sudamina, and from the inflammatory vesicles of eczema. His species of herpes were as follows:

Herpes zoster, or zona, an eruption erythematous, it is true, in its anatomy and course, but which is so demonstrably connected with nervous disorder that it is rightly separated from all other forms of dermatitis, and will be described in a future chapter among cutaneous disorders of nervous origin.

Herpes circinatus, which we shall afterwards describe as the form assumed by ringworm when it affects the body, is a parasitic disorder, and is now classed with *Tineæ*.

Willan and Bateman's remaining species are *Herpes phlyctænodes*, of uncertain seat, called *H. labialis* or *H. preputialis* when affecting the lips or the foreskin respectively, and *H. iris* when found on the back of the hands or the instep.

These from their course and natural history may be well included in the general group of erythemata. This was, indeed, to some extent admitted by Hebra, and even by Rayer before him.

Herpes labialis or *facialis* consists of a little group of vesicles upon a red patch of skin which appears almost suddenly, most often upon the upper lip, frequently on the alæ of the nose, sometimes on the cheeks or chin. The writer had recently an example of symptomatic herpes or vesicular erythema, which covered one ear of a boy ill with lobar pneumonia, and ran a typically acute course. Or the vesicles may appear on the buccal mucous membrane. In a day or two the clear, pearly contents become somewhat turbid and puriform, and dry up into a thin brownish crust which speedily falls off and leaves no trace behind. The vesicles entirely differ from those of eczema or ordinary traumatic dermatitis by their large size, by their not running one into another so as to form a weeping surface, by their acute course, and by the sharply limited edge of the patch. They also are unattended with itching or pain, and never consist of pure pus like the eruption of impetigo or scabies. Moreover, they are always symptomatic of some internal disorder, most characteristically perhaps of acute lobar pneumonia. Many persons are liable to such patches of herpes, either on the lips, or less frequently on other parts of the face, when they are attacked by acute catarrh. Bronchitis, catarrhal pneumonia, whooping-cough, asthma, are rarely accompanied by herpes. Sometimes the eruption appears to follow a rigor even when this symptom does not prove the precursor of pneumonia or catarrh.*

This curious eruption has clearly little or no connection with eczema and its allies; nor can we link it with zona, for it frequently recurs, it is not unilateral, it does not follow the course of a nerve, and is unattended with pain. Its superficial character, sudden onset, and rapid course agree with the erythematous group as here defined, and the fact that it is symptomatic of internal disturbance and usually of irritation of a mucous tract completes the analogy.

Herpes preputialis, when no longer left as Willan placed it, and as Hebra was content to leave it, among vesicular inflammations, is difficult to classify. Hardy is even driven to the untenable assertion that it is nothing but local vesicular eczema. The rapidity of its onset and course, the superficial

* See an interesting autobiographical account of a case of the kind by Mr C. J. Symonds in the 'Clinical Transactions' for 1884, p. 60.

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lesion, the patches, the absence of notable irritation or pain, all point to a pathological connection with true erythema, while the anatomical lesion and its occurrence at the orifice of a mucous tract bring it into relationship with herpes labialis. The chief difference is that, occurring as it usually does on the inner side of the prepuce or glans, the vesicles are broken almost as soon as they form, and very superficial ulcers take the place of scabs. The condition is exactly like that of a vesicular eruption on the tongue. Like herpes labialis, it often recurs in the same patient; like it also it is often symptomatic of inflammation or stricture of the urethra, although it does not seem to be produced by cystitis, and certainly does not follow inflammation of the kidney as labial herpes does inflammation of the lung. The chief practical importance of preputial herpes is its diagnosis from a soft chancre.

Herpes iris is a rare and remarkable form of eruption, well described by Willan, which is unmistakably erythematous in its nature, and is better named *erythema bullosum*, or *erythema iris*, or *iris*. It occurs sometimes as a single, sometimes as two or more rose-coloured patches with all the characters of erythema, almost always upon the back of the hand, the wrist, or extensor aspect of the forearm, more rarely on the corresponding part of the foot, the instep, and ankle, and exceptionally on the face. It rapidly becomes annular, but before the ring is faded the patch of erythema reappears in the middle, and may thus be surrounded with one or (from a repetition of the process) by two or even three concentric rings. The surrounding ring may exhibit similar vesicles or they may be more or less abortive, so that one might often question whether, if we adopt the anatomical nomenclature, we should describe the lesion as erythematous or bullous or vesicular. In its most striking form, with a single large tense bleb like one of pemphigus, surrounded by vesicular circles, the whole patch as large as a crown-piece, it is one of the most remarkable of eruptions. The inflammation is very superficial, produces little pain or irritation, and after forming thin scabs passes off after a few days, leaving more or less pigmentation, yet not a trace of scar behind. Partly the resulting pigment and partly the rosy red of the rings, the pearly grey of the vesicles, and the more or less yellowish contents of the older bullæ seem to have combined with the bow-like form to give the title iris. The course of this curious disease, its superficial character, and its locality, all make it unmistakably erythema, as also the fact that it occurs almost exclusively in young persons; but it appears to be symptomatic of nothing.

Iris is not, however, the only bullous form of erythema. Other cases of bullous erythema have been recorded by Dr Duffin ('Pathological Transactions,' 1875), by Dr Crocker and Dr Frederick Taylor ('Clinical Society's Transactions,' Feb. 25th, 1881), and the writer reported two typical cases in the 'Guy's Hospital Reports' for 1880 (3rd series, vol. xxv, p. 211).

*Erythema gestationis bullosum.**—There is a remarkable and rare affection which has been described as a species of pemphigus, of herpes, or of erythema, or has been included under the title "Hydroa." Its pathological alliance appears to be with the form of erythema which depends on ovarian irritation, but the existence of bullæ makes it liable to be confounded with pemphigus.

It occurs only in women during pregnancy. The bullæ, vesicles, and

* *Synonyms.*—Hydroa (in part)—Pemphigus uterinus, hystericus v. pruriginosus—Herpes gestationis—Impetigo herpetiformis—Dermatitis herpetiformis.

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vesiculo-pustules appear in abundant crops over the trunk, and often on the face and limbs also. Pruritus is marked. There is more or less constitutional disturbance, and sometimes the temperature rises high. The clinical aspect is therefore serious, and occasionally alarming. But the result appears to be always favourable. The disease is cured by delivery.

We have had one instance—and others are on record—in which this remarkable form of pemphigus or bullous erythema appeared again and again in successive pregnancies.

Cases of "herpes gestationis" have been carefully described since Chausit and Hardy, by Dr Liveing, Dr Bulkley, and other observers in this country, on the Continent and in America. The general features are very uniform, and there is no doubt of the reality and distinctness of the disease; but its true pathology and the means of prevention or treatment are still obscure.

Dermatitis herpetiformis.—Under this name Dr Duhring, of Philadelphia, described, in 1884, a polymorphous inflammation occupying the greater part of the trunk and limbs and causing much irritation. The first eruption is a papular erythema, but vesicles and bullæ soon appear upon the inflamed patches, and smarting succeeds irritation. Successive crops of eruptions follow, with pigmentation, until weeks or months have passed; but the ultimate tendency is to recovery.

This description coincides with that of cases to which Bazin applied the title of *Hydroa* (p. 820). He distinguished a herpetic form (*Hydroa vesiculeux*), a pustular (*H. vacciniforme*), and a bullous (*H. bulleux*). The first may be referred to Willan's Herpes phlyctænodes or Hebra's Erythema multiforme, the second, perhaps, to Hebra's Impetigo herpetiformis,* and the third to Willan's Herpes iris. The group would include the *Herpes gestationis* above described and Erythema bullosum, as well as Pemphigus pruriginosus.

There appears to be no advantage in using the vague term Hydroa or that of herpetiform dermatitis for this group of eruptions, which may better be included among the herpetic, vesicular, or bullous forms of Erythema. The distribution is more general than than in typical cases of Erythema iris or E. multiforme, but it agrees in avoiding the scalp and face, the palms and the soles, the shoulders and back; the course is more recurrent, but not truly chronic; there is much more of itching and less of smarting, but the broad clinical features of the affections so variously named separate them from all but the erythematous group of diseases.

When vesicles and bullæ appear in the course of an erythematous rash, we must first make sure that they are not due to local irritants, whether accidentally or designedly applied; next we must separate them from the bullous and pustular eruptions produced by iodide of potassium, from the bullous form of syphilodermia, from symptomatic Herpes, and from that which follows the course of cutaneous nerves and will be afterwards described as Zona; and, lastly, we must diagnose them as well as may be from the bullæ of typical Pemphigus, although in many cases the line is here difficult to draw.

* This affection is described by Kaposi in a paper published in 1887 in the 'Vierteljahresschrift f. Derm. u. Syph.' Duhring would include it under his Dermatitis herpetiformis, but no case has yet been described in England, and at present there seems great doubt as to its nature. Possibly it is a pyæmic eruption, for its course appears to be malignant.

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3. *Erythema nodosum.*—This curious affection was well described by Willan, and subsequent authors have added little to his account. It occurs "in large oval patches, the long diameter of which is parallel with the tibia, slowly rise into hard and painful protuberances, and as regularly soften and subside in the course of nine or ten days, the red colour turning bluish on the eighth or ninth day, as if the leg had been bruised." In this form of erythema the anatomical lesion is especially characterised by œdema; the spots do not itch, but are somewhat painful and very tender, more so than in any other of the erythematous group. There is almost always not only deep venous congestion of the typical erythematous rose-tint, exaggerated by its position on the legs, but there is almost always a slight indication of actual hæmorrhage. Probably the pigmentation, which like other forms of erythema it is apt to leave behind, is much deepened by chemical transformations of the effused hæmoglobin such as we see in a bruise.

The locality of erythema nodosum is, as Willan says, most frequently over the tibia, but it is not confined to this part, for it may be seen on the ankle or the calf, and it is not uncommon over the corresponding surface of the ulna. It is usually symmetrical, and may affect the whole extensor surface of both forearms and both legs. It is very seldom seen elsewhere.

It has a slower course than most kinds of erythema, but like them is prone to recur. Willan and Bateman, and also Green in his 'Practical Compendium,' state that erythema nodosum only affects women; but Plumbe in 1824 ('Practical Treatise on Diseases of the Skin') notes its occurrence in children, and it is not unfrequently seen in boys under or about the age of puberty, who are also liable to hysteria, chorea, and other female disorders.

It occurs very frequently in those who have suffered from *rheumatic fever*. Dr Stephen Mackenzie brought before the Clinical Society in April, 1886 (vol. xix, p. 215), more than 100 cases of erythema nodosum collected from the four largest hospitals in London. Ninety patients were females, and 18 males. Only 25 were over thirty years of age, 30 were between twenty and thirty, 39 between ten and twenty, and 14 were children under ten. In 17 cases there was also past or present rheumatism (acute in 13, subacute in 4), beside about as many more in which the existence of true rheumatism was asserted or probable. There was a cardiac murmur in 13 cases, in only two of which there was history of rheumatic affection of the joints.*

The course, the lesion, the œdema, the hæmorrhage, the locality, the subjects of this affection, are all typically erythematous.

4. *Urticaria.*†—Willan rightly placed urticaria in close relation to erythema. Almost all subsequent writers have followed this indication; and if convenience did not forbid innovations, it might be called "erythema pomphosum," for the characteristic lesions are *pomphi*—wheals, *i. e.* raised flat white patches, sometimes surrounded by an erythematous blush. Their histology is that of acute inflammatory œdema of the cutis, which fills the lymph-spaces and expels blood from the venules. The effusion takes place very rapidly, and may be called forth either by a mechanical or by a

* See Dr Thos. Barlow, 'Brit. Med. Journ.,' Sept. 15th, 1883, p. 511. Dr Cæsar Boeck has also published a monograph on this point.

† *Synonyms.*—Nettle-rash—Cnidosis: including lichen urticatus, purpura urticans, and much of strophulus.—*Fr.* Urticaire.—*Germ.* Nesselsucht.

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chemical irritant, as in the wheals produced by the nettle (*Urtica urens*), from which the disease receives its name. In persons liable to the affection it can be produced by the finger only drawn across the skin, so that it is possible to write characters in raised wheals. This last has been defined as "factitious" urticaria. The anatomical lesion has therefore its counterpart in the traumatic wheals produced in any skin by the sting of the nettle or the stroke of a whip, and produced by much slighter irritation in susceptible subjects. The relation of such traumatic urticaria to the idiopathic disease precisely corresponds with that which was expounded at some length between common superficial dermatitis, from the sun or other irritant, and idiopathic eczema (p. 763), between prurigo senilis a pediculis, and idiopathic prurigo of Hebra (p. 801), between erythema congestivum et bullosum, and chilblains (*infra*, p. 880).

Beside the well-marked oval or linear wheals of ordinary urticaria, we often see the lesion in the form of small round patches or as large white plateaux formed by the coalescence of several smaller ones ("giant urticaria," *U. tuberosa*). Both these forms are frequently produced by nettles. We may also include as essentially of the same nature the large, flat, white papules, which are obviously distinct from those of ordinary eczema, and which have been described as *strophulus albidus*, and also not unfrequently under the name of *infantile prurigo* and *lichen urticatus*. These papules are distinguished by rising rapidly, and by following, not causing, pruritus; for they are the result and not the occasion of the patient's scratching. They are most often seen in infants, but may be observed along with more obvious wheals in ordinary cases of adult urticaria. In exceptional cases it is said that the wheals last, so as to produce a chronic condition (*U. perstans*). Sometimes, but also as an exception, bullæ are described as mingling with the wheals—a fresh sign of affinity between urticaria and erythema.

The *distribution* of nettle-rash is less definite than that of other forms of erythema, and, indeed, of most other cutaneous affections. We do not observe any predilection for the erythematous regions, the extensor surface of the forearms and legs. It is quite as common on the back and trunk generally as on the limbs; the only parts it avoids are the scalp, face, arms, and soles. It is not symmetrical. The mucous membrane of the mouth is occasionally affected.

Urticaria is abrupt in origin and sometimes acute in *course*, but often persistent and obstinate in successive attacks.

Of all forms of erythema, urticaria is the most irritable, the severity of the itching being comparable to that of eczema, scabies, or prurigo. There is no pain or smarting, and no subjective symptoms except from the restlessness and sleeplessness which it occasions, especially in children. It is most frequent in them or in young adults, but is not confined to any age. According to Dr Liveing it sometimes alternates with neuralgia of the same parts.

The *ætiology* of urticaria is uncertain. As above explained, it is often purely secondary to some local irritant, as pediculi, or complicates a previously existing malady, as prurigo; and it is probably always aggravated by the patient's scratching. Its close alliance with erythema is shown not only by sometimes alternating with it, but also by its following precisely the same kind of gastric disturbance, both in the most marked forms which are the direct result of drugs or of poisons, and in the less evident cases

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associated with ordinary dyspepsia. When there is no internal irritant, and no gastric disturbance, urticaria in women can often be traced to ovarian disorders, breaking out at each menstrual period and subsiding when dysmenorrhœa is cured. Like erythema, again, it is, according to general experience, a not infrequent complication of rheumatic fever.

*Urticaria pigmentosa.** — A singular and rare form of skin disease belonging to the erythematous type, but as chronic as erythema nodosum, was first described by Mr Edward Nettleship, and again by Mr Morrant Baker and the late Dr Tilbury Fox, in the 'Clinical Society's Transactions,' vols. viii and x. A case in a child of two years old was shortly described in the twenty-fifth volume of the 'Guy's Hospital Reports,' 3rd series, pp. 212, 213.

It has received several names, among others the uncouth and misleading term Xanthelasmoidea; but Dr Sangster's proposed title, urticaria pigmentosa, is now generally accepted.

It is an erythematous eruption with occasional wheals and considerable yellowish pigmentation, lasting for an indefinite period, though its chronic course is probably always made up of more or less distinct subacute attacks. It affects the back and trunk generally, rather than the limbs, and the face very little. The characteristic buff colour always follows the rose-rash.

An excellent account of this affection is given by Dr Colcott Fox in the 'Med.-Chir. Trans.' for 1883, where nineteen recorded cases are tabulated, to which Dr Crocker has since added one ('Clin. Trans.,' vol. xviii, p. 12). See also Dr Cavafy's article in 'Heath's Dictionary of Surgery.'

Dr Paul Raymond has written a valuable monograph on this curious malady, in which he has collected sixteen cases observed in England, two in America, eight in Germany, and four in France. All these cases occurred in children under two years old, often within a fortnight after birth. The majority of the infants were boys.

Microscopical examination of the affected skin by Dr C. Fox has shown that the lesion is truly a wheal, the tissue of the corium being opened out by œdema. Pick had found minute hæmorrhages in another case.

The affection recurs again and again, but as the child grows older gradually ceases, and the pigmentation still more gradually fades.

In an exceptional case recorded by Lewinski it was still present in a lad of eighteen. In one case of erythema under the writer's care in a woman of thirty-two, a condition which might be called *E. perstans* had been present for four years on the trunk, and was followed by decided pigmentation.

Statistics.—In 100 consecutive cases of the writer's, there were 38 of *Erythema multiforme* (marginate, circinate, annular, gyrate, papular, and roseolar), 17 of *E. bullosum*, including one of Herpes gestationis, 13 of *E. nodosum*, 18 of Urticaria, and 14 of multiform erythema and urticaria in the same subject. Several cases of erythema and of urticaria occurred during, or after, rheumatic fever, and four cases of urticaria came immediately after the patient had eaten shell-fish.

There was in each group (except Urticaria, where the numbers were almost equal) a preponderance of female patients, the total numbers being 57 to 39

* *Synonyms.*—Urticaria perstans pigmentosa—Xanthelasmoidea—Erythema tuberculatum—Permanent erythema.

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beside 4 infants. In addition to these 4 children under two years old there were in all 33 patients between four and sixteen, 48 between seventeen and thirty five, and 14 above thirty-five.

Treatment of erythematous affections.—The various erythematous diseases which we have grouped together in this chapter are seen in their true relationship from a practical point of view. They are none of them contagious, they are none of them attended with serious consequences, they are mostly indicative of some other primary disorder, and they are rather to be palliated by local applications or indirectly cured by treating their internal cause when discovered, than met by a specific plan of treatment. In particular it may be said that they are either unaffected or aggravated by arsenic, and this is one of the most important points which separate them from pemphigus.

In many kinds of erythema, especially symptomatic herpes and iris, no treatment is needful.

The local treatment of the other erythemata consists in the astringent and sedative applications described at p. 774 *seq.*; although the surface is dry, it is found by experience that lotions in most cases answer better than ointments. Goulard's wash, evaporating lotions of spirit and water, or eau de Cologne, hydrocyanic acid well diluted, or solution of borax are the best local applications. When urticaria is severe and these means fail, chloral hydrate may be used locally in solution or chloroform and ung. cetacei (♏x—xx ad ℥j). Warm baths should be avoided, as also excessive heat and perspiration; tepid water is better than either cold or hot. The patient should be urged to stoical abstinence from scratching; tepid bathing or continued, steady pressure will be found to relieve the intolerable irritation of urticaria without aggravating it afterwards as scratching always does.

For the painful swellings of erythema nodosum, strong lead lotion gives most relief, or lead and opium. Collodion painted over and allowed to dry is often useful, or alum, tannic acid, or other astringent remedies may be used with advantage, or the affected part of the leg may be painted with a strong solution of nitrate of silver. Ointment containing zinc or zinc and lead may be applied to the herpetic and bullous forms of erythema.

Internally, our first care should be to relieve the gastric disorder which often accompanies common congestive or papular erythema, most often by discovering certain articles of food to which it is due. Salt fish, pickles, preserved fruit in the form of jams and crystallised sweetmeats, pork, sour or otherwise inferior wine, malt liquor, stone fruit, and even strawberries—any one of these may in certain persons excite erythema or urticaria, and it is said that eggs and other articles of diet may occasionally act in the same way in certain persons. Those first on the list should be strictly forbidden. Of all kinds of food, lobsters and crabs, and by a curious coincidence mussels and other molluscs united with them under the title of "shell-fish" are the most frequent causes of erythema or urticaria, and they are the most severe in their effects. Oysters are harmless. Many drugs have a similar result, copaiba being probably the most effectual.

If the eruption continues after its supposed cause is removed, or if we are unable to discover any cause of disorder, such remedies as bicarbonate of soda with gentian or calumba or chiretta or a few drops of liquor potassæ in peppermint or cinnamon water should be prescribed. Where there is evidence of gastritis, bismuth is a most valuable remedy, given either in powder or

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thus: ℞ Bism. subnitr., sodæ bicarb., pulv. tragac. co: āā gr. x., aq. chloroform. sive menthæ pip. ℥j. M.; to which ten or twelve drops of solution of morphia may be added if the pain is severe. With flatulent disorder, thymol, creosote, or carbolic acid in the form of pills are often the most effectual mode of treatment. In the more atonic forms pepsine given before meals is found practically useful, notwithstanding our physiological doubts; and occasionally dilute mineral acids with nux vomica or bitter infusion will be more valuable than anything else. Gentle saline laxatives taken before breakfast in a large draught of warm water are almost always indicated; and, for women especially, a pill containing aloes or rhubarb, taken before a late dinner or on going to bed, is a useful adjunct. In many patients occasional doses of blue pill are of unmistakeable value.

In cases of erythema nodosum and in other forms of erythema which follow rheumatic fever, and occur in pale young women or lads, the preparations of steel are strongly indicated. When there is constipation a good formula is three or four grains of sulphate of iron, half a drachm of sulphate of magnesia, and five drops of dilute sulphuric acid, in peppermint water or calumba. When this is not the case the tincture of steel is a most valuable remedy. In some patients sulphate of iron with carbonate of potash and extract of Barbadoes aloes forms the most valuable Martial remedy. In whichever form iron is found to agree best it is important to increase the dose until a decided effect is obtained.

In the more severe forms of recurrent bullous Erythema, quinine in full doses is of great value, and opium may sometimes be combined with it. Dr Crocker has found arsenic efficacious in some of these cases; and Dr Duhring recommends arsenic in the recurrent forms of Dermatitis herpetiformis. Impetigo herpetiformis is said to be uninfluenced by treatment, and the cases reported from Vienna ended fatally.

Eruptions produced by Drugs.—Since the most frequent and characteristic effects of drugs upon the skin are erythematous eruptions, it will be convenient to consider this group of dermatoses here. Drugs and poisons act much in the same way upon the skin as do irritant or poisonous articles of food.

The most striking and frequent of these eruptions is perhaps that produced by *copaiba*. This has sometimes been confounded with an early syphilide. It usually takes the form of a papular erythema, often combined with urticaria and not unfrequently more or less hæmorrhagic. The occurrence of bullæ or vesicles is mentioned by trustworthy observers. In some cases there is no itching, which makes the diagnosis from syphilis the more difficult. The rash is generally distributed over the whole surface of the body, and does not spare even the face, as most other erythemata do. Occasionally it simulates purpura. Some writers have suggested that it is not the copaiba but the urethral inflammation for which copaiba is commonly given which produces the rash. There can, however, be no doubt of the existence of a true copaiba rash. It is not uncommon from the exhibition of the oleo-resin, but is rarely observed in persons who are taking the valuable diuretic, mistura copaibæ resinæ.

Cubebs is generally said to produce a similar eruption, but some of the reported cases appear to have been due to accidental mixture with copaiba. So, at least, Dr Bulkley believes.

Somewhat similar rashes have been observed in patients taking turpen-

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*line, cannabis indica,** and some other drugs, and have been described as purpura, urticaria, pemphigus, herpes, or erythema *a medicamentis.* It is possible that some at least of the eruptions ascribed to salicylic acid were really peliosis rheumatica.

Bromide of potassium comes, perhaps, next in frequency to copaiba as a rash-producing drug. The lesion here simulates very closely that which will be described in the next chapter as acne, but the diagnosis is generally clear from its not being confined to the very characteristic localities of true acne, or to the equally characteristic age which is specially liable to that disease. Occasionally the bromide eruption is more severe, and produces pustules and crusts. The late Dr Carrington reported a remarkable case of solid tumours in an infant, apparently the result of the exhibition of bromide ('Clin. Trans.,' vol. xviii, p. 28, with plate); and also Dr Lees ('Path. Trans.,' vol. xxiii, p. 247, with plate).

Less frequent, but much more varied, more severe, and more misleading is the eruption produced by *iodide of potassium.* This is, perhaps, most frequently a papular erythema, widely or irregularly distributed on the trunk, limbs, and face, free from itching, and usually unfelt by the patient. Sometimes, however, there is considerable erythematous dermatitis between the papules. A follicular inflammation undistinguishable from that described above as bromide-acne is a less frequent effect of the iodide salts. More often the rash which was at first papular becomes vesicular, bullous, or pustular. In these cases the inflammation is often very severe, and the constitutional disturbance considerable. They have been, there is no question, often confounded with herpes and so-called "hydroa," and, indeed, until one has seen several cases, it is difficult to believe that so severe a dermatitis can be due to a drug which in most cases has no effect whatever upon the skin. The eruption may simulate scabies or eczema, but the absence of definite localisation, of chronicity, of the secretion of eczema or of the cuniculi of scabies, should make the diagnosis not difficult. Along with the pustules there may arise what the older dermatologists would have called a tubercular disease of the skin, raised fleshy nodules simulating papillary growths, condylomata, mucous patches, and the later forms of syphilodermia. They may resemble rupia or lupus, or even malignant disease. Inasmuch as these severe effects are apt to follow the large doses of iodide of potassium given in the later stages of syphilis, the difficulty of discriminating them is naturally increased.

In a patient under the writer's care suffering from an ordinary pustular syphilide, some of the lesions on the face and the back of the hand became so swollen, hypertrophied, and covered with profuse granulations, that both cheeks were deformed, the eyes almost occluded, and one hand was covered with exuberant granulations, which, when seen alone, suggested to different observers lupus hypertrophicus or epithelioma. There was, however, no doubt of the nature of the case. The diagnosis was confirmed by the patient's recovery perfectly when the drug was discontinued. The chief point which guided one aright in this case was that, notwithstanding his frightful appearance, the patient was eating and sleeping well, so that it was with great difficulty he was persuaded to come into hospital.

Another form of iodide rash is punctiform, and resembles scarlatina rather than measles, the patches and rose tint of which are more nearly

* In a case reported by Dr J. N. Hyde in the 'New York Medical Record' for May 11th, 1878.

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simulated by the copaiba rash. This iodide eruption is often purpuric (Dr Duffey, 'Dublin Journal of Medical Science,' vol. lxix, April, 1880).

More often pustules may appear, and when deep and occupying a hair-sac, cause crops of boils. The presence of iodine and bromine has been actually demonstrated in the pustules by Adamkiewicz and Guttmann.* The iodide produces its effects on the skin much more rapidly than the bromide. In both cases there appears to be a true excretion of the drug through the sebaceous glands. For an account of the histology see a paper by Dr Thin ('Med.-Chir. Trans.,' vol. lxii, p. 189).

Weeping dermatitis, curious wart-like nodules, and other peculiar eruptions have been described as the result of bromide of potassium by Voisin and Veiel, quoted by Behrend ('Berlin. klin, Wochenschrift,' vols. xvi and xxii, pp. 626 and 714, 1879). Two cases of severe iodide eruption were figured by the late Dr Tilbury Fox ('Clinical Society's Transactions,' vol. xi, November 23rd, 1877).

Various measures have been adopted to prevent these unpleasant effects. The addition of carbonate of potash or aromatic spirits of ammonia is sometimes sufficient. Moderate doses of arsenic have been recommended, but they often fail in preventing the eruption. Changing the potash to the soda salt of iodine is sometimes followed by the disappearance of the rash; this is, perhaps, a coincidence. It is, at least, certain that persevering with the drug in even larger doses is often followed by the disappearance of the unpleasant effect it had produced.

Belladonna in full doses often causes a bright-red and almost universal erythematous rash. It may be recognised by its association with dilated pupils and a dry throat, together with the characteristic delirium if the dose has been large. In one case under Mr Hilton it was caused by the mere application of a large belladonna plaster in a woman who must, one supposes, have been more than commonly susceptible. Children, who bear as large doses of this drug as adults, are also liable to these symptoms of intoxication. A few years ago more than a dozen children were admitted in Guy's Hospital with symptoms of belladonna poisoning. They had broken into a drug warehouse on a Sunday and had eaten some of the contents. The rash in most cases was like that of scarlatina.

Similar rashes have been observed as the result of *hyoscyamus* or *stramonium*.

Opium and *morphia* sometimes produce considerable pruritus, and this leads to erythema or urticaria by the scratching which results.

Chloral-hydrate has occasionally been the cause of an erythematous rash ('Clin. Trans.,' xiii, p. 121), and in one instance the writer observed a severe general acute dermatitis follow the exhibition of *chloralamide* (*ibid*, 1890).†

Quinine.—There can be no doubt that quinine may produce a general acute erythema, which was first described by Skinner, Fleming, and other English authors, and has since been observed abroad. Its symptoms closely resemble scarlatina. It begins in the face, spreads rapidly over the whole trunk, and is accompanied by severe fever, the temperature sometimes reaching 103·5° Fahr. It is certainly a very rare effect of so popular a medicine, and its occurrence may be regarded as due to an idiosyncrasy. In one case of Köbner's the eruption followed the exhibition of quinine

* 'Virchow's Archiv,' 1878, vol. lxxiv; 'Charité Annalen,' vol. iii, p. 381, 1878.

† Mr Hutchinson has figured an erythematous rash of the hands due to chloral in his 'Archives of Surgery,' vol. i, pl. v.

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three times in the same patient. A still more severe local erythematous rash of the face, sometimes vesicular, has been observed as the result of quinine by Hebra, von Heusinger, and some other physicians. (See a case in a child reported by Dr H. Hagan, of Atlanta, in the 'New York Medical Journal,' 1891.)

Dr Morrow, who has collected sixty cases of quinine eruptions, found that in thirty-eight the rash was erythematous, in twelve it resembled urticaria, in two it was vesicular, and in five hæmorrhagic ('New York Medical Journal,' March, 1880). One case was reported by Dr Fagge ('Medical Times,' February 29th, 1868). See also Dr Farquharson's paper in the 'Brit. Med. Journ.,' February 15th and 22nd, 1879.

Antipyrin has occasionally produced an extensive erythematous rash.

Eruptions from *salicylic acid** have been reported. Since erythema and urticaria are common in the disease for which salicylic acid is usually given, and since adulterations with carbolic acid and consequent gastric disturbances are not unknown, the interpretation of these cases may admit of doubt. The internal use of carbolic acid itself, of tar, turpentine, and petroleum have all produced rashes usually erythematous, but the cases are comparatively rare. An excellent bibliography of the whole subject is given at the end of a paper by Dr Van Harlingen in the 'Archives of Dermatology,' Philadelphia, October, 1880.

Arsenic is said to produce in some persons an acute vesicular eruption which has been styled herpes, and in others urticaria. This occurrence is, however, very rare even when large doses of the drug are given. Zona† has sometimes appeared during a course of arsenic, too frequently perhaps to be considered a mere coincidence. But if when a patient is taking arsenic and zona breaks out, the mere facts that he has not been exposed to cold, and that the eruption is not epidemic, may be accepted as evidence that arsenic is its cause, all inquiries into ætiology become at once easy and useless. Arsenic has been observed to cause pigmentation of the skin.

Mercury was one of the first drugs to be regarded as the cause of a cutaneous rash. Early in the present century Alley‡ described what he called Hydrargyria, before the first description of a copaiba nettlerash by Montègre in 1814. Alley's cases were mostly vesicular and corresponded with what we should now call eczema, chiefly of the abdomen, thighs, and scrotum, but sometimes they assumed a more severe pustular form, and still more rarely that of bullæ with severe pain and lymphatic inflammation, combined with angina. We may doubt whether local inunction of the drug or the effects of syphilis itself, or a mere coincident attack of eczema, may not explain these cases. Hebra, with a scepticism justified by his enormous experience, denies that any eruption on the skin is ever brought about by the internal use of mercury. The cases reported by Behrend and Engelmann were erythematous, and sometimes complicated with scarlatina.

* Cavafy, 'Clinical Society's Transactions,' vol. x, 1877, p. 88.

† Hans von Hebra, 'Die Krankhaften Veränderungen der Haut,' p. 204. He, however, is convinced that the relation is merely accidental. See also Hutchinson, 'London Hosp. Reports,' vol. v.

‡ 'Observations on Hydrargyria,' Dublin, 1804; London, 1810.

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DISORDERS OF THE SEBACEOUS GLANDS, THE HAIR-SACS, AND THE SWEAT-GLANDS

"If Rosalinda is unfortunate in her Mole, Nigranilla is as unhappy in a Pimple."
ADDISON.

ACNE—*Nomenclature—Anatomy and course of local lesions—Distribution—Age and sex—Symptoms—Ætiology—Treatment—Acne tarsi—Acneiform eruptions produced by tar—by bromide—Acne varioliformis.*

Comedones without inflammation (acné cornée)—Milium—Seborrhœa oleosa—Seborrhœa sicca—Xerodermia—Steatoma, meliceris and sebaceous cysts.

MOLLUSCUM CONTAGIOSUM — *Name and history—Anatomy — Pathology — Treatment.*

SYCOSIS—*Name—Anatomy and course—Locality—Diagnosis—Distribution—Treatment—Parasitic sycosis—Sycosis capillitii frambœsiformis.*

FURUNCULI—*Pathology—Anatomy—Course and distribution—Age—Contagion—Treatment—Carbuncle.*

AFFECTIONS OF THE SWEAT-GLANDS—*Anidrosis—Hyperidrosis—Bromidrosis or fœtid sweat—Chromidrosis—Hæmatidrosis—Sudamina.*

IN the long series of inflammatory diseases of the skin we find certain affections which may be arranged, on clinical as well as anatomical grounds, in a third large group. The first we considered was that of the chronic forms of dermatitis which in various degrees resemble the common superficial inflammation produced by irritants. Traumatic eczema, idiopathic, symmetrical, weeping eczema, papular eczema, lichen, lichen planus, pityriasis rubra, and psoriasis—these form a natural group, of which pemphigus is an outlying member. Although there is no evidence that their pathological relationship depends upon the presence of an antecedent dartrous or arthritic or gouty diathesis, yet they undoubtedly are really related to each other. The erythematous group of affections treated in the preceding chapter, to which pemphigus may be considered as the link, form as natural though not so extensive a family.

The present chapter deals with inflammatory processes which do not affect the skin generally, but only the hair-sacs and cutaneous glands. They have also, as we shall see, peculiarities of distribution and of natural history which are no less characteristic than their anatomy.

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ACNE.*—This disorder referred to by Cicero, Martial, and other classical writers, but as a blemish rather than a disease.†

The sebaceous glands become occluded, either by their secretion being too thick or by want of cleanliness in removing the accidental obstructions from dirt. The first effect is to produce a number of small, firm, and somewhat pointed papules (*comedones*), each of which is produced by accumulated sebum and is marked by a black head, which is nothing but the dirt obstructing the orifice of the gland. This condition, which has been named *acne punctata*, may continue for an indefinite time, but sooner or later some of the papules show signs of irritation, and in most cases this very speedily supervenes in each obstructed gland. The papule becomes red, swollen, and before long yellow from suppuration having taken place. This pustular form or pustular stage of acne is no less characteristic. When the surrounding inflammatory œdema is considerable the deformity is of course increased. At last the minute abscess bursts, and the inflammation slowly subsides. When slight, no trace remains, but a second inflammatory process with the same course and termination often follows. When severe, a minute white scar is left behind, the gland is destroyed and incapable of renewed action. When the hair-sac into which the sebaceous gland opens is deep, the inflammation is the more severe, and sometimes causes a minute slough which leads to the pain and swelling characteristic of a furunculus. Such little boils are naturally slower in their course and lead to deeper scars. The face or shoulders may be seen covered with acne spots in all the above stages, so that the pain and irritation become great and the deformity distressing. The chronic form used to be called *acne indurata*.

Histology.—A section of an acne pustule shows not only the papillæ but the deeper layer of the cutis œdematous and filled with leucocytes, and the small blood-vessels dilated. In the pustular stage the leucocytes increase in number and assume the character of pus-corpuscles; the acini and duct of the gland are filled with pus, often mingled with blood-discs. The process in the larger acne pustules is found to affect the hair-sac into which the sebaceous gland opens, so that the hair itself is uprooted and the entire follicle destroyed. When the destruction of the papillæ has taken place—in other words, when the inflammation has become "deep" instead of superficial—a scar always results after the acne is cured.

Dr Liveing has found that in a sebaceous sac which is the seat of a comedo many minute abortive hairs may often be found, the growth of which may perhaps be the immediate cause of obstruction.

It may here be observed that the minute parasitic mite known as *Demodex folliculorum* is frequent in healthy sebaceous glands and never causes acne.

Distribution.—Acne is confined, almost without exception, to the *face*, shoulders, and chest. It usually begins about the forehead, the cheeks, the alæ of the nose, and the chin; but pimples may cover the whole of the face and the intervening skin be occupied by an erythematous dermatitis. Comedones, as the black-tipped early lesions of acne are called, are also to be generally seen on the auricle, but here they rarely suppurate. On the *back* the pustular and indurated form is more common, perhaps because it is

* *Synonyms.*—Acne vulgaris—Acne disseminata.—*Germ.* Finnenausschlag.

The derivation of the word is unknown, but is commonly supposed to be a corruption of ἀκμή, and to refer to its occurrence in the prime of life. Its proper Latin name was *varus*, and it was called ἰονθος by the Greeks, and also ἀκμαί.

† "Pæne ineptiæ sunt curare varos et lenticulas et ephelidas; sed eripi tamen feminis cura cultus sui non potest." (Celsus, 'De Med.,' lib. vi, cap. v.)

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more apt to be neglected; and there it is that we see the most extensive cicatrices. The lesion may extend from the back of the neck and the scapular and interscapular regions down to the loins, but very seldom lower, nor does it pass round the flanks towards the chest and abdomen. A few scattered papules may be sometimes found over the deltoid or on the upper arm. The skin over the *sternum* is the least frequently affected of the three acneic regions. The lesions are precisely the same and never extend to the abdomen, the axillæ, or the front of the neck.

Occasionally isolated comedones or acne-pustules may be found elsewhere, most often on the outer side of the thigh and peroneal surface of the leg in men with coarse hairy skins and large follicles. These, however, are not more common in the subjects of acne than in other persons, and are either accidental or connected with inflamed lichen pilaris.

Age and sex.—This singular follicular inflammation is in its origin and greatest extent confined to the age of puberty and early adult life, although acne when thus begun may continue up to thirty or even later.

Comedones may be seen in a few rare cases in children; but though numerous and apparently characteristic they do not suppurate, and they are found upon the forehead and even on the scalp without the characteristic distribution of true acne.

Any chronic inflammation of the skin, eczema, recurrent erythema, and especially that form which will be described as gutta rosea, may lead to pustular inflammation of the hair-sacs, so that the latter affection has been commonly described as *acne rosacea;* but the distribution, the origin, and the whole natural history of the two diseases are different. In fact, the more closely the subject is studied, the more decisively does true acne separate itself from all other affections.

The disease most commonly begins in lads of about sixteen, that is to say, when the changes which accompany puberty have already begun. It is not common for it to make its first appearance after the beard has begun to grow, but it may begin at from sixteen or seventeen up to one or two and twenty. It is very slow in its progress, and the worst cases are usually those of a year's standing or more. When once thoroughly established the morbid process continues until the beard has fully grown, but in most cases it then begins to subside and seldom continues after the age of thirty. When acne occurs in a patient above this age, it is usually confined to the back and has been preceded by ordinary acne of the face. This, as well as the occasional occurrence of severe acne of the shoulders with only slight affection of the face, is probably sufficiently explained by the greater attention given to a visible eruption and the less efficient treatment of all affections which cannot easily be reached.

Although the evolution of acne is, as we have seen, so closely connected with that of the beard at puberty, yet the disease is very far from being confined to the male sex. Indeed, Erasmus Wilson stated in the first edition of his treatise that acne occurs more frequently, perhaps, in the female than in the male, and Dr Bulkley reports the same from New York, where he found, in nearly a thousand cases of acne, 319 occurring in men to 654 in women. This is not the case in the writer's experience, but acne is common enough in young women about the same time as in lads, or perhaps a little later. Probably there are more male sufferers, but more female patients.

The affection is, as a rule, more diffused in the case of women, the

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papules more numerous, not so large and with more erythema between. It is also more often confined to the face; and it is certainly much more rare to see the worst forms of acne indurata, and the disfigurement which follows, in women than in men. On the other hand, while it is somewhat later in its appearance it is decidedly slower in its disappearance, so that acne may be more often seen about the age of thirty in women than in men, and it is chiefly in women that a lingering acne is overtaken by an early gutta rosea, a combination which has no doubt helped in confusing the two disorders.

Course and Symptoms.—Acne is always a chronic affection lasting, if left to itself, for years, but liable to occasional exacerbations. These often coincide with ovarian disturbances in women; in men they are less marked, but sometimes appear to be connected with gastric disturbance, especially with the more acute forms of indigestion, such as in some people result from eating pork or salmon or preserved viands, whether salt, like herrings, or sweet, like crystallised fruits and jams. There is but little local irritation, and the other organs are completely unaffected; indeed but for the disfigurement few patients with acne would apply to the physician.

Ætiology.—The immediate cause of acne is the obstruction and inflammation of the sebaceous glands, and in the severer cases of the hair-sacs also; but when we ask why this obstruction and inflammation occurs, the answer is extremely difficult. To say that the presence of acne indicates a disordered state of the cutaneous nerves, which interferes with the vascular action of the skin; to say that it depends on torpidity of the capillary circulation or general want of cutaneous activity; to say with Biett that it is the result of keeping the head bowed down, as in many sedentary occupations, or of drinking cold water when heated, or of smoking tobacco; or with Alibert, that it is caused by spending nights in gambling and living in anxiety—all this is trifling with pathology. It may be asserted that acne has no such connection with feeble circulation, as is shown by chilblains, nor with local irritation as eczema solare, nor with gout or tubercle, nor with the ingestion of cold water or hot water, or alcohol, or any kind of food, nor with any diathesis or disposition to anything but acne.

It is obvious, if we consider its natural history, that acne has to do with the great change which passes over the organism at the time of puberty; first and principally with the growth of the beard, yet not as a mere mechanical result, for in the great majority of men the beard appears without acne,—men have acne who never develop a beard, and women frequently have it also. Acne, moreover, affects the skin of the shoulders, which is unchanged at this period, as well as that of the face and chest, where hair grows, and it does not affect the hair of the pubes.

It is stated by Rigler ('Die Türkei und deren Bewohner,' Wien, 1862), quoted by Hebra, that acne, though common in the Levant, is extremely rare in eunuchs. Moreover, in young women affected with acne the eruption is often aggravated during the menstrual period. There is no reason to adopt the suggestion of Rayer, followed by many French writers, that acne is connected with vicious habits.

The old adage of Plenck, "Matrimonium varos curat," is well exchanged for Hebra's dictum, "Tempus varos curat." It is not continence nor vice, nor celibacy nor marriage, nor even the growth of a beard, which are the causes of acne; it depends upon the general changes which occur in the passage from childhood to adult life. The glandular apparatus of the skin

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is then apt to be disordered, most apt on the region where the beard is developing and loses this aptitude when complete development is once attained. With regard to acne in women we can only say (as conversely of hysteria in men) that though they have no beards their fathers had, that is that secondary sexual characters are more or less transmissible to both sexes.

Acne attacks those in good health and those in ill-health, the blonde and so-called lymphatic, as well as the dark and atrabilious; it is said to be seldom seen with red hair, and to be less common in Ireland than in England.*

There is no proof that acne is an hereditary disease, although it is not infrequently seen in brothers and sisters, and although the disposition to its development at puberty would, we might expect, be transmitted more or less completely, in the same way as the early growth of a beard, its weakness or abundance, and the early or late supervention of baldness.

Prognosis.—Few cases of acne cannot be decidedly relieved by careful treatment, and in many the face can be restored to its natural appearance; but success depends not only upon the physician's adapting his treatment to the wants of each case, but also upon the perseverance with which the patient will follow it out. In many cases irremediable mischief has already been done when the patient comes before us. "Tempus varos curat," though generally true, proves often tedious in performance, and when such a cure is complete the disfigurement it leaves is often considerable.

Treatment.—In the early stage of acne, when comedones are present with little or no inflammation, the principle of treatment is to set free the obstructed ducts, to keep them clear by extreme care, and to stimulate the local circulation. The plan found most successful is the following: On going to bed the face should be first steamed over a basin of boiling water. It should then be thoroughly washed with a piece of flannel and yellow soap, and dried with a rough towel. On careful scrutiny in the glass the patient will then find that the acne punctata has lost a good many of the black points; but he should go over the whole of the face, and wherever a pimple shows by the slightest point of yellow that suppuration has begun, it should be emptied—not by squeezing with the fingers, but by pressing over it the end of a key of suitable diameter. When this has been effectually done, the face should be again washed and a lotion applied which should be allowed to dry. This drying lotion may be of sulphur suspended in liquor calcis, alum water or lead lotion, or a dilute solution of corrosive sublimate (gr. ¼ with tr. benz. co. ʒss in ℥j of mist. amygd.). The old cosmetic known as *lac virginum* was of somewhat the same composition, as was also the famous Gowland's lotion, which is said by Bateman to have contained oxymuriate of mercury in an emulsion of bitter almonds.† The sulphur is the more stimulant; the mercurial wash when too strong is apt to cause a feeling of constriction and tension of the skin. Next morning any fresh pimples which have ripened should be emptied and the face again washed with soap and

* According to Bazin acne is of scrofulous origin; and even Hardy, while denying this, thinks that acne has a preference for lymphatic subjects, although "on peut avoir un tempérament lymphatique sans être atteint de scrofule."

Dr Erasmus Darwin, who properly distinguished acne from gutta rosea (in his 'Zoonomia'), nevertheless named the former affection *gutta rosea hereditaria*, "because it seems to be hereditary, or at least has no apparent cause."

† "Merely Gowland," said Sir Walter Elliot, "I should recommend Gowland, the constant use of Gowland, during the spring months." ('Persuasion,' vol. ii, chap. 4.)

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water and a little dilute mercurial ointment (ung. hydr. ox. rubri with two parts of benzoated lard) applied to each. With many patients thorough washing and the application of white precipitate ointment serves the same purpose very well.

The same plan of treatment answers, even if a good many pustules are present, supposing that there is not much inflammation around them; but the more pustules there are, the less vigorous should be the friction used and the more important it is to apply some ointment containing mercury to the pustules. Ung. hydrarg. ammon. or dilute citrine ointment are often well borne; in other cases the unguentum metallorum (p. 775, *note*) suits better.

When the inflammation, judged of by the erythema between the papules, by the amount of swelling, or by the presence of true furunculi, is severe, we must begin with other measures. Steaming is still useful and generally proves soothing, but friction must be much more sparingly used, and sometimes omitted altogether. Instead of stimulating lotion or ointment, the patient must at bedtime use Goulard wash or a drying lotion of oxide of zinc suspended in water, or the almond wash may be used alone; during the day lead ointment or zinc or a combination of the two must be applied, but for women and others who are not obliged to be out of doors, the frequent application of lead lotion is better than ointment.

It is in these cases only that diet needs regulation by abstinence from stimulants, spices, and the other viands which we found tend to excite erythema of the face (p. 833). It may also be desirable for the patient to take a little carbonate of soda or citrate of potash with a saline laxative. By these means the erythematous inflammation will soon be subdued, and it is then desirable to return as quickly as may be to the more stimulant treatment.

In inveterate cases the stronger mercurial ointments are indicated. When, as occasionally happens, large furunculi are present, they must be treated with carbolic oil, and if necessary with poultices.

Acne of the shoulders, though often severe, is naturally less troublesome than that of the face; the skin is also less susceptible to irritation and almost always bears rougher treatment with advantage. The individual attention to the several papules which is so important in the case of the face is of course difficult to carry out here, and we must depend more upon the use of mercurial ointments and on friction with rough towels or flesh gloves.

Acne tarsi.—Anatomically allied to acne is the inflammation which not unfrequently, especially in children, affects the large and specially modified sebaceous glands that serve to lubricate the eyelashes. These Meibomian glands are apt to become the seat of chronic inflammation, when a gummy secretion is exuded which sticks the eyelids together. It may either occur independently or as a complication of catarrhal or other forms of ophthalmia. It is generally cured by the application of unguentum hydrarg. ammon. or yellow oxide of mercury ointment.

Tar and bromide acne.—Inflammation of the sebaceous glands, papular or pustular, is occasionally called forth by external irritants, and especially by tar. This so-called tar-acne differs, however, in its distribution and natural history from the true disease, and needs no treatment but the removal of the exciting cause.

Bromide-acne is the name given to a somewhat similar follicular inflammation caused by the internal use of the bromides in certain patients (p. 835).

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*Acne varioliformis.**—This name was given by Bazin, who has been followed by other French authors, to what will presently be described as *molluscum contagiosum.* It is unfortunate that Hardy has accepted such a confusion of nomenclature and of pathology. The name has, however, been since applied to a rare and curious affection which consists in large pustules resembling those of the more severe kinds of acne, and situated chiefly upon the forehead, the temples. and the sides of the cheeks. After they have burst and healed a deep scar is left, sometimes pitted but not pigmented, resembling that which follows the most severe forms of acne. It is in these scars that the resemblance of the affection to variola chiefly consists, for the distribution, the course, the absence of a vesicular stage, and the unimpaired health of the patient could never allow of its confusion with variola. In a patient of the writer's, a man about forty, the affection encroached upon the scalp, and also spread to a considerable part of the chest, shoulders, and back. In this, as in the other cases, this curious affection was quite distinct from true acne. It is not preceded by comedones, and it seems doubtful whether the pustules are really seated in the sebaceous glands. The distribution and the severity of the eruption also distinguish it from acne, and it is unconnected with the period of puberty. It is more difficult to distinguish it from a pustular syphilide, and some cases which have been described as acne varioliformis were probably syphilitic. In one patient, in whom there was neither history nor proof of venereal disease, the pustules, which had lasted for a long time, disappeared under iodide of potassium. Dr Liveing, however, has seen instances of acne frontalis which resisted anti-syphilitic treatment and yielded to large doses of arsenic. We must wait for further observations before the true nature of this affection can be decided.

It will be convenient to refer briefly in this place to other affections of the sebaceous glands, less important than acne.

Comedones are generally the first stage of that disease, and then occur in the persons and under the circumstances above described; but beside the accidental comedo or even pustule which may be produced here and there by obstruction of a duct in any part of the body, and which no more make acne than one swallow makes a summer—there are occasionally to be seen large numbers of comedones in children affecting the forehead, scalp, and other parts, not undergoing inflammation, and without the locality or other characters distinctive of acne.

Acné cornée is the name given by French writers to this remarkable and rare condition, which does not deserve the name of acne, first, because it is not inflammatory, and secondly, because it has not the natural history of the disease of that name. It consists in the presence of a multitude of *comedones*, which remain as passive papules, hard pointed and black tipped. They occur in children before the age of acne, and upon the scalp and other parts unaffected by acne. We have had several cases of this singular condition, which requires to be distinguished from "lichen pilaris." Once it occurred on the forehead and scalp of two brothers, once in a brother and sister between seven and nine years old, once on the temples of a boy of eight or nine suffering from pleurisy, and once on the lumbar region of a girl aged thirteen, under treatment for erythema multiforme, but without a trace of

* Neumann, following Hebra, calls it *acne frontalis*, and describes it as a variety of true acne. Dr Bulkley, of New York, names it *acne atrophica*, after Chausit's *acné atrophique*, or lupoid acne. See a case figured by Dr S. Mackenzie in the 'Clin. Trans.' for 1884, p. 227, and remarks by Dr Mackey in the 'Lancet' (Jan. 22nd, 1876).

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acne, or even comedones on the face, chest, or back (see 'Guy's Hospital Reports,' 3rd series, vol. xiii, p. 213).

Milium.—A commoner condition is passive obstruction of a sebaceous gland with complete occlusion of the orifice. A minute white or yellowish papule is thus formed without the pointed top or the black mark of a comedo. It has been called milium from its size. It never inflames, and is of no practical importance; it occurs most often on the thin skin of the eyelids and the genitals; occasionally it grows larger than a pin's head; when this occurs it usually affects only a single gland. Its contents then not unfrequently become liquid, and it forms a small cyst, such as may be occasionally seen on the eyelids, and have been noticed by Mr Hutchinson to occur in association with xanthelasma and with sick headaches. In young children it is not uncommon and corresponds to Willan's *Strophulus albidus.*

On dissection the acini of the gland are found filled with a dark refracting substance, which yields on analysis cholesterin, olein, palmitin, and stearin. A good drawing is given by Neumann (fig. 10).

Seborrhœa.—The functional disorders of the sebaceous glands may lead to too abundant liquid secretion, or to too solid scaly products, or by suppression to abnormal dryness and harshness of the surface. The first of these conditions has been named *seborrhœa oleosa* or steatorrhœa. It is physiological at a certain period of life, when it forms the *vernix caseosa* of newborn infants. It is not uncommon about the face, and on especially the alæ of the nose. It also occurs on the genitals, where a local vernix caseosa may lead to pruritus and inflammation: cleanliness and a little lead lotion or ointment is sufficient treatment.

The affection described by some authors as seborrhœa corporis has been mentioned above as *Lichen circumscriptus* (p. 794).

Seborrhœa sicca appears to depend upon the more solid fats, stearin and palmitin, being secreted in greater abundance than the liquid olein. The secretion forms little yellowish scales, added to by the natural desquamation, and frequently by the local irritation of a slight dermatitis which increases the desquamation. The condition is most common on the scalp, where it constitutes what is known as pityriasis capillitii, dandriff, or scurf. In most cases this is rightly termed seborrhœa sicca; but, although it begins as a sebaceous affection, in cases which have lasted long one finds that the scales consist in large part of epidermic cells, and there is often beside local irritation, injection, and other signs of dermatitis. That this is secondary is shown by its not spreading beyond the scalp, and by its being unconnected with eczema or psoriasis of the scalp. Beside the irritation of this common disorder, it undoubtedly leads to the hair becoming thin and weak, and in some cases produces early alopecia.

The treatment is not very satisfactory. Mild mercurial ointments or carbolic oil sometimes prove useful.

Xerodermia.—Diminution or absence of sebaceous secretion leads to the skin being dry, harsh, and apt to crack. The sweat-glands may be active and abundant; but sweat is apt to exert an irritant effect upon the skin undefended by its natural oily secretion. This condition is usually congenital, and was rightly described under the name of "xeroderma" by Wilson as the slightest degree of ichthyosis. It will be again referred to under that head.

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A similar state of skin is, however, not unfrequently observed in children who are thin and ill-nourished, and in patients of any age suffering from prolonged wasting diseases, especially phthisis. The diminution of subcutaneous fat is accompanied with diminished supply of oily material to the sebaceous glands, and the skin becomes dry, pale, rough, scaly, and dirty. This condition, which is of only symptomatic interest, has been described as *asteatosis* and as *pityriasis tabescentium.*

The only treatment indicated is to supply the deficient oily material by inunction with olive or cod-liver oil.

STEATOMA.—When the orifice of a sebaceous gland is obstructed and an accumulation of the secretion takes place, it does not always inflame; the secretion may go on until a large cystic tumour is formed.

The orifice of the duct can still usually be found, and sometimes by mere pressure the contents can still be evacuated. They consist of inspissated sebum without, on the one hand, the pus which mingles with the secretion in inflamed acne, and without, on the other hand, the remarkable modified epithelial cells which will presently be described as characteristic of molluscum. When the watery parts are absorbed, the sebaceous secretion consists of the ordinary animal fats, palmitin, stearin, and olein, some butyric and caproic acids, either free or united with glycerin to form neutral fats, a small amount of albumen, or rather globulin, with a larger proportion of casein, epidermic cells, the flat tabular crystals of cholesterin, and earthy salts. Occasionally the fatty material appears to be absorbed as well as the water, and there remain behind only calcareous masses like those found in a diseased aorta, or in the apex of the lung in a case of obsolete phthisis. These cutaneous calculi are, however, of very rare occurrence. The yellow, somewhat granular, half liquid, and half solid mass has been compared with porridge, with putty, and with mortar. Although the word *atheroma* (from ἀθήρη, oatmeal) is now generally applied to the very similar products of chronic inflammation of the arteries, yet "atheromatous tumour" is still used by some writers as synonymous with what is otherwise called *Steatoma, meliceris*, or, perhaps better, a sebaceous cyst.

These tumours, called, when they attain a large size, *wens*, most frequently occur upon the scalp, where they are often multiple, and may grow to the size of a fist, or even bigger. They may also be seen upon the eyebrows, face, and neck, less frequently on the trunk, and most rarely upon the limbs.

Sometimes a sebaceous cyst is pedunculated instead of sessile (*acrochordon*).

Occasionally normal sebaceous secretion is again excreted after the duct has been long blocked; and this may continue for several years.

Cysts with similar contents, but of very different origin, and, probably (some of them certainly), congenital, occur on mucous membranes and in deeper parts of the body, especially about the root of the tongue and hyoid bone; and such *cholesteatomata* have also been described in the brain and in the bones. Many of them are true dermoid cysts and may be compared with those of the ovary, which contain not only sebaceous matter but hairs and sebaceous glands.

A steatoma is easily recognised by its smooth rounded surface, the presence of a dimpled occluded orifice, the absence of pain, and its situation.

Sebaceous cysts are perfectly innocent, but occasionally require removal

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from their inconvenience or unsightliness. The plan usually adopted is to incise the tumour and tear or dissect out the secreting cyst wall, or, if this be difficult, to rub the interior with caustic.

MOLLUSCUM CONTAGIOSUM.*—This somewhat rare disease was first described by Bateman, who added it to the small group of Tubercula as defined by Willan. The case figured by him in his 61st plate, on the face and neck of a young woman, was a typical example of the disease, and Bateman traced the contagion from a nursling of this patient and two other children in the same family back to a fourth patient with the same affection. He also mentions a second case in an infant apparently contracted from an older child. It was from these facts that the epithet *contagiosum* was applied by English physicians,† and also by von Bärensprung, Virchow, and Rindfleisch, and although the correctness of the epithet has often been doubted it is now satisfactorily proved.

Carswell, Rayer, and other writers recognised Bateman's disease, and Huguier, in 1846, had described it as a non-syphilitic affection of the vulva. Caillaux, in his 'Treatise on Diseases of the Skin in Children,' named it "acne molluscum."

The disease occurs in the form of small rounded tumours of a pink colour, sometimes sessile but more often pedunculated. They are scattered irregularly over the skin, which remains quite healthy between them. Their number varies from a single tumour to a countless multitude, and the size from that of a vetch, to use Bateman's comparison, or a large pin's head, to that of a marble or occasionally much larger dimensions. The colour also, though usually pink and waxy, is sometimes scarcely distinguishable from that of the skin, and at others it has a dead white or even yellowish tint. These last, however, are probably not uncomplicated examples of the disease. A minute dimple is to be found on each tumour, which is the orifice of a sebaceous duct. This disputed point, however, will be presently considered. The growth of the little tumours is very slow. They may retain a size not exceeding a pea while increasing in number during many months, and perhaps longer. As they grow bigger they become more rounded and the groove at their base becomes deeper until they may hold to the skin by only a slender pedicle, a condition which was formerly described as *acro-chordon*. The colour usually becomes paler and more translucent as they increase in size, but this is not constant.

No *symptoms* are produced in the most marked and typical cases; as before stated, the skin between the little tumours is perfectly normal, and they themselves are no more than a disfigurement.

* *Synonyms.*—Molluscum sebaceum—Epithelioma molluscum—Acne varioliformis.

† Wilson, 'Diseases of the Skin,' 1842, p. 302; Paterson, 'Edinburgh Medical Journal,' vol. lvi, 1841, pp. 213, 240. Cases were also recorded by Alibert, Biett, Cazenave, Schedel, Gibert, and Jacobovitz, 'Le Molluscum: Recherches Critiques,' Paris, 1840. Most of the foreign cases, however, were examples not of Bateman's disease but of what will afterwards be described as fibroma molluscum.

The term molluscum, there is no doubt, was, as Mr Wilson suggested, taken by Bateman from the celebrated case of Tilesius, described by C. F. Ludwig, of Leipzic, in 1789. His words are, "Corpus tectum est verrucis mollibus sive molluscis." The word is obviously used as a synonym of mollis just as mollusca was first applied to the mollusca nuda et testacea, the *soft*-bodied animals or malacozoa. Alibert, followed by Cazenave, misinterpreted the meaning of the term. Bazin unfortunately described the disease under the term *Acne varioliformis*, and this has led to much confusion, especially since the same term has been employed for a singular variety of acne mentioned above (p. 844) before its misapplication to molluscum contagiosum had been forgotten.

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Anatomy.—On incising the tumour, a white opaque thick material can be usually squeezed out, and a hollow sac remains flaccid behind. Herein a molluscum tumour resembles an ordinary sebaceous cyst or steatoma, but the contents are white instead of yellow, and to the naked eye have not the atheromatous appearance so characteristic of accumulated sebum. Moreover, tested chemically and microscopically, instead of fat, cholesterin and earthy salts and epithelial scales, the white material seems to be made up almost entirely of characteristic oval transparent bodies with a pearly lustre, without a nucleus and not readily staining with logwood. These have been described as "molluscum corpuscles." They were first recognised and well described by Wilson in 1842; they were rediscovered at Saint Louis and described as cryptogamic spores, the source of the contagion. This view, however, is certainly incorrect. Their size, their aspect, their reaction to potash and their inability to develop are conclusive against it. No doubt they are epidermic cells which have undergone a hyaline transformation. Along with these molluscum corpuscles there is often found a certain amount of fatty sebaceous material; but in some well-marked cases there is an entire absence of sebum.

A section horizontal to the surface shows that each little tumour is made up of loculi more or less separated from each other by septa, and in most cases a central axis may be demonstrated, which is supposed to be the duct of the gland. The question remains whether the origin of the cyst is in the sebaceous gland, and whether the metamorphosed epithelial cells are derived from those lining its acini or from those of the duct, or whether the whole tumour is a new growth unconnected with the sebaceous apparatus and starting in the deeper epidermic cells. The latter is the opinion held by Virchow and supported by Dr Sangster's observations.*

Distribution. — Molluscum is most common on the face and neck, especially the eyelids and cheeks, and also on the mammæ of women; but when a multitude of small tumours occur, they may be found upon the arms, especially the thin skin of the flexor surface, as well as on the face.

Mr Hutchinson describes molluscum as not uncommon on the penis and scrotum of young adults, and refers to a case of Dr Paterson's where similar tumours appeared on the vulva of a woman whose husband was thus affected. He also refers to the molluscous growth occasionally suppurating, and points out that it may resemble an indurated chancre.

In the cases which the writer has seen of molluscum occurring on the male genitals, the tubercles have been yellow and not either pearly and translucent nor pink and waxy looking, and on incision or even on pressure without incision have yielded opaque yellow oily material, so that they should rather be called steatomata. In the Guy's Hospital museum, a model shows the appearance of molluscum upon the thigh ('Catalogue,' p. 240). Sometimes a large molluscous tumour will suppurate, burst, and thus cure itself. A case of this kind occurred under the late Dr Addison in a little girl ten years old.

Molluscum contagiosum is most common in infants and children, less so

* See papers by Drs Morrison, Crocker, and Thin in the 'Pathological Transactions' for 1881, p. 245, and also a description by Mr Davies-Colley, 'Guy's Hospital Reports,' 3rd series, vol. xviii, 1870, p. 350, and figs. 1 and 2, p. 364. He describes most of the characteristic oval cells as nucleated. Virchow's original paper was published in 1865 in the 33rd volume of his 'Archiv,' p. 144; and Thin's in the 'Journ. of Anat. and Phys.,' vol. xvi, p. 202.

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in women, and decidedly rare in men. In children it almost always affects the face, in women the mammæ, and in men the genitals. But this is only true of the larger and fewer tumours. All the cases of numerous very small molluscum simulating warts and sometimes described as molluscum verrucosum which the writer has seen in adults have been situated on the arms. Mr Huchinson has observed similar cases in which the trunk or lower extremities have been so covered with little tumours as to resemble some papular eruption, as lichen.

Molluscum appears to be much more common in England than abroad, though cases of the true disease are reported from France and Germany. It is well known in Scotland and also in America. The majority of cases occur in dirty neglected children, but it is often seen in those who are clean, rosy, plump, and in every respect healthy.

Ætiology.—There can be no doubt that the epithet contagious is rightly applied to this disorder, notwithstanding the frequent failure produced by inoculation and the incredulity of Hebra and other dermatologists. The subject is well discussed by Dr Duckworth in two interesting papers in the 'St Barth. Hospital Reports' for 1868 and 1872. No fungi and no bacteria have yet been observed; but the transformed epithelial cells above described are found to contain corpuscles like pseudo-navicellæ, which Darier identifies with psorosperms (cf. p. 784).

Treatment.—Molluscum tumours, beside, as above stated, sometimes suppurating, which appears particularly apt to occur when they are confluent, may also undergo passive involution by gradually shrinking and subsiding. This must be the case with many infants who have never come under medical treatment; but these modes of spontaneous cure are rare or slow and uncertain, while treatment is rapid and efficient. Each tumour should be removed either by being snipped off with a pair of sharp scissors curved on the flat, or by being incised and emptied. In either case the whole of the diseased structure must be removed. Where the tumours are very numerous, it may be better to apply nitric acid or the acid nitrate of mercury to each one in the early stage. When the growths are not larger than pins' heads, Mr Hutchinson believes that white precipitate and sulphur ointment in equal parts will cure the affection; but it is rare for an opportunity for this treatment to occur.

SYCOSIS.*—Closely allied to acne is a disease which consists in pustular inflammation of the large hair-sacs of the beard. Anatomically it is difficult to draw a broad line between them, for although we speak of acne as inflammation of the sebaceous glands, yet since all these glands open into a hair-sac, obstruction of their duct and obstruction of the corresponding hair-sac are almost the same. The anatomical difference lies in this, that whereas on the general surface of the body the hairs are small with shallow sacs, and the sebaceous glands large, in a man's beard the hair-sacs are

* *Synonyms.*—Mentagra—Varus mentagra—Acne menti vel barbæ—Barber's itch.—*Fr.* Impétigo sycosiforme (Devergie), Adénotrichie (Hardy).—*Germ.* Bartfinnen.

The name *sycosis* was applied to this affection by the Greeks, from its supposed likeness in the worst cases to the inside of a ripe fig, the red pulp answering to the inflamed and swollen skin, the seeds to the little pustules. "Est etiam ulcus quod a fici similitudine σύκωσις a Græcis nominatur" ('Celsus,' lib. vi, cap. 3).

The terms *mentagra* and *lichen menti* of Pliny and Martial, like the sycosis of the Greeks, were often applied to syphilitic affections of the lips and other parts, probably to what we now call mucous patches and condylomata; but the Latin *ficus*, like the Greek σῦκον, was certainly applied not only to condyloma ani but also to hæmorrhoids.

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large and deep, and the glands comparatively small. Moreover, there is no doubt that in acne it is the gland which is first obstructed so as to form a comedo and which afterwards inflames, while in sycosis it is the hair-sac which is the primary seat of disturbance.

It was Bateman who first accurately defined the characters of sycosis in the modern sense of the word. He placed it under Willan's order Tubercula on account of the hard swelling which often surrounds the pustules.

Anatomy.—Hebra expressed the opinion, since supported by Liveing and other observers, that the immediate cause of a sycosis pustule is the presence of two or more hairs growing together in the same follicle. Wertheim, in 1861, published a paper in the 'Transactions' of the k. k. Ges. der Aertze of Vienna, in which he referred the origin of sycosis not to the growth of more than a single hair in the follicle, but to its being abnormally thick.

Whatever the immediate cause, suppurative inflammation takes place in the hair-sac, and the hair-bulb becomes loosened, but the shaft still blocks the sac. The drop of pus first formed is pent up and produces pain and fresh inflammation. By the time that the hair is at last detached, a small but deep cutaneous abscess has formed, and considerable congestion and œdema around it has produced what Willan called an inflammatory tumour or nodule. When the pus at last finds exit, it dries into a scab; and this is rendered much more adherent than that of impetigo by the numerous hairs which tether it to the skin. Fresh accumulations of pus take place beneath it, and thus in severe cases of sycosis a most repulsive and "malignant" aspect may be produced.

Excluding parasitic sycosis, which is of course contagious, Hebra and most German writers maintain that inflammation of the hair-sacs of the beard is non-contagious; but this seems to be very doubtful. As before stated (p. 787), pus is itself in many cases an extremely contagious product. We see that it is so in cases of contagious impetigo and of furunculi, and therefore it is wise to consider all cases of sycosis as more or less capable of spreading by inoculation of neighbouring hair-sacs in the same person, or even under favourable conditions, to another person.

It is right to add that many good observers maintain that, apart from ordinary ringworm of the beard, pustular sycosis vulgaris can in many cases be proved due to original infection with *Trichophyton tonsurans*, though they admit that the fungus is often impossible to find.

Course.—If left to itself, sycosis is a most obstinate disease. The hair-sacs are successively destroyed and cicatrices result which are sometimes deep and obvious. When at last the disease has worn itself out, the greater part of the beard is often permanently destroyed and the face disfigured by scars. The affected part is usually very tender, though, except when touched, there is rather tension and heat than severe local pain.

Distribution.—As explained above, the peculiar kind of inflammation described can only occur in large and deep hair-sacs like those of the beard. The disease usually begins upon the chin, frequently on the upper lip, on the cheeks, or under the jaw. It may, however, occasionally be observed in the eyebrows and on the pubis, and still more rarely similar pustules have been seen on the chest, thighs, and other hairy parts.

Bateman asserts that women are not altogether exempt from sycosis, and Wilson admits that in rare instances it has been seen in a female patient. This, however, is a question of diagnosis (or at least of definition)

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rather than of fact. The typical disease, excluding acne, inflamed ringworm, impetigo, and all syphilitic affections, is confined, if not absolutely, with the rarest exceptions, to the chin, lips, and cheeks of male adults. It is rare to see it in a soft beard which has never been shaved.

Diagnosis.—Sycosis must be distinguished from eczema of the face, for ordinary papular eczema and impetigo sometimes invade the cheeks and lips, and simulate sycosis. It is possible for the dermatitis thus produced to penetrate to the deep hair-sacs, and then a condition ensues which must be termed true secondary sycosis. But this is certainly very seldom the case; the superficial dermatitis as a rule preserves its superficial character, the pustules and crusts are those of impetigo, and when removed leave the surface but little affected; the treatment by ung. hydr. ammon. is simple and rapidly successful; no scars are left behind, and no hairs are destroyed.

Treatment.—In cases of true sycosis which affects the hair-sacs, mercurial ointments must be combined with epilation. It is not, however, necessary in most cases to remove all the diseased hairs, and certainly not healthy ones. It is enough if those which are already loosened are extracted, so that the rule is for the patient to pluck out all the hairs which will come easily and without pain, that is, those which are already detached from their sacs. The first step in severe cases is to steam the face, and, if necessary, to soften the crusts with poulticing and sweet oil; then to remove the loose hairs with broad-pointed forceps. The beard should be cut short, but not shaved. If there is much local pain and swelling the inflammation should first be subdued by lead lotions, lead and zinc ointments, or other soothing and astringent applications. When this is accomplished the treatment above advised should be begun and followed out day by day. In most cases the result is successful. When cure has resulted it is generally better for the patient not to shave for several months, but to allow the beard to grow naturally.

Not infrequently, however, sycosis proves very obstinate in spite of all care and diligence. The possible presence of a parasitic cause should in such cases be carefully looked for. In the most obstinate cases complete epilation on Plumbe's and Hebra's plan is no doubt the only effectual treatment, but it should be carried out piecemeal, and with the help of previous application of potash soap, and other remedies which soften and loosen the hair. During epilation dilute red oxide, or better, perhaps, the yellow oxide of mercury ointment, should be rubbed into the surface.

Bateman recommended diluted unguentum hydrarg. nitr., or white precipitate ointment with an equal part of zinc or lead.

Parasitic sycosis.—Since Gruby, in 1847 ('Gazette Médicale,' No. 37), published an account of a cryptogamic plant which he discovered in cases of sycosis, French writers have generally described sycosis as a parasitic affection. Bazin named it Teigne Mentagre. Gruby's name for the fungus was *Microsporon metagrophytes*, but Bazin and Rubet proved that it is identical with *Trichophyton tonsurans*, the name given to that of common ringworm in 1846 by the Swedish writer Malmsten. So also Köbner in 1864. Hardy followed Bazin, and diverged from Cazenave and from Biett, who had placed sycosis among pustular dermatoses, for he practically maintained that all sycosis is parasitic. In his recent work, however, while regarding "true sycosis" as a teigne sycosique, he admits non-parasitic sycosis as an impetiginous inflammation of the hair-sacs.

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Hebra, after reviewing the statements of previous authors, affirms that in more than 300 cases of sycosis or "follicular inflammation of the beard" he has never seen a single case accompanied with parasitic fungi.

There can, however, be no doubt of the existence of what the French writers call parasitic sycosis. The writer saw many cases at St Louis, and others, though much more rarely, in London. He never saw a case in Vienna, and those who have studied dermatology in several schools will probably agree that this is one of the instances in which we must admit local differences in the frequency of diseases. Parasitic sycosis certainly does occur, and will be found if looked for in London, but it is far less common here than in Paris; and it must be added that the presence of the fungus is often only to be ascertained after prolonged and repeated search. When detected, however, the case acquires at once a new character, and for practical as well as scientific reasons it is desirable to separate "parasitic sycosis" from the non-parasitic disease.

*Sycosis capillitii.**—This title of Willan's should perhaps be given to five remarkable cases of sycosis or pustular eczema of the hair-sacs observed by Hebra on the occiput and nape of the neck.

This very rare condition was named by Kaposi *dermatitis papillomatosa capillitii.* Hans von Hebra ('Archiv für Derm. und Syph.,' 1869, p. 382) prefers the name, suggested by his father, of *Sycosis frambæsiformis.* He describes it as beginning in very small, somewhat red papules, each traversed by a hair, which grow together and form hard tumours resembling raspberries, and at last end in a long, tough, cheloid-looking band. The disease occurs on the nape of the neck where the hair is growing; the skin around is eczematous and red, and the place painful. The course is very slow, and as new papules arise they fill with thin pus. When raised flat papules have been formed, hairs are seen pushing out in bundles. This, with the hardness of the growth, its extreme slowness of development, and its locality, are the characteristic points. It may, however, occasionally occur on the scalp. This same affection has been described by Dr Sangster as "a papillary tumour of the scalp" (Internat. Med. Congr., 1881), by Dr Vérité as *Acné kéloïdique* (Académie de Médecine, 9 Mai, 1882), and by Mr Morrant Baker, with a figure, under the same title ('Pathological Transactions,' 1882).

The treatment of this remarkable disease consists in destruction with caustics or removal of the tumours while still small with a sharp spoon, by galvano-caustic, or by other means. When the disease has already gone far excision is the only remedy.

Histological sections show that there is true enlargement of the papillæ, very scanty exudation of leucocytes, and gradual formation of parallel and interlacing bundles of fibrous tissue, among which the sebaceous and sweat-glands are squeezed and atrophied.

FURUNCULI—*Boils.*—Recognising the complete impossibility of any complete and satisfactory classification of skin affections which can set forth

* *Synonyms.*—Sycosis frambœsiformis (Hebra)—Dermatitis papillomatosa capillitii (Kaposi)—Acne keloid. The sycosis capillitii of Willan, p. 66 of his 'Atlas,' is not unlike the curious affection described on p. 845 as *Acne varioliformis.* The *Pian ruboide* of Alibert (pl. 35) may have been the same disease, unless we suppose with Bateman that these were mismanaged porrigo favosa, or with Hebra that they were undiagnosed syphilis.

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all their complicated mutual relations, it seems convenient to associate the troublesome and painful affection of boils with acne and sycosis; for although it is not possible to demonstrate that the seat of inflammation is always in a hair-sac, yet in many cases this may be readily ascertained and it is probably true of all. However, the depth of the inflammation, its pustular character, and the scars which it leaves behind, are points in which it is closely related to the affections described in this chapter, and particularly to the deep and painful suppuration which affects the glands of the vibrissæ of the nostrils and the ears.

The characteristic pathological feature of furunculus is that the inflammation leads to the death of a minute portion of the deeper layer of the cutis. This slough or core of necrosed connective tissue is passed out by a process of liquefaction and suppuration, and the abscess which is formed then slowly heals. In its early stage the disease appears as a pimple, distinguished by its excessive pain, a pain which resembles that felt from the plucking out of a hair, and in all probability depending upon inflammation of a hair-sac under somewhat different conditions from the comparatively painless pustules of acne and sycosis. The papule speedily shows a yellow spot at its pointed summit and this little pustule is never preceded by even a transient vesicle. Meantime, a bright, intensely injected halo appears around the pustule and considerable inflammatory œdema swells the whole skin into a conical elevation; the pain increases and becomes throbbing in character, while the dull constant aching and sense of tension is varied from time to time by sharp stabbing pains. When the abscess has ripened and is lanced or bursts of itself the core becomes visible, and is sometimes not expelled for a day or even longer. This stage is accompanied by a sharp pricking pain which is very characteristic; the pain rapidly subsides when the core is got rid of, a small scab forms, the redness and œdema disappear, and soon nothing but a minute scar remains.

Unfortunately, however, it is seldom that this process is confined to a single furuncle. Most often a second and a third appear before the first is completely healed, or a whole crop may spring up almost simultaneously. A succession of painful abscesses may thus be established and last for weeks or even months, until the patient's health seriously suffers from the pain and the discharge. Sleeplessness, loss of appetite, and much depression, both physical and mental, may be the result. When a crop of boils thus appears, they are generally found to vary in size and severity, from those which are so large and deep as to challenge the name of carbuncle, to small superficial pustules which formerly would have been called ecthyma.

Distribution.—There is scarcely any part of the surface which may not be the seat of a furunculus, but the affection has nevertheless a decided predilection for the back of the trunk, from the hair at the nape of the neck to the fold of the nates. The thick cutis, thin epidermis, and small but numerous hairs of the dorsal region appear to furnish the most favourable conditions for this kind of inflammation. The back of the neck, especially at the edge of the scalp, is perhaps the most frequent seat of all; the buttocks come next to the nape of the neck in liability to boils. Moreover, the friction of the collar of the dress in the latter situation, and that occasioned by riding or by rowing in the former, aggravate the misery of the complaint and probably keep it up.

Boils are far from unfrequent in the coarse skin of the outer part of the thighs, which resembles that of the dorsal and scapular regions anatomically.

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They may also appear, though less frequently, on the leg below the knee, on the upper and forearm, on the wrist, and on the back of the hand. The chest and abdomen, and even the face, are not exempt, but boils very seldom occur on the scalp, and never on the palms, nor the soles of the feet. They are rare on the male genital organs, but not unfrequently occur in the neighbourhood of the anus, in the perinæum, and on the vulva.

Ætiology.—The true cause of this form of inflammation is unknown. There is little ground for supposing that boils are the result of indigestion or of overwork or exhaustion from any cause; when numerous and long continued they produce, but are not the product of, anæmia and weakness. Nor, on the other hand, do they come from plethora and over-richness any more than from poverty of the blood. In the practice of the water-cure it is customary to wrap patients who suffer from dyspepsia, paralysis, and most other chronic diseases in wet sheets, which are then surrounded with blankets; free perspiration is thus produced, and in many cases it is offensive in odour and accompanied by a copious crop of boils and pustules. This is supposed to indicate the efficiency of the plan in bringing out the poison from the system, but in reality it only means stimulation of the sudoriparous glands, possibly vicarious excretion of urinary or fæcal products, and certainly traumatic inflammation of the skin.

Age.—Boils are far the most frequent during youth. They are rare in infants, and not common in early childhood, but schoolboys are very liable to them, especially to the most characteristic form of successive crops on the neck and shoulders or on the nates. After thirty, this painful affection becomes decidedly rare, and we seldom find boils in an elderly man. Women during the whole of life seem less liable than men, though some of the most severe cases occur in young women.

Furuncles are sometimes contagious from one patient to another; and almost an epidemic may sometimes run through a school; but more frequently they spread from place to place upon the same patient.

Treatment.—If this view be correct it furnishes an important indication for treatment. While the first furuncle is developing, no doubt a poultice gives great relief from its warmth and the relaxation of tissue it produces, but by making the skin sodden and softening the epidermis it predisposes it to inflammation and renders the access of the chemical or morphological contagion of the pus more easy. The constant application of poultices over large surfaces affected with boils very much tends to spread and continue the disease. A much better plan is to dispense as much as possible with poultices, using water dressing instead, and to apply to the skin immediately around the boil the lotio plumbi, Goulard wash, or a somewhat stronger lead lotion. Tannic acid may be used with the same object; or, as each boil appears, a circle may be drawn around it with tincture of iodine or dilute solution of silver. Sometimes collodion, especially the flexible collodion applied in the same way, seems to act best both as an astringent and a protective. Meantime the pustules should be covered with lint soaked in carbolic oil (one in ten) and the same antiseptic dressing should be continued after the pustule has burst. It was formerly the practice to open each boil successively with the lancet, but though the process is undoubtedly thus hastened, the pain and the dread of the pain are so severely felt, especially in young people, and when a sensitive part of the body is affected, that at least in such cases the furunculi may be left to ripen and burst of themselves.

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There is no evidence of special advantage in the purges and alteratives which are the traditional treatment for persons affected with boils. Where only one or two exist no internal treatment is necessary; but where the crops are numerous and successive, the treatment above indicated by local astringents and antiseptics should be combined with the internal administration of wine or porter with the meals, and of either bark with mineral acids or tincture of steel in full doses. Small doses of calcic sulphide are frequently prescribed for boils as well as for other chronic suppurative affections, and apparently with benefit. Thin delicate boys will often be much benefited by cod-liver oil, either alone or, if anæmia indicates it, in combination with steel. There is reason to believe that a stay at the seaside is particularly useful during convalescence.

CARBUNCLE.—This term, as the diminutive of *carbo*, is the Latin translation of ἄνθραξ, a coal, and was applied to any red, angry, inflamed pustule. The word anthrax has in recent times been restricted to the disease known as splenic fever, accompanied with a characteristic boil or carbuncle of the skin derived by contagion from cattle and associated with the presence of a specific bacillus, which has been fully described in the first volume (p. 350).

A carbuncle is pathologically identical with a boil, differing only in its severity and extent, but its natural history is sufficiently different to justify the old distinction being retained.

Anatomically, a carbuncle is the inflammation which accompanies a considerable cutaneous and subcutaneous slough. It differs from the larger and deeper boils by the affected tissue being so extensive that not a single opening forms, but several, giving a characteristic perforated aspect to the broad summit of the tumour. If left to itself this gradually opens by ulceration, and a deep and wide aperture is formed, through which the slough is at length extruded, often with considerable hæmorrhage. The surrounding redness is commonly deeper and more lurid in hue than that of a boil; the œdema also is more extensive. A carbuncle almost always occurs singly. Its most frequent seat is the nape of the neck and the shoulders; it may occur on any part of the trunk, but is rarely seen on the limbs or the buttocks. Occasionally it appears on the face, and is then severe and often dangerous. Carbuncles are more common in the old than in the young, and frequently occur in diabetes (p. 574).

AFFECTIONS OF THE SWEAT-GLANDS.—The sudoriparous glands are less liable to disease than the sebaceous, and their affections are less important from a local, though far more so from a symptomatic, point of view.

Anidrosis, or deficiency of sweat, is seen as a concomitant of many forms of pyrexia, and usually accompanies erythematous and roseolous eruptions. In most of the forms of superficial dermatitis, also in psoriasis and in pityriasis rubra, little or no sweat is secreted, and probably the same is true of eczema madidans. Ichthyosis, including "xerodermia" (dry skin), is also marked by absence of sweat. That the function of this secretion is only supplementary to that of the kidneys and lungs as an excretion of water, and that its chief purpose is not excretory but regulative of temperature, is shown by the fact that patients with universal ichthyosis or pityriasis rubra show no symptoms of blood-poisoning from retained excreta.

Hyperidrosis.—General or profuse secretion of sweat takes place under two conditions. First, along with hyperæmia; this occurs in health during

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the natural sweating and warmth of skin induced by active exercise, and pathologically in rheumatism and the sweating stage of ague. Secondly, profuse cold perspirations take place with an anæmic state of the skin, as in the cold perspirations of terror, the night sweats of phthisis, and the cold perspiration which sometimes marks the approach of death. Modern physiology teaches that the vascular supply and the epithelial activity of sweat-glands, as of other secreting organs, are governed by distinct nerves.

Local hyperidrosis, when it affects the hands and feet, is sometimes the source of considerable annoyance. Astringents are often useful, particularly tannin and alum. In a troublesome case, profuse perspiration of the palms of the hands in a young lady was cured, after other treatment had failed, by the local application of belladonna. Internally the same drug is indicated by our knowledge of the physiological action of atropine upon the submaxillary gland.

Bromidrosis and *osmidrosis* are names given to fœtid perspiration, which is usually also excessive. This most frequently affects the feet, and may become a source of the utmost discomfort. The persons it affects are almost always young adults, and women more frequently than men. A horrible stench results from decomposition of the fatty matter which mingles with the sweat, particularly the fatty acids which belong to the formic acid series—butyric, caproic, and caprylic. Dr Thin has figured a bacterium to which this decomposition is probably due ('Proc. Royal Soc.,' 1880). The chief seat of evil-smelling sweat beside the feet is the axilla.

The treatment of this distressing affection is often extremely difficult. The first step is to check the secretion by astringents, and to prevent its soaking into the clothing by absorbent powders, such as lycopodium; the next is, by frequent change of linen, to remove the products of excretion as rapidly as possible. Antiseptics like thymol and salicylic acid may be usefully applied, and the latter preparation, especially in the form of collodion or a salicylic plaster, has the further advantage of softening the accumulation of macerated cuticle. With the same object Hebra used to envelope the foot and toes in strips of diachylon plaster, and many can testify to the efficiency of this treatment, the details of which will be found in the English edition of his work (vol. i., p. 89). Dr Thin found a saturated solution of boracic acid efficient ('Brit. Med. Jour.,' Sept. 18th, 1880).

Chromidrosis is the name given to the occasional secretion of coloured sweat. The sweat of the axillæ in some persons contains enough pigment to stain their linen of a reddish tint. The writer has met with one well-marked case, and Hoffman has recorded another in the 'Wiener med. Wochenschrift' for 1873, No. 13. Sometimes, however, a bluish pigment stains the sweat on the face or elsewhere. Cases of supposed chromidrosis occurring in young women should be watched. In most cases the apparently dark sweat is an *arte factum*. But although most supposed cases have proved to be factitious, there is no doubt that true chromidrosis does occasionally occur; and in some cases it has been proved to depend on indican being excreted in the sweat, and turning to blue indigo when oxidised by exposure to the air. Dr Foot published a case in the 'Dublin Quarterly Journal' for August, 1869, and collected no less than thirty-seven others. Another source of colour is the production of blue or greenish fungi in decomposing sweat. When coloured sweat affects the eyebrows it is usually of black colour, looking almost like soot. A remarkable case of red-coloured sweat was reported by Dr Wilks in the 'Guy's Hospital Reports' for 1872.

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In this case a chemical analysis by Dr Thomas Stevenson proved the presence of iron but the absence of hæmoglobin.

Uridrosis, or the excretion of urea in the sweat, probably only occurs as a morbid phenomenon. The observations of Funke on the normal excretion of urea through the skin have not been confirmed, but in Bright's disease urea has been visibly discharged in the sweat (p. 474).

Hæmatidrosis (or *hæmidrosis*), or bloody sweat, is an extremely rare but undoubted morbid condition. It does not appear to accompany purpura or other diseases in which one would anticipate such hæmorrhage from changes in the blood or capillaries; and in some of the very few authentic cases it appeared during apparent health, as in that of a friend of Hebra, who observed the exudation of blood-stained sweat upon his hand while sitting at table. A similar condition has been observed in the lower animals.

Dysidrosis was the name given by the late Dr Tilbury Fox to a curious affection of the skin of the hands ('Path. Trans.,' 1878), described as *chiropompholyx* by Mr Hutchinson ('Lancet,' 1876). It consists in large vesicles without any surrounding inflammation, occurring in groups upon the palm and back of the hand and the fingers, especially near the web. These vesicles have been compared to sago grains, though they sometimes reach a much larger size. Rasori, who published a case in the 'Transactions' of the International Medical Congress for 1881, vol. iii, p. 146, calls it hydro-adenitis diffusa. Hans v. Hebra records a case in his 'Kr. Veränderungen der Haut,' p. 426. It affected the palms and soles of a woman forty-two years old, and some of the larger bullæ were surrounded with a red halo. Mr Hutchinson has observed relapses of this singular affection on several occasions. Whether it depends, as Dr Fox supposed, upon obstruction of the duct and accumulation of its contents is doubtful. Possibly two rare affections have been confounded: bullous erythema and true retention-blebs of sweat (cf. p. 819).

Sudamina.—However this may be, there is a well-marked cutaneous affection which undoubtedly depends upon accumulation of sweat in little vesicles under the cuticle, and has been known for centuries as ἴδρωα, sudamina, or miliaria. It is often seen during the profuse sweating of rheumatism. The orifice of the duct becomes obstructed, and the horny cuticle is raised as a thin transparent layer enclosing a drop of transparent fluid (*miliaria crystallina*). This ruptures before it exceeds the size of a pin's head, but sometimes the contents become turbid and alkaline from slight inflammation. On the chest and back these sudamina are most common; they never occur on the face, and on the thick skin of the palm attain larger dimensions before they burst, so as to form the "sago-grain" vesicles of dysidrosis.

The profuse sweat which causes sudamina also produces, especially if not quickly removed, local irritation. This seldom goes beyond the stage of papules or erythematous redness except where it is aggravated by friction. The commonest seat for this *dermatitis a sudore* is the vertebral groove from between the shoulders to the sacrum, and the front of the chest.

The more severe inflammation which occurs in the axillæ, between the toes, and between the cheeks of the nates, is known as *intertrigo*.

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RINGWORM AND ITS ALLIES, WITH OTHER AFFECTIONS OF THE HAIR

"Under thy long lockes thou maist have the skall."
CHAUCER.

RINGWORM—(1) *Tinea tonsurans—Anatomy—Course—Events—Histology—Detection of the fungus—Ætiology—Prognosis—Treatment—Parasiticides—Irritants—Mode of application—Epilation—Precautions against contagion*—(2) *Tinea circinata—Form and locality—Burmese ringworm—Tinea marginata—Treatment of ringworm of the body*—(3) *Onychomycosis—Rarity and obstinacy.*

FAVUS—*History—Anatomy—The fungus—Treatment.*

TINEA VERSICOLOR—*Names—Parasitic nature—Appearance—Distribution—Diagnosis—Treatment—Tinea vel Pityriasis rosea.*

ALOPECIA—(1) *Physiological*—(2) *Febrile and syphilitic*—(3) *Area—its appearance—locality—spread—question of its parasitic nature—prognosis—diagnosis*—(4) *Universal alopecia*—(5) *Congenital alopecia.*

TRICHOCLASIA, *or brittleness of the hair—Piedra—Beaded hairs.*

WE have seen in other parts of this work that the human body is liable, besides animal parasites, to the invasion of the lower forms of vegetable life. The Schizomycetes, often spoken of generally as *Bacteria*, are by far the most important of these, since they probably form and certainly convey the contagion of some, possibly of all specific fevers. They are described at p. 14 of the first volume.

Of far less practical importance are the fungi which are parasitic on the human body. Some of these affect the mucous membranes and have been already described in the present volume as *Oidium albicans* in the mouth, p. 134, and *sarcina* in the stomach, p. 197. We have now to consider diseases of the skin which depend upon the growth of similar microscopic fungi. In most cases the cryptogamic spores and mycelium lodge in the deep hair-sacs of the skin.

We will take, first, the most important of this group, then the remaining parasitic affections, and, lastly, it will be convenient to deal in this chapter with non-parasitic affections of the hair which need to be distinguished from ringworm.

RINGWORM OF THE SCALP.*—This troublesome disease was only proved to depend on the presence of a cryptogamic parasite in 1844 by Malmsten, the Swedish microscopist. He named the fungus *Trichophyton tonsurans.*

Origin and spread.—We seldom see the earliest stage of the disease, but the first obvious effect of the entrance and growth of the fungus in the hair-sacs is for the affected hairs to lose their glossiness and colour and become dry, shrunken, and brittle. They break short and probably thus expose fresh

* *Synonyms.*—Tinea tonsurans, the Porrigo scutulata of Willan.—*Fr.* Teigne tondante.—*Germ.* Herpes tonsurans, Kopfgrind.—*Angl.* The Skall (in part), the Ringworm.

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spores to spread the contagion. At the same time the growth of mycelium in the hair-sac produces slight irritation, partly from the inflammation directly excited and partly from the patient's scratching. Moderate hyperæmia and corpuscular exudation follow, so that by the time a small bare patch appears it is raised, slightly red, and covered with a few scales. The process extends, partly by the spores being conveyed to fresh places, partly by their steady advance to the next adjacent hair-sacs. Thus, one, two, and often numerous round patches are developed, each of which closely resembles the other. The form is often geometrically circular, sometimes oval or irregular; the hair is replaced by a few broken, dark, and thick stumps, which can be recognised by the naked eye, while their characters are still more obvious under a lens; the surface is usually covered with greyish-yellow desquamation composed of epithelial cells and sebaceous material mixed with broken hairs, spores, and mycelium. The scales have a uniform, granular, closely adherent look, which is almost decisive to a practised eye. At the edge of the circle a little redness may sometimes be observed, occasionally a few papules, and, still more rarely, a vesicle or two. In the immediate neighbourhood individual hairs may be found by the aid of the microscope to be already affected by the spreading evil.*

A ringworm patch may increase to several inches in diameter without materially altering its appearance, but more often it is modified as it expands in one of the following ways.

Either from scratching or from the effect of the fungus on the naturally irritable scalp, or as the result of irritant applications, more or less of ordinary superficial dermatitis appears, so that many cases of ringworm appear as impetigo capitis, and their true nature is not manifest until the scabs and crusts have been removed. In neglected cases, moreover, pediculi are not unlikely to breed, and further aggravate and confuse the condition. Such horrible masses of felted hair, mingled with inflammatory products, vegetable and animal parasites, and all kinds of filth, constitute the *plica polonica* of Eastern Europe, which may still be sometimes seen at Vienna.

On the other hand, if the hair is kept short and the head clean, and if the skin is not naturally irritable, the fungus, while spreading at the edges of the patch, appears to exhaust the soil in the centre, and dies away, like the larger cryptogamic fungi which form "fairy-rings" upon the grass. The result is that the middle of the patch is more or less completely bald, with only a few short stumps or thin, feebly-growing hairs, while the circumference is occupied by a zone of flat brownish scales, granular desquamation, papules, and broken hairs. This is the most typical form of traditional ringworm, and probably suggested the specific title *scutulata*. When growing patches of the disease meet they form figures of 8 or dumb-bell-shaped patches, and, as they still grow and unite with others, irregular gyrate figures, like those of old-standing psoriasis, of erythema marginatum, or of syphilodermia. At last almost the whole scalp may be invaded, and reduced to baldness. There is, however, never a perfectly smooth clear skin left, as in alopecia areata, but a few ill-developed, thin, pale, scattered hairs are always to be found. Moreover, the process is seldom or never

* They may be more easily detected by the naked eye if, as Sir Dyce Duckworth suggested, chloroform be first applied ('Brit. Med. Journ.,' November, 1873). This gives the affected hairs a dry, pale, brittle look, like that of burnt-up hay, apparently owing to its solvent power on the oily constituents of the hair. But this reaction is far from decisive alone.

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quite universal; on one or the other temple or on the occiput more or less unaffected portions of hair remain.

Events.—The disease does not spread continuously beyond the scalp, but fresh patches arise, sometimes in the eyebrows, occasionally in the beard, more frequently on the skin of the neck and shoulders, and even on more distant parts. If left to itself the course of the disease is extremely chronic, and shows little or no tendency to recovery—if the patient is a child—until the period of puberty is reached. It must not, however, be supposed that among neglected children in a village or a school, where ringworm has invaded the community and scarcely a child has escaped, the disease constantly assumes the severe and inveterate character above described. A single bald patch may remain for months or years, or it may more or less completely recover, and fresh patches go through the same series of changes; or, what is still more important to notice, the spores falling upon an unfavourable soil continue to multiply, and are thus a fresh source of contagion, but yet do not sufficiently interfere with the nutrition of the hair to produce obvious bald patches. In a family or school in which ringworm has appeared one may find evidence of its presence in the heads of children who are entirely without the characteristic bald patches.

Histology.—If one of the broken stumps of a ringworm patch be extracted with forceps, and placed in a drop of liquor potassæ under a quarter-inch objective, it may often be at once recognised by its opacity. When less densely packed with spores, or when soaking in potash has cleared it, the condition is equally manifest by the complete destruction of all the normal histological characters of human hair. The cortex and medulla are undistinguishable, the surface is rough, the pigment no longer normally distributed, and the free end, instead of tapering to a point or being transversely cut off, is broken, slightly bulbous, ragged, or split into a sheaf of fibres. A less degree of infection is recognised by a few spores in nucleus-like chains or a little branch of mycelium, in the substance of an apparently healthy hair. Dr Frederick Taylor has pointed out that the parasitic fungus does not invade the cutis itself, nor even the follicle, and only slightly affects the adjacent epidermis. The inner root-sheath is full of spores, the outer root-sheath free. (Compare his paper, 'Med.-Chir. Trans.,' lxii, with Dr Thin's, ibid., vol. lxi.)

The spores differ from oil-drops, with which they are often confounded, in the following particulars: first, they are uniform in size; secondly, they do not run together; thirdly, they are not perfectly spherical, but some at least perceptibly spheroidal or oval; next, they do not refract light so strongly, and though glistening and having a well-marked outline, the centre is not so bright, nor the circumference so broad and black; they occur in little groups or in chains; lastly, potash, instead of dissolving them by forming a soap, as it does with oil-drops, is powerless to affect their protoplasm, which is protected by a cell-wall; thus it only serves to bring them out clearly by making the surrounding keratin and oily matter transparent. Ether is also without effect. Carmine and other staining agents act slowly, but in the end stain the cell. Often the most characteristic objects are not the extracted hairs, but short broken fragments, which are conveyed to the glass-slip with scales and *débris*.

Diagnosis.—The recognition of ringworm is in most cases sufficiently easy after a little experience, but we must remember that it may be masked

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by secondary impetigo, as above described; also, that when of long standing it may produce patches of almost bald skin which may simulate the atrophic patches to be described (p. 872) as alopecia areata; and, thirdly, that the trichophyton may exist in hair which, as explained above, does not show the ordinary signs of ringworm which are visible without a microscope. In all doubtful cases, therefore, we must depend upon careful microscopical observation. This is particularly important when we have to decide whether the disease is cured or not. It is only by taking numerous specimens that we can assure ourselves of the fact. We must sometimes, where to the naked eye the ringworm has disappeared, hunt through a dozen slides without finding a single diseased hair, until in the last we may find unmistakeable evidence of the ringworm being still incompletely cured.

Ætiology.—The only efficient cause of ringworm is the growth of the *Trichophyton tonsurans;* and its almost universal spread under favourable circumstances shows that individual difference of soil has but little to do with it. All that we can see are differences in the luxuriance of its growth, in the irritation it occasions, and in the obstinacy with which it clings to the affected scalp.

It has often been stated that ringworm occurs chiefly in pale thin children, who are called "scrofulous" or "strumous," without enlarged glands or any sign of tubercle. There is little evidence for this opinion, or for its supposed predilection for light-haired, "lymphatic" children. One often sees ringworm in those who are the picture of rosy health. It occurs more frequently in light-haired children than others because most children in England have light hair, but it is common enough in those with brown hair, black hair, or red hair.

What is really important in its ætiology is that it is most frequent between the ages of three or four and nine or ten. It is not very common in infants, and when present is usually cured without difficulty. This probably depends upon the less development of hair. Why ringworm of the scalp is so seldom met with in adults is difficult to say. Not only do mothers and nurses rarely take the disease from their children, but when it does occur it is far more readily cured. In children above ten or twelve years old it is easier of cure than in younger ones, and about fourteen or sixteen years of age its treatment seldom gives trouble, and it sometimes disappears spontaneously.

Among 100 consecutive, mostly private, cases of ringworm under the writer's care, 13 of the patients were between twelve months and five years old, 34 between five and ten, 40 between ten and fifteen, 3 between fifteen and twenty, 9 between twenty and thirty-four, and one was aged fifty.

Ringworm is probably as common in boys as in girls; but of the above 100 patients, it happened that 63 were male and only 37 female.

It must be remembered that the lower animals are liable to this disease, and the source of contagion may sometimes be a cat or a horse.

Prognosis.—In infants and in adults ringworm of the scalp is a very manageable disease; in children, though the majority of cases may with care and attention be cured, it often proves obstinate, and now and then, in spite of the best available treatment, may persist for years, and at last yield to advancing age alone. In a school or a family, from a third to a half of the cases will be cured in from three to nine weeks, a few of them by three or four days' application of the remedy. The majority of the rest will yield to persevering treatment in from three to six or eight months. A

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few only out of a large number will last beyond this time, and some of these are pretty sure to prove inveterate.

Treatment.—The principle of treatment is the same as that of scabies. In both cases we know the cause of the disease; we know the natural history of the invading organism and the means of checking or destroying it. The difficulty in the case of ringworm is that most frequently before the case comes under our observation the fungus has already fixed itself deeply in the hair-sacs of the scalp, and it is extremely difficult to apply remedies to reach it. It is moreover protected by the epithelial scales which closely surround the hair-bulb, and by the sebaceous and other products which block its mouth. We shall see that when the same parasitic growth invades the surface of the body its cure is easy.

So great are the practical difficulties of treating ringworm of the scalp that, although with perseverance and skill we can cure the vast majority of cases, and some of them rapidly as well as safely, yet everyone who has had much experience in this disease must have met with cases which are so intractable that when after many months or even years they at last get well, it is to time and the increasing age of the patient that the cure is due.

Preparations of *mercury* are poisonous to all cryptogamic plants, to bacteria as well as to fungi, and probably the most poisonous is corrosive sublimate. A solution of perchloride of mercury in alcohol, two grains to the ounce, is sometimes rapidly effectual in curing recent cases of ringworm. It should, however, only be applied to separate patches, since there is at least one case on record in which its free use over a child's scalp produced (by some unusual accident in the application, or possibly some idiosyncrasy in the patient) absorption of the drug, and death by mercurial poisoning. Lotions, however, have the disadvantage of being repelled by the oily sebaceous infiltration of the natural and diseased structures of the scalp. We therefore usually prefer lard or vaseline as a vehicle, and in early cases of ringworm the white precipitate ointment (*ung. hydrarg. ammon.*) is often completely successful. It should be well rubbed into each patch morning and evening after thorough cleansing with hot soap and water and flannel. Instead of white precipitate ointment the *oleate of mercury*, of the strength of one in twenty or one in thirty-five, is effectual, and by many preferred to the older preparation. The 10 per cent. oleate is too strong unless applied to a very small patch in an elder child.

The effect of contact with white precipitate and with citrine ointment upon the life of the *Trichophyton* has been experimentally studied by Dr Thin. His results confirm those gained from clinical experience, as to the sterilising power of the mercurial ointments, while he found that fats and oils including oleum ricini have no such effect in themselves.

Another parasiticide which has become popular is tincture of *iodine*. This also is sometimes effectual with recent cases.

There is, however, another method of destroying the fungus which is often found to be practically more efficacious. It consists in setting up a local inflammation, the products of which destroy the parasite. This plan is most applicable to the first stage of the disorder. If a mercurial application does not prove effectual within a few days, then, with elder children, and especially on the first appearance in the family, it is probably better, after isolating the infected member, to attempt the immediate destruction of the fungus by exciting local inflammation. A stronger solution of iodine acts in this manner, but probably the most effectual and least

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painful application is the blistering fluid made of *cantharides*. The affected spot should be first shaved, including half an inch around it, and a circle of oil be drawn round the margin to prevent the blistering fluid from spreading. The pain of its application does not last long, and in many cases success is immediate and complete.

Too often, however, the fungus has already spread too far to be treated in this decisive manner, which is scarcely applicable except to recent cases with only a single diseased patch.

We will suppose that a child is brought to us with the disease established for several weeks, with numerous rings, and, perhaps, with crusts and pustules from attempts to cure by various irritant applications. The first step is to have the hair cut quite short over the whole of the scalp. Scabs and crusts must then be removed with the help of poultices, and the whole surface made as clean as possible. We then see the real extent of the primary disease. It is often much less than it at first appears; the secondary superficial dermatitis is readily cured, and the diseased patches are soon ready for treatment. Sometimes we find no impetiginous crusts and little active inflammation, but scattered over the whole scalp small spots of ringworm, while the apparently healthy hair between often furnishes evidence of infection. Under these circumstances the shortest and most effectual way is not merely to cut the hair short, but to shave it completely off. In inveterate cases it is much better to wait until the hair is removed, the crusts or scales got rid of, and the inflamed glands reduced, before beginning active treatment. Meanwhile, the whole scalp should be well anointed morning and evening with carbolic oil, one in fifteen or one in twenty, and the child's head covered with a linen cap both by night and by day. In this way no time is lost, and the spread of the affection to other children is prevented. If, without much active inflammation, there is found considerable accumulation of dead epithelium, and especially when it takes the granular adherent character above described, this must be removed with potash-soap, or other alkaline applications. Dr Foulis ('Brit. Med. Journ.,' 1885, vol. i, p. 536) has recommended for this purpose spirits of turpentine rubbed in until the child begins to feel it tingle, and then washed off with abundant warm water and carbolic soap. This is sometimes a rapid and effectual treatment, but it is only applicable when comparatively small patches are affected, and should not be used in the case of young children.

When the way has thus been cleared for parasiticides, we may in the slighter cases obtain good results by rubbing into each patch the white precipitate ointment as above recommended, anointing the intermediate surface with *carbolic oil*. In many cases, however, this proves inadequate, and we must then use stronger applications, although if the disease is extensive they must be applied only to a limited portion at a time. Equal parts of unguentum hydrarg. nitratis and sulphur ointment form an efficient and usually not too severe application. Dr Alder Smith, whose experience of ringworm at Christ's Hospital has been very large, recommends in obstinate cases a mixture of carbolic acid one part, citrine ointment one part, sulphur ointment one part. With children under ten, two or three instead of one part of the sulphur ointment should be used, and it will then cause no pain. Instead of carbolic oil (1 in 10 or 1 in 5) the carbolic glycerine of the British Pharmacopœia (1 in 4 or diluted to 1 in 8) is often preferred. It is preferable where lotions are being used. Another plan is to use carbolic

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oil (1 in 10) to the generally diseased surfaces and carbolic acid lotion to successive portions; but this is apt to produce more pain and less certain curative effects than the compound ointments.

Some writers recommend *chrysophanic acid*, which is the efficient constituent of Goa powder, much used in the East Indies.* As stated in the chapter on psoriasis, it is sometimes an extremely severe irritant, and always stains both the skin and linen unpleasantly. Chrysophanic acid has been tried, dissolved in chloroform, by Dr Alder Smith (seven grains to the ounce); and he recommends it in recent cases with only one or two spots as more successful than blistering. At the same time he uses a lotion of hyposulphite of soda (two drachms to the ounce) or of liquor sodæ chlorinatæ (one part in eight). Dr Crocker ('Lancet,' January 27th, 1877) reports careful and impartial trial of Goa powder in twenty cases of ringworm. Only eleven were slightly improved after three months' treatment, and only two were cured. Another objection to chrysophanic acid is that it is apt to get into a child's eyes, especially during the night.

A better application in every way is the ointment of *pyrogallic acid*, which is much used against ringworm in Vienna.

Among the more severe applications is one introduced by Dr Coster, of Hanwell Central London Schools, and afterwards published in the 'Medical Times and Gazette,' vol. i, 1867, p. 34. This *Coster's paste* consists of two drachms of iodine dissolved in an ounce of colourless oil of tar, obtained by distillation from common tar and known as light oil of wood-tar or rectified spirit of tar, of sp. gr. 853 to 867. It is applied with a brush to the affected parts and forms a cake which separates at the end of a week or fortnight. (See a letter by Mr Martindale, in the 'British Medical Journal,' January 19th, 1880.) This was used by Dr Ringer at University College with success. Mr Morrant Baker, at St Bartholomew's, preferred iodine in the same proportion with creasote.

The most severe application is *croton oil*, which produces an artificial pustular dermatitis known as "kerion." A favourite ointment both in Germany and in France is that which is also used in the cure of scabies, a combination of sulphur with an alkali (Wilkinson's and Vlemincqx's ointment). Hardy gives the formula: Carbonate of potash a quarter to half a gramme, sulphur one to one and a half gramme, lard thirty grammes.

Instead of ointments or aqueous solutions the cure of ringworm has often been attempted with alcoholic lotions, but until lately without marked success. Dr Cavafy, however ('Brit. Med. Journ.,' June 24th, 1882), has devised a lotion composed of boracic acid, alcohol, and ether, in the following proportions: boracic acid, twenty grains; ether, one drachm; spiritus vini rectif., one ounce. The object, of course, is to dissolve the sebaceous material in the hair-sac and thus enable the boracic acid in solution to soak down to the spores which lurk there. This plan has been adopted and recommended by several dermatologists of experience. The writer has found that this lotion, rubbed into the patches not less than four times a day, has proved cleanly and painless. It sometimes effects speedy cure, but, like all other applications, it not unfrequently disappoints us.

Salicylic acid has also been employed dissolved in alcohol, ether, or

* Chrysarobine is the trade name of Goa powder used at Bombay. It appears to be identical with the araroba powder of Brasil (Thin).

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chloroform; and corrosive sublimate may be used in alcoholic solution—two grains to the ounce.

If watery lotions are preferred, sulphurous acid gas in solution (*Acidum sulphurosum* of the British Pharmacopœia) is one of the best parasiticides. It must be applied on pieces of rag to each patch. Or the hyposulphite of soda (two drachms to an ounce of water) may be used.

Thymol is another unirritating parasiticide which may be employed. It is soluble in alcohol and ether.

Dr Alder Smith recommends Barff's *boro-glyceride* as one of the best applications if the scalp is tender and sore, especially if impetigo is present.

Oleate of copper is an imitation of the old verdigris ointment, as that was of pennies laid in vinegar. It is of a bright green colour and said to be not ineffectual.

With oleates, frequent washing is unnecessary and even undesirable. With solutions, whether in water, alcohol, chloroform, or ether, constant cleansing with common or soft soap is absolutely necessary.

Is it desirable to aid the action of parasiticides by removing diseased hairs? This plan of *epilation* is generally carried out both at Paris and Vienna, and is adopted by many English physicians. Others believe that it is ineffectual. The fact is that to pull out all the diseased hairs over an extensive surface affected with ringworm is impossible even by a skilled manipulator. A certain number are sure to break off in the forceps, and still more are too short to be laid hold of. Moreover, the attempt is extremely tedious and painful, and the result insignificant. Where, however, a very small patch is for the first time seen, it is well to pull out at once all the hairs not only from the obviously diseased skin, but from a small circle around, before applying acetic acid, blistering fluid, or any other agent by which we hope to destroy the parasite at once. Again, in chronic and extensive cases, removing loose hairs helps to prevent contagion, to clear the scalp, and also to ensure minute observation and care on the part of the nurse. It is therefore well to give her a pair of broad-tipped, well-roughened, and weak-springed forceps, and to instruct her to remove every morning after washing the head as many hairs as seem to be loose, but not so as to cause the child pain.

Contagion.—While ringworm is under treatment the whole of the child's hair should be kept short—cut, in fact, as close to the head as may be; and this is probably as effectual as shaving. With girls a fringe of hair may be left round the forehead and behind the ears, so that a cap may be worn during the day, and the child's appearance attract no attention out of doors. At night a linen cap should be used. Impervious coverings of gutta percha or oiled silk make the scalp hot, and are unnecessary.

There is no need for a quantity of ointment to be left on the scalp at night. The free application of carbolic oil, or carbolic glycerine, or oleate of mercury to the head is best undertaken in the morning. Although mothers and nurses very rarely suffer from the most assiduous dressing of ringworm, it is well to instruct them to anoint their hands with carbolic oil each time they touch the child's scalp.

With these precautions, and scrupulous avoidance of contact with caps, brushes, &c., it is possible for a child with ringworm to be treated and cured without removal from the family. But if the infected member or members cannot be separated from those who are healthy, they should sleep in separate bedrooms, and, if possible, meet only out of doors. It generally

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happens that in a family, while most of the cases are cured quickly, there remain one, or perhaps two, extremely obstinate. These may, if necessary, be removed for the sake of treatment; but practically when the child has once been cured it is little liable to take the disease again, especially if the hair is kept short, if carbolic oil is used as a pomade, and if the nurse (who if at all intelligent will by this time be able to recognise the disorder) is careful to wash and inspect the scalp every week.

It is obviously wrong for a child suffering from ringworm to be sent to school, for other children to be admitted to the house, or for its hair to be cut except by its own nurse.

The only proof of complete cure is the careful microscopical examination of the hairs, not only from the previously diseased spots, but from the surrounding scalp. When the skin is itself healthy, and the hair which grows on it is soft and downy, when no broken stumps and black points are seen under a lens, and when these good signs are associated with an absence of spores in the hairs examined, we may pronounce the child to be cured. It should, however, not be sent back to school for at least a fortnight after this, and should then be carefully examined again before the risk of relapse can be considered past.

RINGWORM OF THE BODY.—*Tinea circinata.**—This affection occurs in the form of small rings with a red, papular, vesicular, or scaly margin. They are mostly confined to the face and neck, but are sometimes seen elsewhere on the trunk. Among 50 cases the writer found the eruption confined to the neck, cheek, or other parts of the face in 23, affecting the trunk in 12, the shoulder, arm, or forearm in 10, and the thigh in 5. The majority of the patients were children under twelve, 1 was fifteen years old, 2 seventeen, and 8 were adults. Ringworm of the body seldom causes much irritation.

The disease is contagious, and if scrapings from the ring are placed in potash under the microscope, the mycelium and spores are apparent. There is more of the former in proportion to the latter than in ringworm of the scalp, and the fungus is not so readily seen, but when thoroughly soaked in potash it can always be discovered.

It often appears in children along with common ringworm, but may also be seen when the scalp is quite free from disease. It occasionally occurs in adults, especially in the form which will presently be described.

Burmese ringworm is the name given to what is described by the late Dr Tilbury Fox as nothing but a somewhat severe and troublesome form of Tinea circinata (see his account of this and other exotic forms of ringworm in his work on 'Skin Diseases,' p. 541). *Tinea imbricata* is the name given to the ringworm of Tokelau, Malacca, and other parts of the South Seas by Dr Geo. Turner and Dr Patrick Manson ('Edin. Med. Journ.,' with figs. by Dr McCall Anderson, Sept., 1880).

Tinea marginata.—There is a form of tinea only observed in adults, and of which the parasitic nature was first recognised by Köbner. It was formerly called *eczema marginatum.*

Its distribution is very characteristic. Unlike all other forms of ringworm, it is symmetrical, and occurs only on the thighs, abdomen, perinæum, and buttocks. It begins, probably in all cases, with minute spots, which rapidly form rings; but as these extend and coalesce, they produce gyrate

* *Synonyms.*—Herpes circinatus—Vesicular ringworm—Trichophytie circinée.

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figures, as above explained in the case of psoriasis, erythema, and other disorders which spread at the edge. When a case comes before us it has usually already assumed its characteristic aspect of a somewhat sinuous, broad, yellowish or brownish red, more or less inflamed band, which runs over the upper and inner part of each thigh, passes back to the fold of the nates, or even as high as the sacrum, and returns over the lower part of the abdomen or the groin to the pubes. This peculiar distribution no doubt depends upon the mutual contact of the parts, and is aided by the warmth and perspiration which favour the growth of the fungus. The centrifugal spread is that of all forms of tinea, but the central parts are sooner free from the disease, the margin is more inflamed, and the duration more prolonged than in tinea of other parts of the body.

This curious affection is apparently confined to the male sex and adult age. It is most common in those whose occupation necessitates a sitting position for a long time; thus it is most frequent in cobblers and cavalry soldiers. There is generally much irritation and discomfort; and like all long-continued forms of dermatitis, it produces pigmentation, not only in the growing margin, but also upon the inner exhausted surface.

The microscope demonstrates the same mycelium as is found in *Tinea circinata;* but the disease may last for ten years or more, and when of very long standing it is often difficult, and sometimes perhaps impossible, to discover the parasite. Fortunately the aspect and locality are sufficiently characteristic.

Treatment.—Tinea circinata is very easy of cure. Among popular domestic remedies, ink is one.* White precipitate ointment or oleate of mercury, verdigris ointment (subacetate of copper two scruples, benzoated lard one ounce), tincture of iodine, boro-glyceride, sulphurous acid in solution —may each be employed with a certainty of speedy cure, in striking contrast with their action in ringworm of the scalp. In England there is no need for resorting to the more severe parasiticides, but in India Goa powder (chrysarobine, chrysophanic acid) was first introduced for so-called Burmese ringworm. It should certainly never be employed in the cases which come before us in this country.

Ringworm of the body is, of course, contagious, and may not only propagate itself, but may lead to the development of tinea in the scalp. Its easy cure, however, renders precautions by isolation almost unnecessary.

Tinea marginata, however, is, as above stated, very obstinate and difficult of cure. Sulphurous acid of the British Pharmacopœia freshly made, hyposulphite of soda (a drachm to the ounce), boracic acid (ten grains to an ounce of spirit), and corrosive sublimate (two grains to an ounce of water), may each be used with good effect.

In one very obstinate case, in which the patient, there seemed no doubt, had contracted the disease with a pair of knickerbockers which had been mended by a village tailor in Switzerland, most of these remedies were tried ineffectually for some months. At last the effect of pyrogallic acid ointment (half a drachm to the ounce of benzoated lard) was so rapid and unmistakable that the patient complained of this cure not having been used at first.

Onychomycosis.—It is happily very rare for tinea to attack the nails.

* "How shall we make this mode of writing sink?
A mode, said I? 'tis a disease, I think,
A stubborn tetter that's not cured with ink."—CONGREVE.

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When present, ringworm of the nails is usually a complication of ringworm of the scalp. Cases were recorded by Meissner in Vierordt's 'Archiv,' by Virchow and by Bazin, as early as 1853. It was carefully described and the microscopic appearance figured by Neumann ('Hautkrankheiten,' p. 347, figs. 48 and 49), by Dr Purser ('Dubl. Quart. Journ.,' Nov., 1865), and by Dr Fagge ('Guy's Hosp. Rep.,' 3rd series, vol. xv, p. 553, and 'Clin. Trans.,' vol. i, p. 77). The nails become yellowish and brittle, but not rough as when affected by eczema or psoriasis. The fungus is occasionally that of favus (*achorion*, to be next described), but more often that of common ringworm (*trichophyton*). Good models of onychomycosis due to each of these parasites will be found in the Guy's Hospital Museum, Nos. 536 and 537.

It is the most obstinate of all forms of ringworm, and will often persist during the whole of childhood, and only disappear after puberty.

The *treatment* recommended is scraping the affected nail, softening it with alkalies, and when other means fail, complete removal, together with the sedulous application of sulphurous acid or hyposulphite of soda; but in one case an eminent dermatologist adopted this method, in addition to every other possible parasiticide treatment, without curing the disease.

Tinea erythrasma.—Under this name has been described a slight form of inflammation of the skin, which is due to a fungus, but is probably distinct from tinea versicolor. It occurs, not on the exposed surfaces of trunk, but where two parts come in contact, most often on the scrotum and adjacent part of the thigh, sometimes in the cleft of the buttocks or in the axilla. The patches are pigmented, dull reddish yellow or reddish brown, with a slight branny desquamation. The growth of the fungus is evidently favoured by warmth and moisture, but it does not produce the irritation of intertrigo or of tinea marginata; it spreads very slowly, and, in fact, is rather a blemish than a disease. The fungus was named by Burchardt and Bärensprung *Microsporon minutissimum*. Dr Payne* ('Path. Trans.,' 1886) has described and figured it; and regards it as a specific organism, though mixed with other epiphytes, without spores, and rather like an involution-form of a bacterium than a true fungus. It consists of jointed threads as well as cocci, and requires staining and a high power to be identified. The patches must be distinguished from other kinds of tinea and from syphilitic maculæ. They are readily cured with sulphurous acid or mercurial ointment.

FAVUS.†—This is a rare affection of the scalp and body, due to the presence of a fungus named *Achorion Schönleini*.

The disease was recognised and named by Bateman, and was figured by Alibert. But it was not till 1839 that Schönlein published in Müller's 'Archiv' the discovery that the yellow crusts of favus were neither pustular nor sebaceous, but were composed of the mycelium and conidia of a parasitic fungus. This discovery preceded that of Malmsten above mentioned (p. 858), and therefore to Schönlein belongs the merit of opening the whole chapter of cutaneous mycology.

In its earliest stage favus is probably undistinguishable from common ringworm, but very soon a characteristic flat, round, yellow object is seen,

* 'St Thos. Hosp. Rep.,' 1887, and 'Rare Diseases of the Skin,' p. 31.

† *Synonyms.*—Tinea favosa—Porrigo lupinosa.—*Fr.* Teigne faveuse.—*Germ.* Erbgrind.

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depressed in the middle, opaque, adherent, and perfectly dry. Its colour has been compared to a honeycomb (*favus*), and its shape to the disc of a lupine seed. The sight of a single case of the disease, of such models as Nos. 523—527 in the Guy's Hospital Museum, or even of a well-executed drawing, is sufficient to enable anyone to recognise favus.

The individual crusts grow, coalesce, and form thick, rugged, porous, yellowish masses, resembling the rind of old worm-eaten cheese. They have a characteristic mouldy odour like that of mice.

The disease may affect any part of the body, but is particularly severe upon the scalp, where it destroys the hair-sacs and often produces complete baldness. It is, as above stated, extremely rare in England, but is less so in Germany, and comparatively common in France. It appears also to be not infrequent in Scotland. Mr Hutchinson published forty-four cases with instructive remarks upon the disease in the 'Med. Times and Gazette' for 1859 (vol. ii, p. 553).

Favus has been recorded by Dr Purser, of Dublin, in a cat (1866), and by St Cyr in rabbits and mice ('Ann. de dermatologie et de syphilographie,' 1869), quoted by Dr Fox ('Skin Diseases,' p. 431).

The *treatment* is unsatisfactory. Ordinary parasiticides produce improvement, and if perseveringly employed, apparent cure; but relapse is almost sure to occur. The old French treatment of epilation by a cap of pitch-plaster, applied to the head and then forcibly pulled off, is no more effectual than less barbarous methods; but epilation is probably necessary for even a temporary cure (see Dr Bulkley's paper in the 'Arch. of Derm.,' April, 1881). Several cases of this remarkable disease are described by Dr Fagge in the 'Guy's Reports' for 1870 (p. 354), where more than one apparent cure by epilation is recorded.

TINEA VERSICOLOR.—This affection, described by Willan as *Pityriasis versicolor*, was formerly named *maculæ hepaticæ*, a translation of the vernacular German name *Leberflechte* (liver spots, *chaleur de foie*). Another name still often applied is *chloasma*, but this is better reserved for true maculæ produced by pigment.

In 1846 Eichstädt published in Froriep's 'Journal' the discovery that this affection is due to the presence of a fungus. It is worth noticing, now that its real nature is understood, Bateman's remark, that "the causes of this pityriasis are not well ascertained; fruit, mushrooms, sudden alternations of heat and cold, violent exercise with flannel next to the skin, have been mentioned as probable causes: the most extensive eruption I have seen occurred in a Custom-house officer after drinking spirits freely during a day of fasting on the Thames."

Tinea versicolor occurs as yellowish-brown spots of various shades, scarcely rising above the level of the skin, and yielding a branny or furfuraceous desquamation when scratched. The spots vary from a pin's head to several inches in diameter. As they multiply and coalesce, they form larger patches and thin rings, which, when united, produce the gyrate or serpentine outline before described as the result of this mode of development of an eruption. It is rare, however, to see such perfect rings as in tinea circinata, and the central parts seldom completely recover, and remain more or less discoloured.

The *distribution* of this affection is very characteristic. In the great majority of cases it occupies the chest, it often spreads to the abdomen, and

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is frequently seen on the back, especially between the shoulders. It may overspread the whole trunk, but rarely descends below the waist or ascends above the neck. Occasionally a patch or two may be also found on the border of the axilla and on the soft skin of the inner part of the arm and on the bend of the elbow. Even when the abdomen is not affected it is common to find this form of tinea on the inner side of the thigh, whence in males it is apt to spread to the scrotum. We may say, therefore, that the affection never occurs upon parts which are exposed to the air, and that its favourite seat is on skin which is the most protected and the most constantly warm and moist.

On scraping some of the surface and putting the scales in a drop of potash under a microscope, both spores aud mycelium can be seen without difficulty. The spores of the fungus, *Microsporon furfurans*, are somewhat larger than those of *Trichophyton tonsurans*, and occur in heaps, which are surrounded by mycelium threads.

The presence of the fungus is of course the decisive point of diagnosis; but with a little experience the colour, the branny desquamation, and the locality of this affection are sufficiently characteristic.

Sometimes patients complain of the irritation occasioned by tinea versicolor, and it may be accompanied, especially in hot weather, by slight erythematous dermatitis or urticaria, as the result of scratching. Most frequently, however, it produces no symptoms whatever, and is either discovered accidentally, or is only regarded as a disfigurement. The superficial layers of the epidermis are alone affected by the parasite.

The *cause* of this curious affection, or rather of the fungous growth on which it depends, is quite unknown. It is remarkable that it seldom, if ever, occurs in children, and is rare after middle age. It is most often seen in men between twenty and forty, but may also be observed in women, especially under the fold of the mamma. Although the fungus has been proved by experiment to be capable of transmission by direct inoculation, the disease is not practically contagious, or, if at all, to a very small degree.

Treatment.—If left alone tinea versicolor continues indefinitely, but it may be readily removed by any of the milder parasiticides. After thorough washing with hot soap and water, or, if a rapid cure is desired, with soft soap, the affected parts must be well rubbed with oleate of mercury or unguentum æruginis (℈ij ad ℥j); or, if preferred, sulphurous acid or hyposulphite of soda (ʒj ad ℥j) may be applied in watery solution.

Tinea rosea (?)—*Pityriasis rosea.*—This affection was first described by Gibert as an acute centrifugal erythema of the trunk. Bazin called it *P. rubra maculata et circinata*; Behrend, *Roseola furfuracea herpetiformis.* Vidal regards it as parasitic ('Trans. Intern. Med. Congr.,' vol. iii, p. 133). Most German writers agree in this view, and follow Hebra in classifying it as a variety of ringworm of the body, *Herpes tonsurans maculosus.*

Believing that it is not a form of ringworm of the body, but unable to admit it as a true erythema, as above defined, the present writer has described it for convenience in the chapter on Pityriasis (*supra*, p. 811).

It will be convenient to consider in this chapter the remaining affections of the hair, some of which were formerly confounded with ringworm, and are still liable to be mistaken for it.

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ALOPECIA.*—Baldness, or loss of hair, when not the result of the presence of a fungus, is the immediate consequence of atrophy of the hair-bulbs, which occasions the premature fall of the hairs from the follicles. When this is only partial, and followed by fresh growth of weak hair, the result is thinness or partial baldness; but when the hair-sac is no longer capable of producing a fresh hair complete alopecia results.

Although a senile change, baldness cannot be considered strictly physiological; for it is often absent even to advanced age in men, it is usually absent and rarely complete in women at any age, and it sometimes occurs very early without any other signs of senile decay. In these cases it is frequently hereditary, but by no means constantly so.

The atrophy of the hair-sacs certainly does not depend upon general deficiency of healthy nutrition, nor upon locally-deficient supply of blood. It is not accompanied by anæsthesia, by numbness, or by any other evidence whatever of nervous disorder, so that to ascribe alopecia, whether premature or not, to "vascular" or "neurotrophic disturbance," is an arbitrary hypothesis. It has been asserted that adhesions of the pericranium, and particularly want of mobility of the aponeurosis of the occipito-frontalis, produces alopecia, but many instances disprove the assertion. Neither wearing tight hats, nor going without hats, nor wearing turbans indoors, nor exposure to the sun—nor gout, nor scrofula, nor intemperance, nor abstinence—none of these will in the least explain either senile or premature baldness, for each supposed cause fails on examination.

Alopecia of this quasi-physiological character begins usually in the frontal region, sometimes at the central point at the back of the head from which the hair falls forward, backward, and laterally; and not unfrequently in both regions at once. There is often seborrhœa sicca or a slight degree of pityriasis which precedes and accompanies baldness; but if this is the cause the thinness of hair can be cured by restoring the skin to a healthy condition, and even if neglected it does not go on to complete alopecia. Moreover, in many cases both of senile and premature baldness the skin is healthy throughout. When the hair has fallen off from the mid region of the scalp the process almost always ceases, and that on the temples, behind the ears, and on the occiput persist without change. This ordinary alopecia, moreover, never affects the beard, the eyebrows, or other parts of the body.

Many attempts are from time to time made by physicians, as well as by hair-dressers, to check the loss of hair or to restore it. They consist either in applying stimulating lotions, of which cantharides is usually the basis, or in shampooing and manipulating the scalp. It is very rarely that these attempts have even partial success. A process introduced a few years ago by Dr Pincus, of Berlin, promised better, but had not in the sequel fulfilled the expectations of its author.

Alopecia as the result of febrile diseases.—Although this often proves the first step of ordinary baldness, yet it is distinguished therefrom by its affecting both sexes and all ages, by the fall of hair not being confined to any region of the scalp, and by its thinning rather than completely stripping the surface affected. Moreover, it is not only secondary in origin, but usually passes away of itself after convalescence, instead of being practically incurable, either by nature or by art.

* *Synonym.—Calvities*, or more frequently in classical Latin *calvitium*. *Atrichia* is a modern name. *Alopecia*, ἀλωπηκία, fr. ἀλώπηξ, "quod vulpes hoc malo sæpe corripiatur," is the real Greek name. Celsus distinguishes it by occurring in patches and only in adults from ὄφιασις, which spread in a serpentine form on the back of the head in children.

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Syphilitic baldness agrees in these characters, and its frequency, apart from any other affection of the scalp, as well as its early appearance, likewise point to its ætiology as a febrile alopecia.

AREA.*—A curious and not uncommon form of baldness appears in round, smooth patches on the head, most often in children. The skin is as polished and white as ivory, and the margin is abrupt. There are almost always more patches than one, and as they grow they may unite and form larger areæ. The skin looks thin, and the hair-sacs and sebaceous glands seem atrophied.

The patient is in most cases at or under puberty, but area is also seen in adults, and may then affect the hair of the beard or pubes. The eyelids are sometimes involved with the scalp. The process of atrophy may go on until little hair is left on the head or elsewhere, but it very seldom or never ends in such complete alopecia as will presently be described. So peculiar is the appearance of this disease, that it is less needful to insist upon its distinction from other kinds of alopecia than upon the fact that it is a true alopecia, anatomically identical with the other forms of atrophy of the hair, though differing in its origin and course. It is independent of the presence of a fungus.

Hebra believed at one time in the statement of Gruby that alopecia areata was parasitic, but before long changed his opinion, so that one can only share in the surprise expressed by Dr Kaposi that Hebra is associated with Bazin as a supporter of the parasitic nature of area by his disciple Dr Neumann. It is possible that the single observation of Gruby in 1843 ('Comptes Rendus,' xvii), which gave rise to the question, was made upon a case of true ringworm. Neumann, who has no doubt that area is not parasitic, once, like the writer, found some spores in a case of the disease, but doubts rather the significance of a single observation than the accumulated testimony of his own and others' experience. In fact, M. Bazin's statements (and those made recently by Malassez and by Eichhorst) are the only ones which rest on large experience and assert the presence of a fungus. The French dermatologists call many cases "pelade" or "teigne pelade," which in England or Germany would be regarded as true ringworm in its later stages. In M. Hardy's lectures it is not difficult to recognise in the swelling, irritation, and desquamation of the skin, which he describes in pelade, the characters of ringworm.

Apart from the microscopic evidence, the naked-eye appearance and natural history of the disease would almost disprove the parasitic hypothesis. The hairs around the affected spot are not swollen at the roots, nor brittle in the shaft, but are merely atrophied like normal hairs which are ready to

* *Synonyms.*—Alopecia areata (Sauvages)—Area Celsi—Porrigo decalvans (Willan)—Teigne pelade (Bazin)—Tinea decalvans. Celsus did not particularly describe this variety of baldness, but applied the word "area" ("a bare space," *locus sine ædificio*) to any form of baldness, distinguishing ἀλωπηκία and ὄφιασις as varieties. The "porrigo" of Willan meant any eruption of the scalp, including true ringworm and impetigo or pustular dermatitis, and the term is now almost out of use. The appellation Tinea or Teigne depends upon the erroneous doctrine of the parasitic nature of the disease.

I have many times sought for a fungus and have never found the smallest evidence of its presence, with one single exception. This occurred nearly twenty years ago when working under the late Professor Hebra. In one of his patients suffering from area I discovered some spores and scanty mycelium in one of the neighbouring hairs. I showed it to the professor, and he told me that he had never seen it before. He doubted whether its occurrence was more than accidental, and with my present experience I doubt it also.

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fall out. There is no evidence of local irritation in the hair-sac. The disease, above all, is not contagious, at least as we observe it in England, and it is not curable by antiparasitic treatment.

Dr Thin ('Proc. R. Soc.,' 1881, No. 217) has figured minute schizomycetes, which he calls *Bacterium decalvans*, but which are rounded rather than rod-like, and probably identical with those described by Dr v. Sehlen in 'Virchow's Archiv.' Even if this were of ætiological importance, it would not make area a true tinea.

Another theory is that area is a tropho-neurosis, but of this there is at present no sufficient evidence. The subject was discussed in the International Medical Congress of 1881 by Liveing, Hardy and Vidal, Kaposi, and others (vol. iii, p. 158). There is no anæsthesia of the bald patches to be demonstrated in at least the great majority of cases.

Area is certainly more common in children and young adults than after thirty. Among 112 patients under the writer's care, 43 were children from four to fifteen, 52 were young men or women from sixteen to thirty, and 17 were above thirty, one being forty-seven and one fifty-eight. In the last case area supervened after ordinary senile alopecia had begun to appear, and the two affections were perfectly distinct ('Guy's Hosp. Rep.,' vol. xlvi, p. 373).

Of these 112 cases of area, 72 occurred in male and 40 in female patients.

There were several cases of recurrence of the affection in the same patient, and three of its appearance in two or more children in the same family (see Dr Tyson's case, 'Clin. Trans.,' 1886).

In many cases area would probably pass away of itself, but recovery is often hastened if not brought about by treatment. This consists in local irritants, and, when necessary, internal corroborants. We may begin with a lotion containing ℥ss to ℥ij of acetum cantharidis to a pint of water. This will often cause slight erythema in children, but in adults and in many children we may increase the strength to two, three, or four ounces with advantage, letting the irritation subside whenever it goes beyond redness. A mild and often efficient application is *linimentum myristicæ* (one part of the expressed oil to three of olive oil). With brown hair the *unguentum iodi* of the Pharmocopœia is a useful application.

Area occurs in persons of all degrees of health, complexion, and temperament; but if the patient is pale and thin, steel is certainly useful, and bark or cod-liver oil may be prescribed when indicated by some other symptom than the bald patches.

A second or third attack of area sometimes follows after the first has been completely cured and an interval of time has elapsed.

Universal alopecia.—There are some cases of complete and rapid loss of hair which are neither senile, syphilitic, nor febrile, and which cannot be classed as examples of area. They are distinguished first by the hair falling off almost simultaneously from the whole of the scalp, not gradually from certain regions as in ordinary baldness, nor by the confluence of separate patches as in area; secondly, by the baldness not being confined to the scalp (nor even to the scalp and beard or eyebrows, as is occasionally the case in area), but affecting the whole of the body; thirdly, by its not following an illness.

In one case of this kind the patient was a young man in robust health, and wearing a full beard. Without any assignable cause he lost the whole of the hair of his body in a very short space of time.

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This universal alopecia occurs in both sexes, always beginning in adult life, and usually in young adults. It is quite incurable.

Nine cases have come under the writer's care, the ages being 9, 17 (*bis*), 25 (*bis*), 23, 35, 40, 55 : five were men and four were women. In three of them the baldness had begun some years before, then the hair had more or less grown again, and, lastly, a fresh *defluvium capillorum* had ended in total alopecia.

We may at present distinguish these somewhat rare cases of *alopecia universalis acquisita* from the still rarer cases of *congenital alopecia*. In these the nails, as well as the hair, are affected; and like other deficiencies of development, the condition may be hereditary. Such cases are comparable with congenital ichthyosis, especially in such marked examples as the "porcupine boy;" and still more closely with the "hairy family" of Burma, and the blue and hairless horse exhibited a few years ago in this country.

A striking series of examples of this congenital form of baldness occurred five years ago in this hospital under Dr Fagge. It is remarkable that the development, both of hair and nails, was tardy and imperfect, but not absolutely deficient. The italic letters denote the female sex, as in Mr Galton's nomenclature.

F. Born without hair or nails. Hair began to grow when he was about twenty-three years of age, and at thirty he had a full head of hair. The finger-nails also grew after puberty, but were always ill-formed, and he never had toe-nails. *F.* Normal.

B. 1. Born without nails or hair; the former appeared while teething, the latter when she was ten years old. *n.* Born without hair and nails; none yet grown.

B. 2. Born with hair but without nails; died, aged seven. B. 3. Born without hair or nails; died, aged five months. B. *B.* 4—9. Born with normal hair and nails.

B. 10. Born partly bald with ill-formed nails; he is now twenty-two and has a fair head of hair, but his nails are not good.

The patient herself, then nineteen years old, the eleventh and youngest of this large family, was born without hair or nails. She had, in 1876, only thin lanugo on the scalp and imperfect nails.

TRICHOCLASIA (*Wilson*).—A singular disease of the hair which has been described under this title, and also as *fragilitas crinium* and *Trichorrhexis nodosa* (Kaposi), is characterised by each hair dilating at intervals and breaking at these enlarged points. The dilated node consists of separated cortical fibres which look very much like the splitting and enlargement of a cane when broken across, and the air which enters between the fibres makes them appear white by reflected light. They have thus a superficial resemblance to the ova of pediculi.

It is almost always confined to the beard, and is non-contagious and non-parasitic. It was described by Devergie as "trichoptylose," and subsequently by Beigel and by Wilks. The writer has seen three or four cases of it, one of which he figured in the 'Pathological Transactions' for 1879 (p. 439), where references will be found to the scanty literature of the subject.*

* See also a valuable paper, with fuller references, by Dr T. C. Fox, in the 'Lancet,' Dec. 7th, 1878, and Hans v. Hebra, *loc. cit.*, p. 391.

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This is apparently quite distinct from a parasitic affection of the hair known as "Piedra" from its stony hardness, which occurs in the hair of the scalp, among women only, in Central and South America. It has been described by several French writers and by Mr Malcolm Morris in the same volume of the 'Pathological Transactions' (p. 441).

A third distinct disease or malformation of the hair consists in "beading" like that of *Trichoclasia nodosa;* but the hairs break at the internodes, not at the nodes. Cases have been published by Dr Walter Smith, Dr McCall Anderson, and Dr Payne (see the account and figures of the last writer in his 'Rare Diseases of the Skin').

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CHRONIC DEEP INFLAMMATIONS AND HYPERTROPHIES

"His face is all bubuckles and welks and knobs and flames of fire."
Henry V, iii, 6.

Deep and chronic dermatitis—its definition—its relation to eczema and other forms of superficial dermatitis—to hypertrophy—and to new growths.

GUTTA ROSEA — *Origin in recurrent erythema — development — localities—causes and pathology—relation to dyspepsia—to drink—to ovarian irritation—treatment—Chilblains.*

EPIDERMIC AND PAPILLARY HYPERTROPHIES—*Callosities and corns—Leucoplasia lingualis et buccalis—Warts—Condylomata and mucous patches.*

ICHTHYOSIS — *Anatomy — varieties — xerodermia — Prognosis and treatment—Ichthyosis intra-uterina—Horns.*

SCLERODERMIA — *History — description — distribution — histology — diagnosis—prognosis and treatment—Sclerema neonatorum—Linear atrophy.*

ELEPHANTIASIS—*Nomenclature—anatomy—pathology—relation to chyluria and filaria sanguinis—clinical characters—Dermatolysis.*

XANTHELASMA — *History — course and symptoms — histology — relation to jaundice—Xanthoma diabeticorum.*

As stated in the introductory chapter, the great majority of affections of the skin consist pathologically in superficial inflammation; that is to say, inflammation which affects only the papillary layer of the cutis and the Malpighian layer of the epidermis, with the resulting change in the cuticle. In no form of this superficial dermatitis are the papillæ destroyed; and no scars result. We have now to speak of a far less frequent kind of inflammation of the skin which involves, if it does not originate in, the deep layer of the cutis, which destroys the papillæ, which spreads from the skin proper to the subcutaneous connective or adipose tissue, and which after recovery leaves scars behind. Eczema, psoriasis, and their allies, scabies, the erythematous eruptions, and the parasitic affections—are all, in the sense in which the word is here used, superficial; and however severe and protracted their course, when cured, they leave either no trace behind or only a pigment spot.

It is true that when inflammation occupies the deep sacs of the hairs and the sebaceous glands a cicatrix is not unfrequently the result.

Thus acne in its severe forms leaves scars behind, varying from white spots, very slightly depressed and otherwise inconspicuous, slight local atrophies, up to the hypertrophied scars which sometimes simulate cheloid. The same applies to sycosis, though obvious scarring is less frequent. Some other pustular diseases destroy the papillæ and thus produce scars. This never occurs with true impetigo (which is one of its pathological as well as diagnostic characters), nor with the pustules of scabies or bullæ of pemphigus; but variola, when unmodified by vaccination, almost always leaves indelible traces of its presence—either deep-pitted, depressed, white scars, or more extensive and hypertrophied puckering. The same is true,

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though less constantly, of varicella, and the deeply pitted cicatrix is the well-known mark of successful vaccination. Lastly, the pustules of zona very often (though by no means constantly) leave more or less marked cicatrices; and sometimes, especially upon the forehead, these are deep and indelible.

But beside these deep pustules, we meet with inflammation of the skin which, uniformly and over large surfaces, penetrates below the papillæ and affects the whole thickness of the integument, together with the subcutaneous tissue. Such *deep dermatitis* is usually chronic in course; or, if it shows acute characters, they are repeated again and again, without any tendency for the malady to come to a natural end. Such recurrent subacute diseases become practically chronic, as we see in the case of inflammations of the bronchial tubes, of the eye, and of the colon.

Like other chronic inflammations, those of the skin show in many cases little of the classical signs of the process, and are unattended with fever; moreover, the exudation is never purulent, but if œdematous gradually assumes the characters of *œdema durum*; if congestive, those of hypertrophy. The inflammatory corpuscles, instead of dying and undergoing transformation into pus-cells, become organised into connective-tissue corpuscles, and gradually form fibres. Thus chronic inflammations are closely related to, and often undistinguishable from, *hypertrophy* in the humbler stages of that process, hyperplasia of the connective tissues. An analogy is offered by the case of hypertrophic cirrhosis of the liver.

Again, chronic inflammation is apt to lose the uniform and characteristic qualities which distinguish the catarrhal, adhesive, and suppurative forms of acute inflammation as described in the first volume (p. 51). Thus chronic catarrhal broncho-pneumonia is apt to assume a *caseous* form, and ultimately to lead to the new growths which we call tubercle. Thus chronic inflammation of the urinary tract often ends by becoming caseous. Thus, also, chronic deep dermatitis not infrequently acquires a tubercular character.

Moreover, the continued irritation which gives rise to inflammation and thickening of the mucous membrane of the tongue, the lips, the pylorus, or the rectum may in time, by almost imperceptible stages, pass into a *new growth*, perhaps of the most markedly "heterologous" and malignant kind. Warts and other innocent growths, condylomata and syphilitic nodes also arise from and are complicated with chronic dermatitis and cutaneous hypertrophy.

It is therefore pathologically justifiable to associate with *chronic deep inflammations* of the skin, *hypertrophies*, *tubercle*, and *new growths*; and this arrangement we propose to follow.

The only important instance of *acute deep dermatitis* is that afforded by erysipelas, which has been already treated as a specific disease in the first volume. The deep and acute inflammations which result from burns and other injuries are best studied in surgical text-books.

GUTTA ROSEA.*—This affection in its more obvious forms is well known beyond professional circles. A classical instance of it provoked the well-known descriptions of Bardolph's face, as "the lanthorn in the poop," "an everlasting bonfire light," "sometimes blue and sometimes red."

* *Synonyms.*—Acne rosea—Acne rosacea.—*Fr.* Couperose—Acné congestive (Hardy).—*Germ.* Kupferrose—Erythema angiectaticum (Auspitz).

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Gutta rosea, however, is far from being always the result of intemperance.

Course.—The affection begins with slight erythematous redness, usually of the tip of the nose, occurring after food and combined with local irritation; the heat and itching are felt by the patient, the redness and even slight swelling are visible. It passes off quickly and perhaps may not return for days or weeks, but gradually becomes more frequent, until it is at last habitual.

The next step is for the congested vessels to fail to recover themselves in the intervals between the successive states of hyperæmia. What was a recurrent subacute erythema becomes a chronic congestive dermatitis with exacerbations. Frequently-recurrent œdema has moreover ended in hypertrophy, so that the skin and subcutaneous tissue of the affected part are swollen and thickened. Some of the veins become varicose from habitual distension, and remain visible as red tortuous lines. The sebaceous glands are apt to be obstructed or to inflame without obstruction, and pustules resembling those of inflamed acne result. Hence the common name "acne rosacea." But there are no precedent comedones, and the distribution, ætiology, and entire natural history of the disease are distinct from those of acne.

Hypertrophy may go on until great pendulous masses of thick skin, with the scars of past pustules and abundant fibrous tissue, form hideous excrescences upon the nose, growing either from the tip, from the alæ, or from the septum.

Distribution.—By far the most frequent and conspicuous seat of gutta rosea is the nose, but it is not the only one. In persons in whom this feature is characteristically affected, we usually find large red pimples with inflamed base and chronic course upon the cheeks, the chin, and other parts of the face. When the nose is only slightly affected, and the rest of the face decidedly, the general aspect is very different from that of the hypertrophied form above described when confined to the nose, but the anatomical condition is essentially the same, and every gradation between the two forms may be observed. Beyond the face, similar recurrent erythema, with more or less of hypertrophy, may be seen in the lobes of the ears, although here it is very rare to see pimples or pustules. We never find a corresponding condition of the shoulders or chest, as we do in acne.

Causes and pathology.—Gutta rosea is no less distinctive in its ætiology than in its anatomy and distribution. It is essentially an erythema, or rather the result of frequently-recurring erythema. Like other erythemata, gutta rosea is symptomatic (p. 825); it is never the result of local irritants, it always depends upon reflex inhibition of vaso-motor nerves causing active congestion. We saw that the origin of this reflex action is, in some of the most marked forms of erythema, irritation of the primæ viæ by poisons, drugs, or food (p. 833). Gutta rosea is no exception to this rule; almost always in men, and most frequently in women it is the result of gastric irritation.

Common notoriety affixes the stigma of drink to the possession of a nose like Bardolph's, but it would be no less unjust than uncharitable to assume this as the necessary cause. No doubt the excessive use of alcohol produces most frequently and most readily the gastric irritation which leads to gutta rosea; but marked examples of the disease may be seen in persons of habitual temperance, and even in total abstainers. In women,

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especially at the period of the menopause, there is apt to be a form of dyspepsia which leads to flushings of the face, not only after every meal, but in the worst cases upon putting the first morsel of food into the stomach; these flushings are felt by the patient and cause great distress. The frequently-recurring hyperæmia leads to habitual congestion, pimples, and at last more or less hypertrophy, although in these cases the type is more often that of diffused redness with pimples scattered over the face than of marked local hypertrophy. This, however, is occasionally seen, just as the diffused pimply redness is often the result of tippling.

The only cause for gutta rosea, beside alcoholic or non-alcoholic dyspepsia, is uterine or rather ovarian disturbance. The frequency with which the disease occurs when menstruation is becoming irregular before it finally ceases seems to make this probable. In many cases of gutta rosea the affection is decidedly worse at the menstrual periods, and is associated with dysmenorrhœa. Considering, however, the great number of cases of menstrual disturbance in which no such effect is produced, and the extreme rarity of gutta rosea even in the worst cases of dysmenorrhœa in young women, as well as the frequency of what may be called climacteric dyspepsia, it seems probable that in almost every case gastric irritation is the exciting cause of the disease, and that the monthly exacerbations which undoubtedly occur in certain cases are due rather to direct, physiological, vascular excitation at that time of the whole surface than to morbid irritation of a reflex kind from the ovaries.

Gutta rosea is not produced by the most frequent kind of dyspepsia, that of young adults; it is rare before the age of forty, even in persons who drink freely. It is often combined with acute dyspepsia, and with gout, but is the result of the dyspepsia which, like the gout, is produced by over-feeding and over-drinking, rather than directly connected with excess of urate of soda. Gutta rosea is very far from being confined to the male sex, though the most typical cases from alcohol are of course more frequent in men.

Treatment.—The rational treatment of this, as of every other disease, depends upon recognising its pathology and origin. The first indication is to remove the gastric irritation which is almost always present, to discover if possible its cause, whether in excess of food, in imperfect and hasty mastication, or in some particular article which acts as a poison. Salt meat, spices, pickles, melted fats, and sauces—any of these may prove to be the offender; but most frequent of all are wine, beer, or spirits. If we fail to discover the cause of the dyspepsia we may yet do good, apart from regulating diet and the meals, by the exhibition of small doses of soda with rhubarb and calumba, or when gastralgia is marked, by ten grains of subnitrate of bismuth as a powder either before or after meals, to which five grains of carbonate of soda may be added if there is obvious acidity. Gentle laxatives are often desirable, and occasional doses of blue pill. In some cases euonymin is particularly valuable, taken in doses of two or three grains every other night; in others a dinner-pill of colchicum and nux vomica with extract of aloes is found useful. Locally, astringent washes, like Goulard lotion, are useful and pleasant; flexible collodion may also be painted over the congested parts at bedtime; but this should be done when the patient is in retirement, for the closely-adherent film is unsightly and difficult to remove. In advanced cases, scarification by innumerable punctures with a lancet is sometimes efficacious; and very successful cases

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have been reported by Mr Squire and Dr Stowers. When the hypertrophied masses are considerable they can only be removed by the knife.

*Pernio.**—Chilblains are examples of chronic and rather deep dermatitis with congestion and œdema. They are the result of frequent local erythematous hyperæmia, and are, therefore, pathologically allied to gutta rosea. The stimulus is not directly that of cold on the tissue of the skin, as in frostbite; but vaso-motor paralysis causes vascular dilatation, probably preceded by contraction of the same arterioles. The itching character of the disorder is produced by the secondary hyperæmia.

The fact that chilblains are most common in childhood and youth probably depends on the greater susceptibility of the vaso-motor nervous system at this period of life. Their prevalence in the winter, and particularly in changeable weather, is explained by the same hypothesis of their pathology. Their localisation follows from the greater exposure of the peripheral parts to cold; for, as is well known, they are most common on the toes, next on the fingers, and sometimes affect the ears, or even the chin and nose. Moreover, venous stagnation is most apt to occur in the parts most distant from the heart.

The empirical treatment of chilblains is also rational; preserving the extremities from cold by woollen clothing and exercise, avoiding too rapid warmth, and stimulating the local circulation by friction and rubefacients.

Pathologically similar in the local condition, though different in the cause of the peripheral anæmia, are the cold hands and feet which attend a feeble action of the heart, the "dead fingers" to which many persons are liable on exposure to cold air or water, and the "local asphyxia" which was described as the slighter degree of Raynaud's disease (*supra*, p. 439).

EPIDERMIC HYPERTROPHIES.—It was observed by John Hunter that internal pressure produces atrophy, as when a tumour or aneurysm presses upon a vertebra; but that external pressure produces hypertrophy, as in pressure upon the skin of a labourer's hand. It is better put by Paget, that continuous pressure produces absorption and atrophy, intermittent pressure produces hypertrophy.

When pressure is continuously applied, as to a lady's foot in China while still growing, atrophy takes place with only moderate distortion of the bones, and without thickening of the skin; but when it is applied only while walking, as by the narrow-toed and high-heeled shoes of a European lady, there ensues, along with a certain amount of distortion, hypertrophy or thickening of the prominent parts of the skin. This is usually accompanied with a chronic deep dermatitis, whereby the papillæ are affected, and a new growth forms, or occasionally a deep bursa results. These products, in which chronic inflammation, hypertrophy, and tumour are seen at their point of junction, we know by the names of corn and bunion.

When, without unnatural pressure or distortion, the hand or foot or any other part is exposed to intermittent pressure, the result is something short of this. It is a pure hypertrophy affecting only the epidermis (*callositas tyloma*). Such is the case in the thickened skin of the ball of the foot and the heel in adults, and of the palm of the hand in all who do manual labour. In children the thickening is but very slight, probably an inherited character,

* "Fiunt etiam ex frigore hiberno ulcera, maxime in pueris, et præcipue pedibus digitisque eorum, nonnunquam etiam in manibus, ... dolor autem modicus, prurigo major est."—CELSUS, lib. v, cap. xxviii, § 6.

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since we find it in all plantigrade animals. In adults the degree of it varies with the habits of the individual. This most purely physiological form is seen in those races who go barefoot, for wherever shoes are worn there is a chance of corns appearing even on the sole of the foot. Precisely similar callosities appear in the middle of the palm in workmen who use screwdrivers, gimlets, and augurs, in the cleft between the finger and the thumb in shoemakers, and others who habitually pass a strap or cord in this position, over the patella in those who frequently kneel, and on the back of the neck, especially over the seventh vertebra, in those who carry burdens on their shoulders, as may be often observed in railway porters.

In such a callosity it will be found on section that the horny layer or cuticle is enormously increased, the Malpighian layer slightly, if at all, and the cutis vera quite unaffected. Hence these callosities appear lighter than the rest of the skin in negroes.

*Tylosis** was the name given by Hebra to great epidermic thickening, without the inflammation described above at pp. 768, 783. It usually affects the palms and soles. A marked case is described and figured by Dr Crocker in the 'British Journal of Dermatology' (vol. iii, p. 109). This was an instance of tylosis following over-secretion of sweat, but more often the condition is a congenital exaggeration of the natural thickness of the palmar and plantar epidermis. In a case reported in the same article, blisters formed every autumn, perhaps from retained sweat; and the patient could trace the same malformation in his mother and grandfather, in three of his own children, and in a grandchild.

The corn (*clavus*), as was first shown by the anatomical researches of Gustav Simon, consists of a diseased growth of the horny cuticle into the subjacent living Malpighian and papillary layers. The horny downgrowth is of a more or less conical shape, and causes atrophy of the immediately adjacent papillæ, but at the same time a thickened layer of cutis forms around by true chronic inflammation. Here the cuticle is but slightly thickened, and not hard, as in the central part, and the papillæ become gradually hypertrophied. Occasionally the original central hardening appears never to take place, especially in the soft parts of the skin between the toes, which are continually in contact and moistened with perspiration. The result is what is known as a "soft corn." There may be a mere horny plug pressing on the skin beneath, without exciting inflammation around, as occurs most frequently on the naturally thickened skin of the ball of the great toe or heel. In this case the resulting pain is that of an occasional sharp prick, when the sharp, hard, horny plug is suddenly driven home by accidental pressure, and is very different from the continual, wearing, and disabling pain of a clavus mollis. The commonest kind of corn, partaking of both characters, combines the discomfort of each.

When a cyst or bursa forms beneath the corn and increases so as to become obvious it is called a *bunion*. A small cyst is often to be found beneath an ordinary corn of old standing and large dimensions, but the large cysts seldom form except over the metatarso-phalangeal joint of the great toe, when this has been rendered artificially prominent by the distortion

* Tylosis palmæ manus plana (Hebra); Keratosis manuum aut pedum, Ichthyosis palmaris (Auspitz). Τύλωμα and τύλωσις are both genuine Greek derivatives of τύλος or τύλη, from τυλόω, to make callous. So Theocritus, μακέλᾳ τετυλωμένος ἔνδοθι χεῖρας, *i. e.* hands made hard on the palms by using a pickaxe.

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of short, narrow-toed, and high-heeled shoes. The bursa from time to time inflames, and the tension then occasions severe pain, although suppuration is rare.

Treatment.—The proper treatment of corns is prevention. Children's shoes should be made low in the heel, broad in the tread, straight on the inner side, and each shoe markedly unsymmetrical. In measuring for shoes, or for making a last, one should not sit but stand, so that the weight of the body may expand the foot into the natural shape and size which it then assumes. In a perfect covering for the foot similar expansion is afforded by the elasticity of the upper leather, and the yielding of a thick and soft stocking, but the sole should slightly project, so as to equal the largest length and width of the foot. Even in adult life the trouble of insisting upon boots being properly made is well repaid by the increased comfort and ability to walk, and the disappearance of acquired corns and distortions. In bad cases it is well for the patient to wear stockings with divided toes like a glove, and to have a stout vertical piece of leather fixed so as to separate the great toe from the rest, and to press it outwards into its natural position, a plan devised by the late Aston Key.

Besides removing the thickened epidermis and extracting the conical plug of hard keratin from time to time, relief may be obtained by treatment with salicylic acid, 2 per cent., mixed either with mutton suet (the ointment in use in the German army), or in the stronger proportion of five or ten grains to the ounce of vaseline.

In many cases, however, beside the presence of both hard and soft corns, the whole foot is tender and painful. The remedy then consists, first, in large and low shoes, so as to diminish the heat and moisture; secondly, in thick and loosely knitted stockings, which are at once absorbent and pervious; and, thirdly, in soaking the foot night and morning in alum lotions, or brine, or solution of tannin. Thread and cotton coverings for the foot should never be worn. When wool or merino-mixture cannot be borne, silk is the only proper substitute.

Until comparatively lately the shoes supplied to the English army were symmetrical, that is to say, there was no difference between right and left. This is now happily corrected, owing to the efforts of Dr Parkes and other medical reformers; at present our soldiers are probably better shod than the French with their shoes and gaiters, or the Prussians with their high boots. The best foot covering of all is, perhaps, a kind of sandal worn by the Spanish infantry. In the handsome and serviceable costume of the Hungarian army, an excellent laced boot is worn, much like that of our own troops, but somewhat higher, like a shooting-boot, without the addition of a leather legging. The importance of anatomical knowledge in army clothing is conspicuous in this instance; but in civil life, apart from artistic considerations, the misery and ill-temper produced by ill-fitting shoes render the subject one of serious importance.

Tylosis is best treated by soaking the parts in hot water, with the use of alkaline soap. Or the hardened skin may be covered with salicylic acid as an ointment or plaister. The best method of applying this valuable keratolytic agent is probably that introduced by Dr Unna, of Hamburg, as a plaister made by saturating an alcoholic solution of gutta percha with salicylic acid (see Dr Thin's paper, with four cases so treated, 'Clin. Trans.,' xvii, 9). But the writer has seen excellent results from an ointment composed of ac. salic. ʒss to an ounce of vaseline or lanoline.

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*Leucoplacia buccalis.**—Closely allied anatomically to corns and callosities, consisting, like them, in hypertrophy of epithelium, are the milk-white patches or corns upon the prominent parts of the heart, both auricle and ventricle, and the thick gristle-like white fibrous patches on the surface of the spleen, with similar conditions less frequently met with in the pleura and the peritoneum.

Still more closely connected with corns are the white patches upon the mucous membrane of the tongue and inner lining of the cheeks. These patches have been erroneously described as "psoriasis of the tongue" and "ichthyosis." They are of much diagnostic interest. They sometimes occur as the result of irritation from a rough tooth. They also are produced, or at least aggravated, by smoking, not by the chemical action of nicotin, but by the heat of a cigar or pipe or by the friction. Very similar patches may be the result of syphilis, but these may generally be recognised by their being unsymmetrical, and not confined to the mucous membrane but dipping beneath it; moreover, in most cases there is either an ulcer on the patch, or more or less contraction around it from previous loss of substance. The diagnosis from syphilis, however, is sometimes difficult. Such patches of leucoma are not unfrequently met with in cases of lichen planus (p. 799).

These white patches may be the seat of subsequent cancer: they are not its first stage, for they may last many years before malignant action appears, but they are the seat of irritative proliferation of cells, which only needs a determining condition, whatever it be, to produce carcinoma.†

The treatment depends, first, on removing any source of irritation, as rough teeth, smoking, or taking pepper, hot soups, and, perhaps, carbonic acid in water; secondly, in applying such local remedies as borax and honey. Balsam of Peru has been lately recommended by Dr Rosenberg ('Therapeutische Monatsheft,' 1888, No. 10), applied two or three times a day.

WARTS.—*Verrucæ—papillomata.*—These are small cutaneous tumours consisting in overgrowth of the papillæ of the cutis.

A vertical section shows that the horny layer of epidermis is unaffected or is somewhat thinner than usual. The Malpighian layer is sometimes slightly thickened, and in many cases is the seat of more abundant pigment than usual. There is seldom or never any evidence of inflammation, the process is one of hypertrophy and new growth.

Warts are very rarely painful, but their removal is desired from their unsightliness and also because of their inconvenience, or sometimes the pain occasioned when they are accidentally pressed upon. They are sometimes single, more often multiple, and in rare instances occur in innumerable multitudes. They appear to be never congenital, but are most common in children, and are comparatively rare after early adult life. We can sometimes trace their origin to certain definite sources, usually some form of local irritation.

The most common seat of warts is on the hands, not the palm, but the fingers, the dorsum, and the wrist. They may also occur on the arms, the face, not unfrequently on the scalp, and more rarely on the trunk or lower

* *Synonyms.*—Ichthyosis linguæ—Psoriasis linguæ—Leucoma—Tyloma—Keratosis linguæ et oris.

† See the discussion on this affection at the International Medical Congress, 1881 (vol. iii, p. 171), introduced by Dr Schwimmer, of Buda-Pesth.

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extremities. They are decidedly rare on the feet, but are not uncommon on the penis and vulva, around the anus, and at the orifice of the lips and on the mucous membrane of the mouth. A similar condition occurs also in the œsophagus, especially in certain cases where pressure has produced irritation of the mucous membrane, and also where chronic cardiac disease has led to its habitual congestion. Warts are usually of a rounded, hemispherical, or pointed shape, but sometimes are flat at the surface; and by growth or coalescence a large flat warty mass may be formed, which is called a condyloma.

Pathologically we may recognise the following varieties of papillomata:

1. The innocent and painless warts of youth, easily removed, and not recurrent. They are almost always found upon the hands. When one of them appears, others quickly follow, and their prevalence among children of the same age has led to the popular belief that they are contagious. Dr Payne has lately brought forward a case in which he was himself the recipient of the contagion ('Brit. Jour. Derm.,' vol. iii, p. 185). Probably it is bacterial, but none has yet been identified.

These multiple warts disappear as readily as they come, with the help of a charm or without. They may, however, be removed by salicylic acid dissolved in collodion or gutta percha, or by frequent applications of nitric or strong acetic acid.

2. Small multiple warts, usually flatter, and of a pinkish colour, thus differing from the yellowish tint of those first described. Beside their small size and colour they differ in their often occurring in large numbers so as to simulate papular dermatitis, or, again, if rather large, discrete, and somewhat flat they may simulate molluscum, a variety of which has been named "verrucosum" from this resemblance. These multiple warts are usually seen covering the arms, but may also be met with on the neck, face, and forehead. In one case, in a girl of eighteen, they covered the back of both hands, in another, of a healthy woman of twenty-eight, they closely resembled lichen planus. In a third, a young man of twenty, they occupied the neck and left side of the nose, where we counted more than three dozen. He also had warts, though less numerous, on both hands and forearms. They all came in six months, starting with one large one on the pomum Adami. This patient had had warts on his thumbs when a boy.

3. Warts of old age (*Verruca senilis pigmentosa*), usually few in number, large and deeply pigmented. They are apt to occur around the orifices of the body, on the eyelids, the lips, the genitals, and around the anus. Similar papillomata occur on the tongue and mucous membrane of the mouth. They are very liable to degeneration, and often become the seat of epithelial cancer, after having existed for months or years without showing the slightest malignancy. The abdomen of an old woman under the writer's care suffering from internal cancer was covered with these pigmented warts, but they were themselves non-cancerous (Mary, March, 1886).*

4. Warts following gonorrhœa: multiple and confined to the glans and skin of the penis.

Condylomata and mucous patches.—The composite warts, known as condylomata, hard condylomata, Spitzcondylom (*C. acutum*), are true papillomata in structure, but are always local, never scattered about as other warts are. They occur most frequently about the anus and genital organs. They

* A singular case of multiple pigmented warts occupying one half of the body in a girl of seventeen came under the writer's notice in 1883 and 1887, and is briefly recorded in the 'Guy's Hosp. Rep.,' vol. xlvi, p. 408.

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are certainly not always syphilitic. They may follow the irritation caused by the discharge of a soft chancre or a gonorrhœa, in the latter case being identical in all but size with gonorrhœal warts. They may also occur in the cleft of the nates as the result of friction from riding, when there is not the least probability of other than mechanical origin.

The latter variety only need dryness and protection, with the help of citrine or some other mild mercurial ointment. The harder venereal warts and condylomata must be removed by curved scissors, or with nitric acid, arsenical paste, or some other caustic application.

On the other hand, the soft condylomata (*plaques muqueuses* or mucous patches) are believed to be always syphilitic, and they almost alone of secondary lesions have the power of transmitting the virus. They occur on the lips, on the mucous membrane of the tongue, cheeks, palate, and tonsils, occasionally on the eyelid, sometimes on the female mamma, and frequently around the anus and vulva. Here they may grow to great hypertrophic masses, the tertiary *syphilis vegetans* of authors. They are best treated by dusting with calomel, and occasional application of nitrate of silver.

ICHTHYOSIS.—This is a very remarkable and in its fully developed form a rare affection. It is an example rather of hypertrophy and malformation than of chronic inflammation. It was classed by Willan among his squamæ, and its name, "the fish-skin disease," was given for the same reason. The scales of ichthyosis are, however, very different, from the branny desquamation which follows all superficial dermatitis, from the large pearly coherent scales of psoriasis, and from the thin squames of pityriasis rubra. In the most marked cases the surface of the skin rather resembles the rough, dark, and scaly surface of the bark of a tree, or it may be compared to the rugged hide of an elephant. Sometimes the roughness and horny excrescences are so marked that they rather resemble the prickly skin of certain sharks; indeed persons affected with an extreme degree of ichthyosis have been exhibited as "porcupine men" (*Ichthyosis hystrix*).

The malformation is congenital, but does not appear until infancy is past, although the mother will generally admit that the infant's skin was from the first more rough, dry, hard, and shining than that of other children.

Ichthyosis is most marked upon the limbs, but its characteristic feature is that it is practically universal. In a fully developed case no portion of the whole body is absolutely healthy. The parts least affected are the scalp, face, palms, soles, genital organs, and the flexures of the joints; in other words, the thinnest portions of the skin. The greatest accumulation of horny epidermis is on the outer side of the arms and legs, and especially about the elbows and knees, but the back, the nates, and the whole of the trunk are often scarcely less affected. The scales are not large and are more adherent than those of psoriasis, so that, considering the thickness and extent of the mass, there is less free desquamation than might be expected. The surface is dry as well as rough; there is almost complete absence of perspiration; the sebaceous glands, instead of their natural lubricating oil, secrete a thick material (seborrhœa sicca) which helps to form the bulk of the crusts, and gives them more power of attracting and retaining dirt. The difficulty of keeping the rough scaly skin clean is extreme, so that children affected with it have a dingy appearance, which in some cases and in the worst parts becomes almost black. The name "ichthyosis nigra" has been very unnecessarily applied to this condition. There is no deposition

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of pigment, and mere friction will sometimes rub off the superficial dirty scales from the most exposed parts, and leave a grey abraded surface which is characteristic and hideous enough. The thick dry skin is apt to crack in a somewhat regular square fashion like the skin of certain kinds of armadillo, and these cracks may penetrate to the cutis and become painful bleeding rhagades.

Except for this accident, ichthyosis is completely painless and apparently does not affect the general health. One or two children under the writer's care affected with it have been remarkably plump, rosy, and in other respects well developed and healthy.

As above remarked, ichthyosis is a congenital disease or rather malformation, and it is not unfrequently seen in families, as in the famous case of John and Richard Lambert, two brothers who were exhibited as the porcupine-men, and whose father had a similar state of skin.

Among 15 patients reported by the writer in the 'Guy's Hospital Reports' (vol. xlvi, p. 389) eleven were under 18 and the rest aged between 21 and 30. One was a case of ichthyosis or tylosis of the palms, and was present in a brother and sister of the patient although not in his parents.

Histology.—On vertical section the diseased masses are seen to consist of beautifully arranged wavy layers of horny scales exactly like those of the thicker parts of the cuticle. A section of the skin shows that the Malpighian layer of epidermis is proportionately small, and that the ridge-and-furrow cells ("prickle cells") have more or less completely disappeared; in other words, the keratinous transformation of epithelium is here more rapid than usual. The cutis is completely unaffected. Contrary to the statements of earlier writers, the independent observations of Fagge and of Esoff first showed that although the papillæ are often elongated, this is a secondary change,—that they are really atrophied and not hypertrophied. The sweat-glands have disappeared or only exist as cysts, and the sebaceous glands are smaller and less numerous than usual. The hair-sacs are thickened by overgrowth of epidermis, and the hairs are atrophied, tufted at the root, and easily shed.

If the above account of ichthyosis be correct, there is no need for the distinction which Erasmus Wilson attempted to make between true and false ichthyosis. His "false ichthyosis," the *ichthyosis sebacea* of other authors, is seborrhœa sicca corporis. In true ichthyosis there is no doubt a certain amount of sebum, which is mixed with the epidermic masses, and can be extracted by ether in the form of stearin and cholesterin; but this is not the essential part of the disease.

Nor is there any need to continue the distinctions of Devergie and other writers into *Ichthyosis alba*, *I. brunnea vel nigra*, and *I. hystrix*, or Alibert's of "Ichthyose nacrée and I. cornée."

In some cases of ichthyosis hystrix there are papillomata mingled with the epidermic lesions, as in a boy in Philip Ward in 1887, where the horny warts were arranged in long stripes down his arms and legs. The scalp, the palms, and the soles were alone free.

Xerodermia.—We must recognise as true ichthyosis, though of a much milder form, that affection of the skin which was named by Wilson "xeroderma."* This dryness of the skin is accompanied by roughness,

* In this, as in other similar compounds, the name of the disease, the condition of the derma, should be spelt with *i*. So sclerodermia, pachydermia, &c., words analogous in formation to anæmia and anuria.

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to be felt rather than seen, which chiefly affects the outside of the arms and legs. There is but little desquamation, and the morbid change is so slight that it is difficult to believe it can be essentially the same as that which produced the porcupine-men. But of this there is no doubt, for we meet with every gradation between the two conditions. On the one hand, such an extreme degree of the affection as the *ichthyosis hystrix* of Tilesius is extremely rare; and, on the other, even the slighter forms of xerodermia are more extensive, obstinate, and clinically important than they at first sight appear. At the same time we may admit two groups of the affection, the more severe, which corresponds with the classical description of ichthyosis, and the milder forms, for which the term xerodermia might be used, if it had not since been unluckily conveyed to a totally different and malignant form of cutaneous disease (*v. infra*, p. 929). Each case has its own characters from an early period, and when once established in the second or third year of life, does not usually become much worse. Both alike are congenital malformations, both have the same distribution and probably the same histology.

The chief importance of this remarkable disease, even in its mildest form, and quite apart from the hideous deformity of the worst kinds, is that the dry, harsh, unlubricated skin is extremely disposed to superficial dermatitis; or, as it is usually put, ichthyosis and xerodermia are often complicated by eczema.

Treatment.—The first indication is to cure the inflamed, red or weeping patches, and the deep painful fissures by the same methods which have been above described for the treatment of eczema rubrum, madidans, and rimosum. The second indication is to supply the deficient natural lubricant of the skin by oils or ointment; suppleness is thus restored, the characteristic dryness is removed, and the liability to dermatitis reduced to normal limits. In the more severe cases, however, it is necessary, before this can be done, to remove the products of disease; and for this purpose warm baths, alkaline baths, friction with soap and water, and above all with soft soap, are the measures which are necessary. The only caution is not to be too vigorous in softening and removing the diseased epidermis, until local inflammation has been relieved. From time to time the process of cure may have to be interrupted, and the tender skin soothed by zinc or lead ointments or olive oil. Dr Fagge, as also Dr Liveing, recommend glycerine of starch, but often oil is more soothing than glycerine in any form.

It is astonishing what excellent results may be obtained, even in the worst cases of ichthyosis, when treated with perseverance and with an intelligent appreciation of the object in view. Within a few weeks children, whose portraits would almost go side by side with that of the porcupine-men, present an appearance which it requires the scrutiny of an experienced eye to recognise as more than "a little roughness of the skin."

Salicylic acid may be employed in obstinate cases, and "ichthyol" (a mineral oil obtained from rocks rich in animal deposits) has been strongly recommended for this, as for most other cutaneous diseases.

The disease, however, is relieved, not cured. As soon as the patient is neglected it returns as before, and he can only maintain his skin in bearable condition by constant attention to cleanliness, by frequent warm baths, and continual inunction. Dr Fagge recommended antimonial wine ('Guy's Hosp. Rep.,' 1870), and many physicians administer cod-liver oil.

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The term *ichthyosis congenita* has been applied to a rare and remarkable form of disease described by Lebert, in 1864, as *keratosis diffusa intra-uterina*. It affects the whole of the skin with thickening of the epidermis, which is too small for the body, so that the child is literally hide-bound. Numerous and deep fissures result, and the appearance which ensues has been described as the "harlequin fœtus."

The horny layer is greatly thickened, the papillæ and the rest of the cutis unaffected, the sebaceous glands are atrophied, and the ducts of the sweat-glands enormously stretched.

Cases of this curious and very rare affection have been described by several authors. The best account of it is that given by Hans von Hebra in his 'Krankhaften Veränderungen der Haut,' p. 348. Mr J. B. Sutton believes that it consists essentially in a perverted secretion of the vernix caseosa. He has figured a case in a fœtal calf in the forty-second volume of the 'Pathological Transactions.'

CORNU CUTANEUM (*ichthyosis cornea* of Willan and Bateman*) is the name applied to those remarkable cases of horny growth which have been figured as "freaks of nature." They are occasionally seen in old women, less often in old men, and very rarely indeed in early life.

Lebert collected 109 cases. Most often they spring from a sebaceous cyst. They may occur anywhere, often on the lip or the glans penis, and sometimes are followed by cancer.

The growth can always be readily removed, and shows no tendency to return; although, as Bateman remarks, if merely sawn or broken off, they invariably sprout again, like hair or nails.

Two remarkable cases of cutaneous horns, one on the neck and the other on the hand, were modelled by the late Mr Towne for the Guy's Hospital Museum (Nos. 333 to 339).

SCLERODERMIA.†—This is a rare but interesting disease of the skin. Pathologically, it is a chronic, deep, indurating dermatitis, followed by atrophy, and often ending in complete involution.

One of the best contributions of the lamented author of the present work to dermatology was his masterly account of this disease in the 'Guy's Hospital Reports' for 1867, in which he conclusively proved the essential identity of the diffused sclerodermia of authors with the circumscribed sclerodermia which was also known as *Addison's keloid*, and is synonymous with many cases described as *morphœa* by older writers. See also his second paper (ibid., vol. xv, p. 297).

Course.—The disease begins very gradually in a hardening of the deeper layers of the skin. The epidermis is unaffected, the surface smooth, not elevated, and the colour is unaltered; but the patient finds that the affected spot is stiff, and on feeling it a more or less marked induration is recog-

* Bateman objected to calling them horns on the ground that they have no connection with the bones or other part beneath, and are of purely cuticular growth. But this is the only ground on which we call them true horns and not exostoses or antlers. What he meant was that they have no bony core as the horns of ruminants; but they are exactly identical in structure with that of the rhinoceros.

† *Synonyms.*—Scleriasis cutanea—Sclerème des adults (Thirial)—including "Addison's keloid" and Morphœa. Vitiligo (in part), *Cutis tensa.* Morphœa, or Morphea, or Morfea, a low Latin word of uncertain derivation, corrupted into *morphew*, was very widely applied in popular usage, but in Holland's translation of Pliny is used as a translation of *vitiligo*.

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nised, the skin cannot be pinched up into folds as in health, and instead of the natural elastic softness of the integument a characteristic hardness appears.

In the circumscribed form, the edges are well defined, so that it feels as if a disc of hard, smooth leather were let into the skin. In the diffused form the stiffness and induration become gradually less and less, until they are lost in the natural softness of the skin; but even then one may generally find some directions in which the sclerosed patch has a more definite edge. Sooner or later the local appearances become more marked; the affected skin becomes white, or assumes a sallow, yellow tint, or becomes pigmented with a pale yellowish brown, which is usually most marked towards the borders, and is never uniformly diffused over the entire patch. In the early stage a slight rosy circle may be observed around the patch, occasionally forming a distinct ring in the circumscribed form, or a more ill-defined and irregular blush in the diffused form (*scleriasis*). The smooth white patch, with its colour heightened by the pink margin, has been often compared to an ivory disc. There is no œdema of the integument at any period, and this alone suffices to distinguish sclerodermia from pachydermia (elephantiasis) with which Rasmussen would associate it.

A patch of morphœa may go on increasing until a disc several inches in diameter is formed, or it may lose its distinctive characters and pass into the diffused variety.

Diffused scleriasis usually has its own characters from the beginning, and slowly extends, with no definite margin, until it involves a considerable part of one limb, or one side of the neck, or half the trunk. The surface is then as hard as a board, and as unyielding to the touch. After a time contraction begins to appear, and scar-like bands vary the surface of the disease. This, together with increasing pigmentation, gives some resemblance to the contracted cicatrices from a scald or burn, and explains Addison's application of the term keloid to the affection.

Locality.—A patch of morphœa most often develops on the trunk, particularly on the skin of the female mamma, where such parchment-like *plaques*, or ivory indurations, like the skin frozen by an ether spray, have been sometimes called "vitiligo."

Diffused sclerodermia may be seen on the scalp, the forehead, the chin, or other parts of the face; and the expressionless mask-like aspect it gives to the features is very striking, particularly since the immobility is not uniform, but affects one side or certain features only. Scleriasis is also frequent in the arms, hands, and fingers, which become contracted and useless, and on the side of the neck, where a distortion may be produced, which resembles torticollis; or it may invade extensive regions of the trunk or lower extremities. In the well-marked case of a young and healthy soldier, reported by Dr Curran ('Edin. Med. Journ.,' 1871), the disease covered the whole surface of the body. It has been unsymmetrical in all the cases seen by the present writer, and in the numerous drawings, as well as models, in the museum of Guy's Hospital. But in a case described by Dr Van Harlingen in a negro (1873) the disease was symmetrical, and he regards this as the rule. Occasionally, but only as an exception, it may be traced in the course of a cutaneous nerve.

Cases have been described in which it or a similar affection involves the mucous membrane of the mouth.

Symptoms.—Sometimes patients complain of considerable pain as well as

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stiffness in the affected parts, but this is often completely absent. There is little or no itching, and, as a rule, no accompanying inflammation or pyrexia. Sometimes, however, deep and very intractable circumscribed ulcers form on the sclerotic patches, as in a remarkable case brought by Mr Morrant Baker before the Pathological Society (vol. xxxii, p. 261). There is no hyperæsthesia, nor true anæsthesia—at least, as a rule; but patients may complain that they do not feel as distinctly as on the normal skin. The hide-bound state of the affected parts makes them almost immoveable.

Histology.—There is scanty evidence of a true inflammatory process in this singular disease, nor does there seem to be anything which can fairly be called a new growth; the epidermis is unaffected, the papillæ atrophied only in the later stages of the affection, the hair-sacs, sebaceous and sweat glands normal, as also are the unstriped muscles of the cutis. The seat is primarily in the deeper layer of the cutis and the subcutaneous tissue. Here the fibrous bundles become thicker and the fat between the meshes is absorbed, while increased pigment is gradually deposited both in the papillæ and in the cutis. No cell proliferation is to be seen, according to the careful observations of Chiari in the 'Vierteljahresschrift f. Derm. u. Syph.,' 1868. This process of mingled hypertrophy and atrophy leads to the characteristic results, both of the earlier and later stages of the disease. In the earlier stage, by compressing the blood-vessels the peculiar pallor is produced; and by the increase of fibrous tissue and disappearance of fat the scleriasis of the later stages. Beside the pigmentation the affected parts sink below the level of the healthy skin, instead of being, as at first, on the same level, and the contraction leads to the cicatrix-like bands which crumple the fingers or deform the face or breast.*

Ætiology.—The true cause of sclerodermia is quite unknown. It is more common in women than in men. Putting together 22 cases reported from Guy's Hospital (seven by Addison, three by Fagge, and twelve by the present writer: 'Reports' for 1867, 1870, and 1889), nineteen of the patients were women and only three men. In 40 cases collected by Rasmussen the numbers were thirty women to ten men. It has been observed at all ages, including children under six and adults up to seventy. Among our twenty-two patients, ten were between eight and twenty, six between twenty-five and forty, and six between fifty-five and sixty-four.

Prognosis.—Dr Fagge made the remarkable discovery that sclerodermia, both in its circumscribed and diffused forms, is liable to spontaneous involution. He tracked one of the most marked cases described by Addison, and found that the patient's skin had recovered its normal condition. The same thing has been repeatedly observed since, although it is too much to say that complete recovery is an invariable or even a frequent result. The disease, at all events, shows no tendency to develop into any active or malignant form, and beyond the disfigurement and disablement due to contractions and the occasional pain, the most serious result is the rare one of ulceration as above noticed.

No efficient *treatment* has been devised. Emollient oils, warm douches, and manipulation have been tried with some apparent benefit. Electricity

* See a valuable paper on "Sclerodermia" by Dr Rasmussen, of Copenhagen ('Edin. Med. Journ.,' Sept., 1867); and another by Dr Van Harlingen, of Philadelphia, with a full list of references ('American Journ. of Syphilis and Dermatology,' October, 1873). See also an account of the "Histology of a Morphæa Patch," by Dr Crocker, 'Path. Trans.,' 1880.

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has also been employed, either in the form of continuous galvanism to the affected patches, or by interrupted galvanism to the neck in the somewhat vague hope of stimulating the cervical sympathetic, and the equally vague expectation that occasional stimulation of the cervical sympathetic would have any effect upon the disease.

Sclerema neonatorum.—This affection, sometimes called Thirial's disease, is best named as he called it, *sclerème*, in distinction from the sclerodermia or scleriasis just described. It is the condition which is known as "hide-bound" in new-born children, affecting the whole of the surface, and characterised, not only by hardness, want of elasticity, and pallor, but also by œdema. The temperature is lowered and the child generally dies within a fortnight.

Linear atrophy.—Somewhat resembling sclerodermia in appearance, and perhaps also in pathology, is a curious affection of the skin, which takes the form of long streaks, generally broader in the middle than at the ends, or less frequently of round, more or less regular patches: in both cases it appears like a scar, for there is loss of pigment and atrophy of the cutis vera. It was first described by Dr Wilks ('Guy's Hosp. Reports,' 3rd series, vol. vii, p. 298) as an idiopathic affection which exactly resembles the cicatricial marks caused by overstretching of the skin and rupture of its deeper layers—well known under the name of lineæ gravidarum as a result of abdominal distension from pregnancy, but also seen in ascites or whenever the abdominal skin is similarly stretched, and over joints which have enlarged and stretched the skin. The spots are palpably depressed below the level of the healthy surface, and on a microscopical section, which was carried out by Kaposi at Vienna, the papillæ were found atrophied or vanished, the epidermis in both its layers thinned, and the subcutaneous tissue and glands atrophied. This curious affection, which may be idiopathic, has been seen upon the hips, the leg, the knee, the ankle, and the hand. In the early stage the marks are somewhat pink, but there are no signs of inflammation, no pain, or any other symptoms. In a case described by Dr Liveing the maculæ were at first slightly red and raised above the skin; they occupied the upper part of the sternum and neck, and after passing into the atrophic stage above described, ultimately underwent gradual involution.

In a girl who lay ill with renal dropsy in Miriam Ward in 1886, there was during her illness and after recovery a remarkable zebra-like development of atrophic stripes on the forearms, loins and hips. A still more extensive case of the same kind has since occurred in a youth of nineteen, who recovered after more than a year's illness from tubal nephritis with extreme anasarca and ascites.

There are some good models of these striæ atrophicæ in the Guy's Hospital Museum, Nos. 340—347.

ELEPHANTIASIS.*—This, among many other names, has been given to a curious form of chronic inflammation with hypertrophy of the skin, chiefly met with in tropical climates. It is not necessary to enter upon the tangled labyrinth in which this, like so many other names of cutaneous diseases, is involved. It will suffice to say that the word elephantiasis was used by Aretæus and by Celsus for the very different disease known to the

* *Synonyms.*—Elephantiasis Arabum or Elephas—Bucnemia or Bouknemia—Arabic, Dal fyl—Pachydermia—Barbadoes leg.

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Greeks as lepra, and to English readers as leprosy. They used it because of the magnitude and monstrosity of the disease (cf. *infra*, p. 918, *note*). Unfortunately the comparison was supposed to be between the appearance of the disease and that of an elephant's hide, and since the legs affected with pachydermia have some resemblance to the thick and shapeless limbs of an elephant, the two diseases and their names were long hopelessly confused.

The most important pathological fact about elephantiasis, using the term as applied by the Arabian translators of the Greek authors, is that it is hypertrophy dependent upon recurrent deep dermatitis, which we may compare to that of gutta rosea and of pernio. All observers in countries where the disease is endemic agree that it begins and is accompanied by recurrent attacks of what has been called erysipelas, each attack leaving the tissues more thickened and infiltrated. Inflammatory œdema of the skin and subcutaneous tissue is the characteristic lesion. This gradually becomes œdema durum and no longer yields to pressure, the infiltrated tissues undergo hypertrophy, and masses of fibrous tissue are thus produced, which may be described as a diffused new growth. The skin itself appears at first to be unaffected, at least in its papillary and epidermic layers; but after a time it also hypertrophies, the papillæ becoming enlarged and the surface coarse, thick, scaly, and pigmented.

Histology.—On section, the hypertrophy of the deep layer of the cutis, and the massive fibrous bands of white and elastic tissue, with œdematous connective and adipose tissue, are very characteristic; the lymph-spaces of the cutis are enlarged, and the lymphatic vessels are frequently found dilated and varicose. Occasionally an ulcer will accidentally open one of these enlarged lymphatics; and a discharge of normal lymph, more or less milky if it has passed through several lymph-glands, is poured out.

The disease does not spread to the deeper fasciæ or bones, and it never affects internal organs or leads to any but local results.

Such a condition is occasionally seen as the result of long-continued inflammatory dropsy of one limb. An example in a case of old dermatitis of originally syphilitic origin is figured in the 'Guy's Hospital Reports' for 1877, pl. ii. A similar result may also be seen in cases of enormous obesity and general hypertrophy of fat and subcutaneous tissues. Or, again, it may be the result of local pressure upon the veins and lymphatics, as by enlarged inguinal glands or other tumours. But in many hot countries, particularly the West India Islands, in Cape Colony, Egypt, South America, in China and Japan, and in the Pacific Islands—elephantiasis is idiopathic and endemic. Dr Turner, of Samoa, has made numerous photographs of this disease, which presents exactly the same features there as in the other races and climates where it is found.

The *distribution* of elephantiasis is almost limited to the legs and scrotum. Sometimes only one foot is affected, sometimes the thigh remains free. One leg may entirely escape while the other forms a huge tumour, and the scrotum may be diseased independently or along with the legs. Lymph-scrotum is the name given to elephantiasis scroti (p. 556). The size of these scrotal tumours is sometimes enormous, the mass reaches to the ground, the penis is completely lost within it, and the whole weight may exceed that of the rest of the patient. As above stated, the organs involved in this monstrous tumour are, when dissected out, found perfectly normal, except that the tunica vaginalis is often the seat of hydrocele. Many remarkable

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cases with numerous illustrations will be found recorded in Esmarch's and Kulenkampff's monograph 'Die Elephantiasistischen Formen,' 1885.

Ulceration of the unwieldy mass of flesh often occurs, and the pain and discharge of the ulcers may produce a certain amount of cachexia.

The *cause* of the disease was until lately perfectly unknown, but owing to the remarkable discoveries made by Dr Lewis, Dr Manson, and other observers, it is now known that a certain proportion of cases of elephantiasis, particularly when it affects the scrotum, coincide with chyluria and the presence of a parasitic worm in the blood (*Filaria hæmatobia*, supra, p. 300). It is supposed that the lymph-channels are mechanically blocked by the parasites; this leads to œdema and inflammation on the one hand, and, when rupture into the urinary tract occurs, to chyluria on the other.

There is no doubt, however, that many cases of elephantiasis have been observed in which no filariæ could be detected in the blood. See a case with discharge of milky lymph recorded by Dr Wagstaffe ('Path. Trans.,' 1875, p. 215), and in the same volume one with great lymphatic dilatation figured by Mr Stewart, as well as a third case of ordinary pachydermia with histological details by Mr Butlin.

The *treatment* of this disease is purely surgical. There appears to be little or no power of restraining its course until the tumour is sufficiently large to be removed. From the famous cases of Clot Bey in Egypt to those of Dr Turner in Samoa, and other medical missionaries, the removal of these frightful masses of flesh has been one of the most brilliant benefits conferred by European surgery.

Closely allied to elephantiasis are the curious cases, described as *cutis pendula*, *pachydermatocele* or *dermatolysis*, in which the skin hangs in great folds like garments. They are often associated with the presence of multiple fibromata. A classical instance was recorded and figured by Meek'ren in 1657; the patient, a young Spaniard, could bring the skin of his chest up to his eyes and down to his knees. Dr Valentine Mott, of New York, published in the 'Royal Med.-Chir. Trans.' for 1854 five cases, with two portraits; in all of them the redundant masses of skin were successfully removed by operation, but in one the growth twice returned.

XANTHELASMA.*—This remarkable affection was originally described by Addison and Gull under the name of Vitiligoidea. See 'Guy's Hosp. Rep.' for 1851 (plates); *ibid.*, 1866 (plates); *ibid.*, 1877, with thirty-eight tabulated cases; and 'Path. Trans.,' 1866, p. 277 (plates); *ibid.*, 1868, p. 436; *ibid.*, 1882, p. 376, with thirty-six cases of multiple xanthelasma. Afterwards Dr Pavy, Dr G. H. Barlow, Dr Fagge, and many others published similar cases.

Xanthoma shows itself in two distinct forms. One is the commoner, and was first figured and briefly mentioned by Rayer as yellow patches on the eyelids in 1835 (see the writer's paper in the 'Guy's Hosp. Rep.' for 1877, xxii, p. 97); these flat patches (*Vitiligoidea plana* of Addison and Gull) cannot be detected by the finger, although they look raised and have defined margins.

The first indication of xanthelasma is the appearance in one of the upper

* *Synonyms.*—Plaques jaunâtres des paupières (Rayer)—Vitiligoidea plana et tuberosa (Addison and Gull)—Xanthelasma, *i. e.* yellow laminæ from ξανθός and ἔλασμα (Wilson)—Molluscum lipomatodes (Wilson). The term Xanthoma (yellow tumour) was suggested by Dr Frank Smith in 1869, and has been generally adopted in Germany.

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eyelids, just above the internal canthus, of a yellow, cream-coloured, or washleather-like patch. Afterwards similar ones come out in the same neighbourhood, and these may ultimately coalesce so as to form a broad ring surrounding the eyes. Like patches may show themselves elsewhere—on the surface of the body, on the backs of the hands, on the scrotum, and also on the palms and soles, where they either present a peculiar dotted appearance, or form long streaks following the creases of the skin. This plane variety of xanthelasma may affect mucous membranes as well as the skin. It occurs in the gums and palate, and in the larynx and trachea; Dr Legg has seen it on the side of the tongue, and in two cases Dr Fagge found it in the lining of the bile-ducts.

The second and rarer form of xanthelasma consists of raised solid nodules or tumours (*Vitiligoidea tuberosa*). These make their appearance later than the flat patches. They occur on the ears and on the limbs, especially on the extensor surfaces; they form aggregated tubera on the olecranon, and swellings on the knuckles not unlike those of gout. They are occasionally found, not in the skin itself, but in the adjacent tendons of the extensor muscles of the fingers. Several cases have been recorded in the 'Guy's Hosp. Reports' and in the 'Path. Trans.' Two cases are reported in 'Virchow's Archiv' (1883 and 1885) in which the eyelids were unaffected, but nodules and tumours occupied the elbows, fingers, knees, and buttocks.

When the flat plates and the nodules are found in the same patient, the term *Xanthelasma multiplex* has been applied. Twenty-three cases are tabulated in the thirty-third volume of the 'Pathological Transactions.' See also Dr Payne's case ('St Thos. Hosp. Reports,' vol. xiii).

Symptoms and course.—Xanthelasma is important, not only because of its pathological interest, but because it is often attended with much suffering to the patient. The parts affected with it are sometimes exceedingly tender. A patient of Dr Fagge's was unable to stand, or even to sit with comfort, on account of the pain produced by the slightest pressure on the xanthelasmic patches, and for a similar reason she could not use her needle. In this case the affection became much less marked under internal treatment, most of the raised tubera disappeared, and the pains were in great part removed.

Similar involution has been observed in other cases, *e. g.* a remarkable one associated with icterus from atrophy of the liver, recorded by the late Dr Frank Smith, of Sheffield ('Path. Trans.,' xxviii, 236).

Histology.—Microscopical examination shows that xanthelasma is essentially a chronic deep dermatitis with early fatty degeneration, the yellow colour depending upon the presence of innumerable fatty granules in the tissue. In the nodules there is also present a dense fibrous tissue, and even in the plane variety a few ill-formed cells have been detected. The minute structure of xanthelasma is thus identical with that of atheroma in an artery. Some dermatologists, however, taking the less frequent tuberose form as the type, describe the disease as a new growth. The distinction between chronic inflammation, hypertrophy, and granuloma is, as we have already found, by no means easy to make in every case, and we may regard xanthoma as one of the transition forms between the hypertrophic kinds of chronic dermatitis described in this chapter, the granulomata of syphilis, lupus and leprosy, and tumours, such as cheloid and cancer of the skin.

Pathology.—The multiple and tuberous form of the disease is most frequently seen in chronic cases of *jaundice*, from whatever cause it may arise,

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although it may also be found, particularly in children, in cases entirely free from icterus. Of eight cases of infantile xanthelasma multiplex not one was associated with jaundice; and in these the eyelids were not affected, as they almost always are in adults ('Path. Trans.,' vol. xxxiii, p. 383). In most cases it does not make its appearance until the patient has been jaundiced for a year or more, but in one case it began within six months after the jaundice, or perhaps even earlier. The more common *plane* form of xanthelasma which is confined to the eyelids has been shown by Mr Hutchinson to occur frequently in those who have suffered from sick headaches ('Med.-Chir. Trans.,' 1871).

Xanthelasma has been observed more frequently in women than in men, in the proportion of three to two. It occurs, like atheroma, most often in adults over forty years of age. Several examples are recorded in children, and two congenital cases by Dr Stephen Mackenzie and Dr Thos. Barlow ('Path. Trans.,' 1882 and 1884). Dr Church met with six cases of the affection in the same family ('St Barth. Hosp. Rep.,' vol. x).

No plan of treatment is known, but it is only in exceptional cases that symptoms arise, and there is some reason to hope for spontaneous recovery.

Xanthoma diabeticorum is the name given to a curious and rare affection of the skin, of which the relationship to classical xanthoma is not yet clear. The earliest case was the fifth in Addison and Gull's original paper (vol. vii, p. 268). The patient was a man, aged twenty-seven, and during the course of diabetes a papular eruption appeared on his arms, and rapidly spread over the trunk, the head, and the limbs. The papules were large, yellowish, and mottled, and looked like pustules. After several weeks these "tubercles" began to subside, leaving no obvious change behind them. This is certainly very different from the affection above described, and Dr Fagge ('Path. Trans.,' xix) and the writer ('Guy's Hosp. Rep.,' 1877, p. 131) agreed in excluding the case. Several similar cases have, however, been since reported by Dr Bristowe (as "keloid"), by Mr M. Morris and Dr Thos. Barlow ('Brit. Journ. Derm.,' vol. i and vol. iii, p. 106), by Dr A. R. Robinson (from New York), and by Gendre and Besnier (from Paris). They have also been called diabetic 'lichen.'

The histology of these papules is quite different from that of xanthoma ('Path. Trans.,' xvii, p. 414, and xxxiv, p. 284), and so are their anatomy, distribution, origin, and course. The papules are rather large, raised, and yellow, as if they were pustules. They occur chiefly on the limbs, but also on the trunk and head, and come out somewhat rapidly. They last a long time and then gradually fade away.

Instances have been reported of a similar cutaneous disorder without diabetes (*e. g.* Dr Cavafy's case, 'Brit. Journ. Derm.,' vol. i, p. 76), so that this rare condition must at present be regarded as uncertain in everything except its having no claim to the title of xanthoma.

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SYPHILODERMIA

"Indignas premeret pestis cum tabida fauces,
Inque ipsos vultus serperet atra Lues."
MART., i, 79.

Importance of lues as a cause of cutaneous lesions—their pathological character—The roseolous exanthem—the papular—squamous—pustular—bullous—macular—Alopecia—Tertiary lesions—Cicatrices—Diagnosis of syphilitic dermatoses—Hereditary syphilodermia—Treatment.

THERE is no doubt that the only satisfactory classification of diseases, whether of the skin or of other organs, would be an ætiological one; that is to say, one which expresses their true origin and natural history, not as so-called morbid entities, but as derangements more or less extensive and profound of physiological function. For if we know the origin of a disease, we not only understand its nature or pathology, and may hope to adopt rational means for its cure, but we also may still more confidently hope to prevent it, which is better than curing it. The objections to the supposed ætiology of diseases which we have so often urged throughout this book, are that the causes are too often assumed instead of proved, and that no cause can be logically admitted which is not an invariable antecedent of the effect. Hence it is that we demur to explaining pneumonia and other inflammations as due to cold, that we doubt the origin of rheumatic fever from the same cause, and cannot accept a gouty diathesis, a scrofulous tendency, or a neurotic disposition without a careful examination of the grounds on which such causes are assigned. The same reasonable scepticism applies to the dartrous or arthritic diathesis which is invented as a cause for many diseases of the skin; but just as no one believes firmly and intelligently in the use of any remedies if he believes in all, so we shall not either appreciate or trust to true ætiology until we have separated it from what is spurious. Enteric fever and scabies were both at one time ascribed to meteoric causes, to the influence of the seasons, to diathesis, to dyscrasia of the blood and humours, to chills, to mental affections, and to all the other commonplaces of unscientific speculation. We now know that their single, constant, and exclusive cause is the transference, in the one case of an animal, in the other of a plant, to the human organism. So dropped wrist depends solely and exclusively upon the presence of lead, and so a large group of diseases of the skin depend solely and exclusively upon the presence of the syphilitic poison.

In the time of Willan, and even in the present day in some quarters, psoriasis for example was regarded from the anatomical point of view as a scaly disease caused among many other vague and uncertain conditions by the presence of syphilis. We now know that true psoriasis is a definite and well-marked derangement of cutaneous nutrition, with its own natural history, cause, and characters, and is never due to syphilis; but what used to be called syphilitic psoriasis is no more entitled to the name than a rash

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caused by belladonna is to that of scarlatinal. If diagnosis is not to be mere learned or unlearned trifling, it means drawing a distinction—not between one pimple and another, but between morbid processes as distinct in their origin, nature, and practical consequences as poisoning by a drug, invasion by a fungus, and infection with a fever.

The separation, therefore, which we have advocated of the syphilides from all other cutaneous diseases was a great improvement upon Willan's pathology, an advance none the less important because it was unfortunately followed by a premature extension of the same excellent principle upon far less solid ground.

We have already in the first volume stated the general character of syphilitic infection, its mode of transference, generalisation, and transmission to a second generation. We have now to deal in particular with its manifestations upon the skin—syphilodermia (syphilis cutanea, lues in cute, les syphilides, dermato-syphilis).

The first fact to remember is that syphilis, as we then showed, is one of the specific febrile diseases, and its earliest effect on the integument is the production of an exanthem or specific rash strictly comparable to those of measles, typhus, and smallpox. The syphilitic process is, however, drawn out far beyond that of any other member of its class; and its exanthem is accordingly protracted in duration and extremely varied in form. It is as if a case of variola lasted not for a few weeks but for as many months. We should then see an early roseolous rash, followed after an interval by the appearance of papules with characteristic distribution and anatomy; afterwards vesicles would succeed, then pustules, crusts, hæmorrhages, and finally cicatrization; while the orderly march of the eruption would be interrupted by local modifications during its tardy and irregular progress. Hence it is that the cutaneous lesions of syphilis present so multiform a variety, and are so irregular in their appearance, locality, and course.

The second important pathological point is that syphilis, like many diseases, has sequelæ, and is itself so chronic, that it is often difficult to say when the disease is exhausted and the sequelæ have begun. Compared with the secondary lesions, which may be considered as forming part of a prolonged exanthem, the latter forms of syphilodermia may be regarded as sequelæ; but, nevertheless, they are certainly parts of the true syphilitic process, and may in their turn be distinguished from affections like tabes dorsalis which correspond more strictly to what in other cases we call sequelæ, the results that is, but not parts of the original disease.

The syphilitic exanthem ("syphilitic roseola").—The rash is erythematous in form, like that of measles and scarlatina; it occurs in patches, not uniformly; it is not raised above the skin, and disappears on pressure; the colour is in most cases an inflammatory redness tinged with brown pigmentation, so as to produce a tint which differs from the bright red of scarlatina, and from the purplish or rose-tinted hue of measles by the admixture of a yellowish tint, producing a salmon-coloured, coppery shade which French writers compare to that of the lean of uncooked ham. The patches are not sharply defined at the margin, and are irregular in shape. The most characteristic distribution is on the trunk, particularly the front and sides of the chest, on the abdomen, and on the neck. The rash is not infrequently seen upon the face, but is rare upon the limbs, and seldom or

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never reaches the hands or feet. It appears at a variable time after the primary infection, usually three or four weeks, but sometimes much later. It takes, as a rule, several days, and often a week, to develop; when it comes out unusually quickly, it may simulate the rash of measles or that produced by copaiba or some other drug.

The papular form ("syphilitic lichen").—Sometimes as a part of the early exanthem, more frequently after an interval, papules show themselves, large, pointed, discrete, and very early covered with small scales. They are more like those of psoriasis than of papular erythema or lichen planus, and are larger and less confluent than those of eczema. They appear on the trunk and limbs, and also on the forehead, at the roots of the hair. Like the exanthem, they produce as a rule neither pain nor irritation.

One variety of papular rash may occur early in the post-erythematous series. It is marked by each papule being formed around a hair sac. Hence Dr Bäumler has proposed to call it the *follicular* syphilide. The papules are small and pointed, "miliary." They may be discrete or clustered, they appear in successive crops, and may last for several weeks or even months. They sometimes become scaly, sometimes vesicular, but most often, perhaps, pustular.

Squamous syphilodermia, once known as "syphilitic psoriasis," most frequently occurs as a further development of the papular eruption just described. The scales are much smaller than those of psoriasis, and have a dirty yellowish colour instead of a silvery lustre. The distribution of the scaly syphilide is much like that of the papular stage, except that it frequently affects the palms and soles even thus early. The trunk, limbs, and face, the scalp, and the genital organs, may all be the seat of this form of eruption; but it affects the flexor rather than the extensor surfaces, and avoids the regions peculiar to psoriasis, the elbows and the knees. At this stage Mr Hutchinson describes the occasional appearance of a kind of erythematous ring on the arms and trunk, which appears again and again, when the patient has left his bed or has taken his bath, and lasts for a short time.

Pustular syphilide (syphilitic "impetigo" and "ecthyma").—Venereal pustules are usually developed out of the papular stage, but sometimes seem to come independently; they are often large and discrete, whence they have been called by the obsolete name of ecthyma. They are frequently mixed with papules and scales and injected maculæ, giving the polymorphic aspect which is characteristic of syphilis. This pustular eruption is irregular in locality, but is frequent on the scalp, face, and trunk, and comparatively rare upon the limbs. The pustules are very commonly followed by minute white scars, which are often useful in subsequent diagnosis. Crusts form much as they do with eczema and impetigo, but instead of being green or yellow they are usually a reddish brown.

The pustular eruption of syphilis has often to be carefully distinguished from varicella and from the modified form of smallpox.

A form of pustular syphilide, which Bäumler has described as "follicular," is that in which each pustule is pierced by a hair It has a swollen dark reddish base, forms a minute yellowish crust, and leaves a white cicatrix. They come out in great numbers on the face, chest, shoulders, and limbs. This is the form which was formerly known as "syphilitic acne."

Vesicular and bullous syphilides.—The older dermatologists did not admit

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"syphilitic eczema," and there is no doubt that vesicles are extremely rare as the result of syphilis.*

Bullæ are not at all uncommon among the later secondary lesions. They become pustular, and form massive conical crusts, sometimes with a curious resemblance in form and colour to a limpet shell. This is the eruption which was formerly known as *rupia*. When the crust is removed ulceration is found beneath it. A bullous syphilide is also common in congenital cases upon the palms and soles; it used to be known as "pemphigus neonatorum syphiliticus."

Macular syphilides.—All the eruptions just described are marked more or less decisively by the coppery pigmentation characteristic of the disease, but somewhat late in the secondary stage maculæ without any other lesion begin to appear. These primary syphilitic stains are much more common in women than in men. Hardy described them as usually appearing on the neck and front of the chest, consisting of irregular spots of a café au lait colour, as large as a franc or half franc piece, and often confluent. In the Guy's museum is a model and drawings taken from a typical case of this form of syphilide which was under Dr Barlow's care in 1856. The patient, a woman, had dark brown maculæ scattered over her chest and arms; it was eight months since the beginning of the syphilis, and the stains faded under treatment.

We shall see in a subsequent chapter (p. 934) that increased pigmentation (melasma, melanodermia) frequently coincides with an adjacent deficiency of pigment (leucodermia), and this is what often occurs with the pigmentary syphilide just described, defined white patches appearing in the diffused dark patch. Varieties of aspect and distribution have been distinguished as "marble-like," "macular," and "lace-like."

Beside the most frequent position on the neck, it may appear on the flanks, or the chest, or abdomen.

Its period is often early, from three to six months after infection, less frequently somewhat later, but when once developed it continues unaltered for three or four years or even longer. It then disappears spontaneously. Its appearance usually coincides with that of mucous patches and condylomata, but sometimes is as late as tertiary nodes.

Syphilitic alopecia is an early symptom and is not always dependent upon a scaly or pustular affection of the scalp; when this is absent we are probably right in regarding the loss of hair as of the same nature as that which often follows many of the specific fevers. It is distinguished from *alopecia præmatura* by not specially affecting the forehead or the back of the head. It is distinguished from *area* by the absence of circumscribed smooth hairless patches. The hair becomes thin and forms irregular bald patches in various places.

Tertiary dermato-syphilis.—The pustular and especially the rupial eruptions often come so late in the secondary period that they are accompanied by syphilitic ulcers, and, again, true gummata may occasionally appear in the skin at a comparatively early period; in fact, the yellow nodules of early syphilitic iritis are considered by good pathologists to be themselves minute gummata (vol. i, p. 288). We cannot, therefore, draw an absolute line between the preceding forms of syphilodermia as symptoms, and the tertiary

* Hutchinson speaks of a form of syphilide which is attended with clusters of vesicles like those of shingles, but which is bilateral and widely distributed; and Hardy describes three varieties of syphilide which he terms eczematous, varioliform, and herpetiform.

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lesions as sequelæ; but whether we use the word tertiary or speak only of late symptoms, the distinction is practical and important between the roseolous, macular, papular, squamous, and pustular syphilides on the one hand, and the later nodes and ulcers on the other. The former are forms of dermatitis, the latter of granuloma. The former are associated with sore throat and iritis, the latter with the visceral affections described in the first volume (p. 290).

Syphilitic *condylomata* and mucous patches have been described already (pp. 884—5).

The syphilitic *ulcer* may begin in a cutaneous gumma or beneath a rupial crust, or in a patch of syphilitic scales or pustules. It spreads from the margin, or, as the phrase is now applied, it is serpiginous. It is usually, but not always, multiple; the edges are rounded and punched out. It causes remarkably little irritation or pain; it may occur upon any part of the body, trunk, head, or limbs; it may often be distinguished by its presence upon parts which are little liable to other forms of ulcer. Hence an ulcer of the arm is more likely to be syphilitic than one of the leg, and an ulcer over the fibula than one above the inner ankle.

Syphilitic cicatrices follow the pustular, the rupial, and the ulcerating lesions. They are often rounded or horseshoe in form, pigmented, smooth, and atrophic rather than hypertrophic. Their irregular localisation is, perhaps, the best evidence of their nature.

Diagnosis.—It is most important to distinguish syphilitic diseases of the skin from those of traumatic, febrile, and idiopathic origin; for, apart from other considerations, an error in diagnosis is fatal to successful treatment.

The rules for diagnosis of syphilis so far as relates to the skin may be briefly given as follows:

Firstly, what is called *history* is a most fallacious guide. In hospital practice the writer was accustomed to neglect it entirely; and although, when the patient's statements are carefully sifted, they have a certain value, the greatest mistakes are those which are due to reliance upon this misleading character; for we must always remember that even the most truthful patient may be quite deceived in supposing that he had syphilis, when he suffered from a different venereal malady, or may have been erroneously informed that a venereal sore was non-specific; and, as we insisted before, syphilis is, in a considerable minority of cases, a non-venereal disease, and in a certain number may be transmitted without any primary lesions at all (cf. vol. i, p. 291).

Secondly, the elementary lesions of dermato-syphilis are *multiform*, not mere stages of inflammation as in eczema or erythema multiforme, but diverse from the beginning.

Thirdly, the *colour* is an important character, but is sometimes absent from the earliest exanthem, when its presence would be most useful for diagnosis. Even in subsequent forms it is occasionally much less marked than usual, and may be simulated by certain cases of chronic eczema or psoriasis, or still more frequently by Lichen planus.

The irregular *distribution* of the syphilides is an important aid to diagnosis. Syphilis in its early stage may, if extensive, be symmetrical in the same sense as measles. But true symmetry, when homologous regions are independently affected—such as we see in psoriasis and eczema—is seldom or never seen in syphilis, with the exception of that which affects the palms and soles.

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The absence of itching from the earlier, and of pain from the later, eruptions is very remarkable, and almost though not absolutely constant.

Lastly, the presence of concomitant lesions in other organs than the skin, the eyes, the throat, the lymph glands or the testes, the presence of scars or maculæ, will often render a doubtful diagnosis certain, or their absence will be decisive against a suspicious eruption being specific. Nevertheless, we must be on our guard against the common fallacy of supposing, because a patient has had syphilis or is even suffering from syphilitic lesions at the time of examination, that any affection of his skin must needs own the same cause. We have only to remember that a man suffering from syphilis may be attacked by smallpox, typhus, or scabies, and that a patient suffering from chronic psoriasis or varicose ulcers may contract lues, to see the fallacy of trusting to what after all is only a probability.

Congenital syphilodermia has essentially the same characters as that due to an acquired lesion; in infants the commonest eruption is a coppery, blotchy rash on the nates and thighs. The bullous syphilide of the soles is almost peculiar to the congenital disease; this is often accompanied with mucous tubercles of the anus. In later childhood, typical gummata may develop and leave the characteristic ulcers.

The *prognosis* and *treatment* of dermato-syphilis is that of the general disease of which it is part, and has been already fully discussed in the first volume. Mercury is almost always indicated, and iodide of potassium or sodium should only be substituted when cutaneous gummata or tertiary ulcers are present. Phagedænic ulceration always called for treatment by opium.

Locally, the slighter forms of eruption need no special treatment. Ulcers and raw surfaces should have black wash or red ointment applied, and condylomata should be dusted with calomel; or, if there is a sloughing or otherwise unhealthy surface, iodoform is often used with great benefit.

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LUPUS

AND ITS ALLIES

"Heus tibi autem—Quidnam est?—Lupus in fabula."
TERENCE, *Adelphi*, iv, i, 31.

Definition, history, and nomenclature—Anatomy, histology and local course—Locality—Symptoms—Age, etc.—Diagnosis from cancer, rodent ulcer, and syphilis—Pathology—Relation to tubercle and scrofula—Clinical course and prognosis—Treatment, local, internal, and by inoculation with Koch's fluid—Scrofulodermia and tubercular ulcers.
Lupus erythematosus—Pathology—Locality—Course—Histology—Treatment.
Disseminated erythematous lupus—Rhinoscleroma.

Of all diseases which affect the skin alone, lupus is the most destructive. Unlike syphilis, leprosy, and malignant growths, it is a purely cutaneous disease. It affects the deep layer of the cutis, and the epidermis is only involved subsequently. It also spreads to the subcutaneous tissue and occasionally to adjacent mucous membranes, but rarely to the cartilages or fascia propria beneath the skin; and it never attacks muscles, bones, or other deep structures. Lupus is at once a chronic deep dermatitis of a special kind and a new growth in the modern sense of the word. Virchow included it with tubercle, leprosy, and syphilis among the Granulomata, or new growths which consist of the same corpuscular elements which form the granulations of a healing ulcer. By the French writers it has been almost universally assumed to be tubercular in nature, and forms the type of a supposed natural family of Scrofulides. In earlier times lupus was confounded with ordinary chronic ulcers, with cancer, with leprosy, and most of all with the later forms of lues. The lupus exedens of older writers was in most cases tertiary syphilis, *e. g.* the woodcut given as a type of the disease in Druitt's 'Vade Mecum.'

Lupus* was first carefully defined and described by Willan, and was figured by Bateman in his sixty-seventh plate. He placed it among Tubercula and noticed its characteristic preference for the face. The distinctions subsequently introduced:—*Lupus exedens* and *L. non exedens* (Rayer), the lupus "qui détruit en surface—en profondeur—avec hypertrophie" (Cazenave and Schedel)—*Lupus serpiginosus, syphiliticus* and *vulgaris*—with many others—are unnecessary or mischievous. Only one aberrant form, or rather allied disease, *Lupus erythematosus*, need be separately described or named.

Anatomy.—Lupus begins by the formation of minute nodules of granulation-tissue in the deeper layer of the cutis. These can be felt like shot in

* *Synonyms.—Noli me tangere, Tentigo prava, Impetigo rodens,* and *Herpes exedens.* The word "lupus," which has been traced back by Virchow to the school of Salerno in the thirteenth century ('Archiv,' vol. xxxii, 1865), expresses the destructive ravages of the disease. It is applied to an incurable ulcer by Alexis of Piedmont (1578). The dramatist, Webster, uses the English equivalent in the phrase "the ulcerous wolf."

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the skin, although without hardness, and show as reddish spots which mark the "macular" stage. When exposed, they are found to be vascular; and when several are seen together, a yellowish tint is sometimes observable, which with their soft translucent appearance has led to their being compared to apple-jelly.

Histologically, they consist at first of small nucleated exudation-cells, with very scanty stroma. As the disease goes on, these granulation nodules unite, and undergo changes in two directions. The intercellular substance may become a stroma of delicate connective tissue. This may increase and acquire firmness until it becomes connective tissue with spindle-shaped corpuscles; and finally may form a firm, fibrous, contracted, or œdematous tissue which resembles an atrophic or hypertrophic cicatrix. More frequently, however, either universally or with only a certain amount of the fibrous transformation just described, the new-formed lupus tissue breaks down, the nodules become confluent, the cells undergo fatty degeneration, ulceration destroys the new growths, the epidermis gives way, and an ulcer results. The floor of this ulcer is formed by lupous nodules which can be distinguished by the naked eye from the healthy granulations of a healing sore. The edges are somewhat raised and can generally be felt to consist of nodules which have not yet softened. The pus secreted is usually thin and scanty. While fresh deposition of nodules and fresh softening and ulceration ensue, there is usually some effort at repair by the fibrous transformation above described.

The skin around, though red and slightly swollen, does not feel hot, and the redness is of a venous tint.

The whole process is strikingly similar to that which occurs in the lungs during the course of phthisis. There, also, we have minute nodules of granulation-tissue, which have been described both as new growths and as inflammatory. There, also, the nodules undergo softening and ulceration; the ulceration spreads, with chronic inflammation and continual deposit of fresh "tubercles." There, also, the ulcerative process is rarely unaccompanied by some amount of fibrous transformation, which in favourable cases leads to the involution of the disease and the formation of a cicatrix. The bearing of this resemblance on the theory of lupus will be presently seen.

The process above described is extremely slow. We may watch lupus for more than a year before it ulcerates. It usually begins at a single spot and spreads irregularly therefrom, sometimes in a serpiginous form and comparatively swiftly, more often with an irregular rounded shape. It is rare for two independent foci of lupus to be seen, but this may sometimes occur. Whether in separate patches or as a single spreading surface, the disease is decidedly unsymmetrical, unless it happens to begin in the median line.

The epidermis is usually more or less thickened, particularly the deepest layer; but the original seat of the process is in the papillary layer of the cutis, whence it spreads downwards as well as upwards, until the whole thickness of the true skin is infiltrated. According to Auspitz, the sebaceous glands are destroyed, the sweat-glands are unchanged, the hair-follicles disappear or are transformed into cysts. Rindfleisch asserts that lupus begins in the sebaceous glands, not the erythematous or so-called sebaceous form but lupus vulgaris; and he therefore calls it an adenoma. But this no doubt is a mistake: any inflammatory disease will show most exudation in the more vascular parts of an organ, and the most vascular parts of the skin are the papillæ and the sebaceous glands; but the glands themselves are not

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involved except as a secondary result in the disease. Neither Neumann nor subsequent histologists agree with Rindfleisch.

Giant-cells are frequently observed (Friedländer in 'Virchow's Archiv,' 1874; and Thin, 'Med.-Chir. Trans.,' vol. lxii). Bacilli either identical with those of tubercle or closely resembling them were discovered by Doutrelepont in 1883. These characteristic bacilli, though no doubt always present, are very few in number, so that it takes much time and many sections to discover them.

Locality.—Lupus by preference attacks the face, particularly the alæ of the nose, the edges of the lips, the cheeks, the eyelids, and the conjunctiva. It also occurs upon the ears and spreads to the neck. It is rarely seen on the scalp and is not common on the trunk and limbs; but there is probably no part of the body on which lupus has not been observed, and although, as just stated, it is rare to see two lupous ulcers at once, it often, after appearing and being cured upon the face, reappears in another region. In Vienna the trunk and the buttocks are said to be more often the seat of lupus than the arms and legs; the hands and feet are almost exempt.

Lupus also affects the mucous membrane of the nose, the lips, the hard and soft palate, and the larynx (cf. vol. i, p. 900). It very rarely affects the tongue or the deeper mucous membranes. According to Hebra, however, there is no doubt of the cartilages of nose and ears being occasionally affected, and even tendons and ligaments of joints. Lupus vaginæ has been described by Dr Matthews Duncan.

Symptoms.—As the progress of the disease is slow and its local signs torpid, so its symptoms are but slight. It is astonishing how little pain is felt even when extensive tracts of the skin are deeply ulcerated; as we shall presently see, the remedy is far more painful than the disease. The general health is also unaffected; so that, except for the disfigurement, lupus would be one of the most easily borne of all serious and destructive diseases.

Ætiology.—Apart from the question of its relation to tubercle, which will be presently discussed, we have no knowledge whatever of the cause of lupus.

It is probably equally common in both sexes. With respect to *age*, it is, as commonly seen, a disease of young adult life. But it has then lasted in most cases for several years, and it may occur in young children. We have observed it in those not above four or five years old, but it is more common about puberty, and usually begins at from fourteen or fifteen to twenty. After thirty it is certainly rare for it to begin, though cases of undoubted lupus may be occasionally observed which start even later.*

In young subjects it shows an almost constant tendency to spread, even when repair, as generally happens, is to some extent occurring at the same time; but after thirty lupus tends to undergo involution, and if left to itself will in most cases end in cicatrices, disfiguring or disabling the patient, but no longer active.

Lupus is not hereditary, and has never been seen at birth, nor does it often affect more than one member of the same family.

Devergie noticed that lupus is more often seen amongst hospital patients than in private practice, and this is certainly true in England as well as in France. It is more common in Vienna than in London.

* According to Hebra and his disciples, it always begins before puberty, occasionally in infancy, but usually from the fifth to the ninth or tenth year.

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Diagnosis.—The fact of lupus being a deep inflammation of the skin and leaving scars, at once distinguishes it from eczema and all the superficial forms of dermatitis enumerated in the earlier chapters of this section; nor is there much practical difficulty in the diagnosis from varicose and other traumatic or accidental ulcers. The real difficulties of diagnosis arise between lupus, syphilis, rodent ulcer, and cancer of the skin.

Lupus is distinguished from *cancer* by the absence of pain, by its slow progress, by its beginning early in life, by the presence of granulations, the absence of hæmorrhage, and the nodulated, but not uniformly and densely infiltrated, edge. Invasion of deeper structures and secondary enlargement of the corresponding lymph-glands decides the case to be cancerous, but our object is to establish a diagnosis long before this point has been reached.

Rodent ulcer is covered by an adherent reddish-brown scab, which when present is characteristic, but it may often have been removed by accident, by poulticing, or by other remedies before the lesion is seen. The edges are neither thick, hard, and infiltrated like those of epithelial cancer, nor do they contain little nodules as in lupus. Granulations are absent, the ulcer being of the kind known as "indolent," while that of lupus is what is called "weak." Like lupus, the face is its favourite seat, but the neighbourhood of the eyes rather than the cheeks and nose; it is always single and never extends so widely as lupus; it makes no attempt at spontaneous cicatrisation; and it only occurs in those who are past middle life.

Syphilitic ulcers have an undermined, not an infiltrated or nodular edge; the colour of the surrounding skin is brownish or yellowish, whereas that of lupus is of a more venous, *i. e.* purplish-red. In both there may be considerable crusts, forming what is described as "rupia" and "ecthyma" in the one case, and as "lupus pustulosus et crustaceus" in the other; but when these are removed, the more characteristic ulcerated surface beneath will be seen. The scars which result when a syphilitic ulcer is healed often resemble those of lupus, but they are less apt to be hypertrophied, they are much more pigmented, and seldom present the pink aspect and enlarged veins which are often seen after lupus is healed.

Moreover, tertiary syphilitic ulcers begin as a rule in the formation of a gumma in or under the skin, deeper than the nodules of lupus and less early affecting the epithelium. In the nose this distinction is most applicable—lupus begins at the edge of the nostril and slowly creeps on, only affecting the cartilages (if it does at all) in its latest stages; whereas syphilis begins in the perichondrium or periosteum, and has already destroyed much underlying tissue before the ulcer on the skin appears. The nose which has lost its tip or alæ has usually been affected by lupus; that which has lost its bridge by syphilis. Extensive disease of the skin, with the cartilages and septum intact, is most likely lupus; a small ulcer with a deep foul cavity beneath it, and exposed bone and cartilage, is almost certainly syphilis. The frightful cases of destruction of the greater part of the face and opening of the orbit, pharynx, and posterior nares, which figured in museums and plates as lupus exedens, were tertiary syphilis, neglected or ill-treated, cases which the better diagnosis and improved therapeutics of modern times have happily banished from civilized countries.

Apart from the local characters, diagnosis between syphilis and lupus will be much helped by remembering that syphilitic ulcers are frequently multiple, lupus very rarely so; and that secondary implication of lymph-

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glands, with characteristic induration, is common in syphilis—rare and only as the accidental consequence of temporary inflammation, which renders them soft and painful, in lupus. Again, the syphilitic ulcer is usually accompanied by other cutaneous lesions; the lupus ulcer has no complication. Syphilis begins after puberty, often long after; lupus before puberty or shortly after. Lastly, lupus is a disease of the skin and nothing else, whereas syphilitic gummata and ulcers will be generally accompanied by other signs of lues in the bones, glands, tongue, or viscera.

Of the cutaneous lesions of congenital syphilis, the early coppery rashes have no resemblance to lupus, and the later gummatous ulcers are exactly like those of tertiary acquired syphilis.

Pathology: relation to syphilis.—Although it is scarcely possible to confound lupus with the ordinary lesions of congenital syphilis, it has been supposed by respectable authors that lupus is the result of inherited syphilis which does not show itself in the ordinary form. Hebra himself was led into this opinion by a striking case in his own practice, where a syphilitic father had borne to him—first, a stillborn child; secondly, one which died in a few months with the ordinary marks of inherited syphilis; then a third who survived, after suffering from congenital syphilis of the skin and bones; while the last who was born apparently healthy, remained so for some years, but became before puberty the subject of typical lupus. The case is no doubt striking, and is supposed to show that lupus is the feeblest and most diluted effect of transmitted syphilitic virus. But no one pretends that a child with a syphilitic father or brothers is thereby prevented from becoming the subject of lupus, and if so the two diseases must occasionally occur in the same family or the same individual. Persons affected with lupus may acquire syphilis, and persons who inherit syphilis may be attacked by lupus. Owing to Mr Hutchinson's classical observations, we can now recognise congenital syphilis not only in infants but in later life. Such persons are not more liable to lupus than others; and one ought to be very sceptical in admitting that congenital syphilis is present when it shows itself by none of its unequivocal characters.

The assumption that there is such a thing as "syphilitic lupus," a kind of hybrid between two diatheses, is also unjustified; and, like similar diagnoses of "rheumatic gout" or hybrids of scarlatina and measles, is practically mischievous. At the same time the diagnosis between syphilis and lupus is often difficult, and even with care and experience one may mistake the one for the other—at least the present writer has done so.

Relation to tubercle.—Is lupus, as the French school assert, a "scrofulide"? This word was invented by Bazin and Hardy in imitation of the Syphilides of Biett and Alibert. Bazin includes among "Scrofulides bénignes," chilblains, erythema, strophulus, prurigo, lichen, eczema, impetigo, and some forms of acne. These are justly excluded by the sounder judgment of M. Hardy, who precisely defines his scrofulides as depending exclusively upon scrofula as the syphilides do upon syphilis, never developing without it, and diagnostic of its presence. He would place under this definition lupus, which Biett had, with his usual good sense, separated from all other diseases, without inventing an ordinal name for it, while Alibert had lumped it with eczema and psoriasis among the dartres. Cazenave described four species of scrofulide under lupus; erythematous, tubercular, ulcerous, and hypertrophic. Bazin's division of "Scrofulides malignes" was into erythematous, tubercular, and *scrofulide crustacée ulcereuse.* Hardy addresses

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the same reproach to the third of Bazin's as to the last two of Cazenave's species, and himself describes five varieties of scrofulides:

1. *Scr. erythemateuse.*—This corresponds to erythematous lupus, an undoubtedly distinct form, which will be described below.

2. *Scr. cornée et acnéique.*—This is not what other French writers describe as acné cornée, a curious affection of the sebaceous glands unaccompanied with inflammation ("ichthyosis follicularis") very rare, and in the cases which the writer has seen without the slightest claim to the epithet scrofulous (p. 844). The *scrofulide cornée* of Hardy consists of groups of comedones, placed on a purplish red patch, and is followed by depressed cicatrices unpreceded by ulceration. It appears upon the face, has a very slow course, and occurs (we may presume) only in persons who for some other reason are entitled to the epithet scrofulous. It corresponds to Devergie's *Herpès crétacé*, and to Chausit's *Acné atrophique.*

In London, and probably in Vienna, this affection would probably be called lupus erythematosus or lupus sebaceus indifferently or conjointly.

3. *Scr. pustuleuse.*—This is the most frequent variety; it begins either by a number of pin's-head pustules grouped on a small red patch, lasting from a week to a fortnight and leaving a yellow scab, or else with a large pustule, like that of ecthyma, which when ruptured gives place to a dark prominent crust, which other dermatologists would name rupia. The part usually affected is the nose, the course is very slow and unaccompanied by itching or pain, but the most characteristic point is that when the crusts, which are very adherent, are removed, ulceration is found beneath. This ulceration is not deep, the surface is pale and sometimes presents little hard, dry, rough, warty nodules, which led Hardy originally to describe the variety as "scrofulide verruqueuse."

This form would by German, English, and American dermatologists be recognised as typical lupus (*Impetigo rodens*), which, as above described, frequently begins in pustules and is accompanied by large scabs. The slow course of the disease makes the subsequent ulcerated stage much more familiar, but even if watched from the beginning, cases of lupus with pustules and large prominent crusts are seen in London, in a minority not less, perhaps, than a third or fourth of the whole.

4. *Scr. tuberculeuse* is divided again into a superficial and a deep variety, and the former distinguished as sometimes disseminated over various parts of the body and sometimes localised, sometimes inconspicuous and ending in a light atrophic scar, sometimes hypertrophied, especially when it affects the genital organs. M. Hardy speaks of these deeper tuberculous scrofulides as producing "ces vastes destructions, ces plaies épouvantables et hideuses qu'on ne rencontre que trop souvent à la face," and as occasionally proving fatal, with profuse suppuration, cachexia, and hectic. This form, according to the eminent author quoted, produces enormous cicatrices on the eyelids, the lips, the neck, the ears, the nostrils, like those produced by severe burns.

This is obviously lupus exedens in its severest and most destructive form, but not differing from the slighter forms accompanied with true ulceration, except in degree. Moreover, even when untreated, the ravages of lupus, however hideous, are more remarkable for their contrast with the deeper destruction of syphilis and cancer than for their extent and severity considered as a disease of the skin.

5. *Scr. phlegmoneuse.*—This is a superficial ulcer which begins in a phlegmon as big as an almond or a nut; this gradually softens, fluctuates,

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acquires a purplish-red colour, and at last discharges a little thin pus; a scab forms, and this process may be repeated and become chronic until a large surface becomes ulcerated. The disease appears chiefly on the face, but also on the trunk and limbs. It also leaves a scar, at first violet-coloured, afterwards pale, irregular, and reticulated.

This somewhat rare variety will be recognised as what older surgeons, and especially the late Mr Hilton, used to describe as "scrofulous ulcer." No doubt it deserves separate mention, but whether regarded histologically, or from the point of view of pathology or of treatment, it is closely allied to lupus. When, however, it occurs on other parts than the face, the primary abscess is often due to suppuration of a tubercular lymph-gland, of which there are not a few too small to be recognised by the anatomist, but apparent when enlarged by the hypertrophy of Hodgkin's disease or by caseous inflammation.

A review of these varieties of the scrofulides defined by the most experienced and the most rational of the successors of Biett, and described in his admirable 'Leçons'* with the clinical acumen and skill characteristic of Professor Hardy, shows that the only diseases of the skin which have any title to be called scrofulous are those which Willan and Bateman, with their successors in England, and Hebra, with his disciples in Germany, would agree in calling lupus. It is remarkable that the rare papular affection of the skin described by Hebra as lichen scrofulosorum (p. 797), and also the dry, harsh, unoiled condition called pityriasis tabescentium (p. 846), are not included in the above account of scrofulides.

What ground, then, is there for ascribing lupus to Scrofula? This raises the question of the meaning which we attach to that much-abused word.

Scrofula originally denoted a swollen neck, which in some children makes the head pass into the shoulders with scarcely any constriction, as it does in a pig (*scrofa*). It is found that this usually depends upon a chronic caseous enlargement with characteristic suppuration and subsequent cicatrices of the cervical lymph-glands. The word *struma* also meant a swollen neck, and while in England it is used as a more or less vague synonym of scrofula, which had better be discarded, in Germany it is applied to another cause of a chronic swollen neck, namely, bronchocele or goître.

The mere form of degeneration is, as Virchow long ago pointed out, not characteristic; for it may occur in a traumatic abscess and in atheroma, in the middle of tumours and even of cancers. Moreover, most cases of caseous disease of the lympharia would, on careful examination, be found not to be idiopathic but secondary to mucous or cutaneous irritation. If indurated lympharia are discovered, we at once seek for a primary affection in a chancre, if cancerous in a primary tumour or an epithelial surface, if suppurating in a primary wound or inflammation of the skin or mucous membrane. In the same way caseous lymph-glands can generally be traced to chronic inflammation of the surface from which they receive their lymph. In the neck they are most frequently traceable to the throat with its tonsils and other lymphatic organs, more rarely to the scalp, the teeth, or the ear; bronchial lymph-glands become caseous in consequence of chronic or repeated subacute bronchitis and broncho-pneumonia; mesenteric lympharia in consequence of chronic or subacute enteritis and diarrhœa. These three groups of lympharia in the neck, the thorax, and the abdomen are the

* The later volume ('Maladies de la Peau'), published in 1886, does not on this subject deviate from that which was taught in 1864.

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principal seats of so-called scrofula, and the reason is probably because the mucous membrane of the fauces, the bronchial tubes, and the small intestine is pre-eminently rich in adenoid or lymphatic tissue. According to the more rational believers in scrofula as a diathesis, disposition, or general pathological tendency, caseous disease of lymph-glands is clinically found connected with caries of the bones, with chronic inflammation of the articular ends or of the synovial membranes in joints, and with certain forms of catarrhal ophthalmia. On these points we do not presume to speak with authority; but a physician certainly sees many children with caries of bones or chronic inflammation of several joints, who have no affection whatever of their lymphatic organs; and there are many children, and some adults, with caseous inflammation of cervical and other lymph-glands, which lasts for years without their bones, their joints, or their conjunctiva ever being affected. Watson's classical account of the two types of scrofulous children left one sceptical of the same morbid disposition showing itself in such opposite ways; and it is now clear that "pretty scrofula" was in most cases tuberculosis and "ugly scrofula" inherited syphilis.

This brings us to the question of the relation between scrofula and tubercle, and the relation of lupus to each. After phthisis and scrofulous pneumonia had been long assumed to be pathologically identical with scrofulous glands and joints and bones, Virchow introduced the critical light of histology into the confused mass of doctrine on this difficult subject. Regarding miliary tubercle as the type of that condition and as essentially a granuloma or new growth of an adenoid or lymphatic type, he defined the scrofulous diathesis merely as "vulnerability," that is, inability of the organism to recover from slight injuries. Subsequently, however, scrofula, in the only definite anatomical sense of the term, has again approached tubercle; for, first, the giant-cells of Schüppel, which were imagined to be characteristic of tubercle, were found also in scrofulous lymph-glands by Friedländer; and now the still more famous *bacillus* of Koch has been discovered in these same organs.

The nodules and granules of lupus contain a minute bacillus which in form, size, and reaction to staining agents is indistinguishable from that found in phthisical sputum. Nevertheless no physican with clinical experience is prepared to admit that the uniform presence of a bacillus, any more than of a histological element or of a chemical product, can settle the true affinities of morbid processes, which must be judged of ultimately by their natural history and physiology, not by their anatomy, chemistry, or mycology. In this as in other matters, to use Hebra's dictum, where the pathologist and the clinical physican differ, clinical knowledge must be the master: "Wo der Patholog und der Kliniker im Streite sind, muss der Kliniker Meister sein." Admitting, then, that the *bacillus lupi* is constant, and that the same organism occurs in tubercle, in scrofulous lymph-glands, and in lupus, the question still remains whether lupus occurs in persons who have definite signs of scrofula or who are subject to tubercular diseases.

To the latter question we must answer, No. It is extremely rare to see lupus among the countless victims of phthisis, *i. e.* of chronic tubercular inflammation of both lungs, beginning at the apex, travelling down, ulcerating and destroying the tissue, and associated with laryngitis, enteritis, and tubercles in the viscera. Nor, looking at the question from the opposite point of view, has the writer found among patients with lupus, either in hospital practice in London or in the large numbers under Hebra's own

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treatment, any considerable number of cases of phthisis ;* and yet phthisis is remarkably common both in England and in Vienna, so that it has been regarded by English writers as the characteristic scourge of this country, and by Austrian writers as so peculiar to Vienna that its prevalence has been explained by the geological condition of the soil.

Nevertheless, we do sometimes meet with caseous glands or scrofulous scars in patients with lupus. Perhaps the occurrence is not more frequent than mere coincidence would explain, when we remember that both lupus and scrofula principally affect children and young adults. This very predilection, however, for a certain period of life may be fairly brought forward as an argument for a relation between the two diseases. A more powerful argument is the considerable resemblance in the mode of treatment which is found effectual for both.

On the whole, we conclude that lupus has a certain pathological relation to caseous or tubercular disease of the cervical lymph-glands, independent of its histology and of the presence of a bacillus; but that, apart from this relation, it has no connection with phthisis or with general tuberculosis; in fact, the clinical relation between phthisis and general tuberculosis on the one hand and tubercular or caseous lymph-glands on the other, is a slight and uncertain one. It is also clear that with the unimportant exceptions of so-called lichen scrofulosus and pityriasis tabescentium, all the diseases of the skin which have any true connection with scrofula or tubercle may be comprised under the name of lupus. Lastly, nothwithstanding these concessions, we must maintain that it is not justifiable to forsake the old, well-understood, short, and expressive term of lupus, one merit of which is that it expresses no theory and begs no question. In many cases of lupus, those who believe in tendencies and diatheses may call the patient scrofulous, just as in many cases of eczema they may call him gouty, and in many cases of erythema, rheumatic. Few would deny that lupus may be called scrofulous if the patient shows scars which prove that he has had caseous lymph-glands, that eczema may be called gouty when it occurs in a patient who has tophi in his ears or in his joints, and that erythema may be called rheumatic when it occurs in a patient who has suffered or is suffering from rheumatic fever. But in the great majority of cases of lupus, as seen by the writer in Vienna, since then in London, and also in Paris, there was nothing which an unbiassed observer would have called a sign of scrofula excepting the disease of the skin.

Auspitz—who has widely departed from Hebra's classification—puts lupus among what he styles "chorio-blastosen," or anomalies of growth of the corium and subcutaneous tissue. He subdivides this group into simple hypertrophic (macrosomia) and paratypical or abnormal growths, which include the granulomata. Here lupus finds a place side by side with leprosy, scrofulodermia papulosa (or lichen scrofulosus), and scrofulodermia pustulosa (or acne cachecticorum), scrofulodermia ulcerosa (or scrofulous ulcers of the skin), tuberculosis cutis (as a separate condition), syphilis, and lastly, rhinoscleroma.

This is practically following Virchow's arrangement of tubercle, lupus, and leprosy among the granulomata in his primary group of "new growths which are framed on the type of connective tissue."

* At the present time there is a man in Philip Ward, aged 30, who has suffered from lupus for eighteen years, who has had hæmoptysis at intervals since he was seventeen and who now shows the physical signs of phthisis; but such cases are in the writer's experience decidedly rare.

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In 'Ziemssen's Handbook' Neisser places lupus close to tuberculosis of the skin and scrofulodermia, and makes them one division of a group of chronic infectious diseases of the skin, which includes in addition leprosy, syphilis, glanders, rhinoscleroma and frambœsia. He, however, excludes erythematous lupus from the group. Writing in 1883 he admits that no one has established the constant presence of Koch's bacillus tuberculosis in lupus, while Schüller has found only micrococci.

Kaposi and Baumgarten both oppose the recognition of lupus as a tubercular disease. Auspitz and Frederic Lander, Neumann, and other modern dermatologists in Germany admit it, and even Baumgarten allows the possibility of a genetic relation between the two. Plumbe spoke of lupus as a strumous affection. Erasmus Wilson maintained the same relation, Dr Fagge says "it is apt to occur in scrofulous persons," and Dr Liveing that "it belongs rather to the scrofulous diathesis." The late Dr Tilbury Fox "could not subscribe to the view that lupus is an evidence of the strumous diathesis, and was more inclined to regard it as having a predilection for tubercular subjects."*

Clinical course and prognosis.—Lupus is one of the most chronic of diseases. It creeps on, usually with an imperfect attempt at healing, sometimes retreating until it almost disappears, and then again advancing with a persistence and rapidity foreign to its usual character. In the end, if left to itself, it probably heals, leaving, however, indelible marks of its presence in hideous scars, contracted limbs, distorted features, or obliterated orifices. It is singularly free both from pain and from irritation, and never affects internal organs. Whatever its true pathology may be, it does not produce secondary caseous inflammation of the lymph-glands which correspond to the affected skin, and never leads to general tuberculosis of the internal organs. Happily it is amenable to the efficient treatment which has been established within the last twenty or thirty years, so that the prognosis almost entirely depends upon the early recognition of the disease by a skilled practitioner.

Treatment.—Bateman remarks that he knows "no medicine which has been of any essential service in the cure of lupus," and that "it requires the constant assistance of the surgeon." Wilson, in the first edition of his treatise (1842), by a remarkable omission mentions neither the disease nor the name; in the later ones, he recommends caustic applications and a prolonged course of liquor arsen. et hydrarg. iodid., *i. e.* Donovan's solution. The usual practice of the earlier English dermatologists appears to have been to use arsenic and so-called tonics. It was Hebra who, regarding lupus, like most other diseases of the skin, as a purely local lesion, resolutely attacked the diseased tissue, and by destroying it produced a healthy inflammation which ended in cure. The determination with which he carried out this method often led to the most remarkable success. Tilbury Fox introduced the Viennese treatment into England, and maintained that the real treatment of lupus consists in destruction of the diseased tissue by caustics. Even Hardy, though he begins with general treatment of lupus as a scrofulide, admits that in certain cases local measures are also necessary, that emollient applications are unimportant, and stimulating lotions seldom useful. He recommends iodine—one part dissolved in thirty of water with the help of three of iodide of potassium. Even this he admits is useless in most cases,

* On this subject the reader is referred to the summary for and against the tubercular nature of lupus in Dr Payne's recently published 'Manual of General Pathology' (p. 500).

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and recourse must then be had to stronger caustics, as *chloride of zinc*, *potassa fusa*, and particularly *binoxide of mercury*.

Often less severe measures suffice, and Hebra himself accomplished admirable results with the solid *lunar caustic*. A strong solution of the same silver-salt (a drachm to the ounce) may sometimes be substituted with good effect. The acid nitrate of mercury may also be applied, especially to small and comparatively superficial spots.

But the most satisfactory method of treating most cases of ulcerative lupus is by *scraping* with the sharp spoon introduced by Volkmann, of Halle.* Chloroform should be given and the whole of the diseased surface scraped away. It is astonishing how boldly a skilful surgeon can use this instrument or an analogous one, employing enough force to remove all the diseased tissue without injuring the more resistant healthy cutis which surrounds it. Indeed, Hebra's use of the pointed nitrate of silver pencil almost converted it into a scraping or mechanically destructive as well as a chemically destructive agent. The hæmorrhage produced by these operations is less than would be supposed. While it is almost always necessary to repeat the application of a caustic, one advantage of the scraping is that it is sometimes sufficient after a single sitting, and seldom requires more than two or three. The saving of time as well as of pain to the patient is certainly remarkable.

Caustic potash, applied as it used to be in stick, is not only extremely painful, but even with the greatest care will destroy healthy as well as diseased tissue. Hebra's *arsenical paste* is less destructive, but causes great inflammation as well as pain, and is every way inferior to scraping.

The *pyrogallic acid* introduced by Järisch is probably the next best local application. It should be used as an ointment of 10 per cent., which is better than solution or plasters. It causes, however, considerable pain.

Another plan of treatment, also introduced by Volkmann, and carried out by Vidal and Besnier in France, by Mr Squire and Dr Stowers in this country, is *scarification*, or, as the operation is now performed, minute stabs with a lancet, or an instrument made for the purpose. This section of immense numbers of blood-vessels produces temporary hæmorrhage, but afterwards obliteration of their channels and anæmia of the lupus spots.

The *galvanic cautery*, though sometimes applicable and less painful than would have been supposed, has the same drawback as caustic potash and sulphuric acid, that is, it destroys diseased and healthy tissues alike.

Whatever agent be chosen, it is of paramount importance, for the successful treatment of lupus, to recognise its character as a new growth which must be destroyed. So long as any of the granulations remain it is liable to return. Once rooted out, it is rare for this to happen, or even for it to appear in another part of the skin. Occasionally the knife may be employed to excise part of the tip of an ear or some other circumscribed piece of skin; but scraping and caustic, or the two combined, are in a great majority of cases as effectual, and the results are better. Indeed, when early and thoroughly treated, lupus becomes a manageable disease, and the cicatrices which result are often surprisingly slight.

In the more superficial forms of lupus, and especially in the variety to be described as lupus erythematosus, such vigorous means are generally unnecessary, though wherever ulcers or granulations are seen, their destruction

* See his paper on "Lupus and its Treatment," translated for the Sydenham Society in 'German Clinical Lectures,' 1876.

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by some means or other is the only thorough method of cure. The milder applications which have been recommended, such as tincture of iodine, iodoform ointment (half a drachm to an ounce), pyrogallic acid ointment (a drachm to the ounce), and strong solution of nitrate of silver (a drachm to an ounce), may probably stop the disease at an early stage. They certainly check its progress, and may be usefully employed whenever more decisive treatment is counterindicated or postponed. For erythematous lupus especially, pyrogallic acid or iodoform are excellent applications.

Although local treatment is essential for lupus, and is often sufficient without any other methods, many dermatologists strongly recommend the internal administration of *cod-liver oil*. Even Hebra admits its value and used to apply it locally to the sores as well as internally. It is unwise to trust to this remedy without attempting local measures as well; but wherever swollen glands or phthisical symptoms are present, or when want of weight and flabby muscles show malnutrition, oleum morrhuæ ought undoubtedly to be given. Syrup of phosphate of iron, steel wine, or tincture of steel, are indicated by pallor. Arsenic is of doubtful service.

Some of the most scaly forms of lupus are said to be cured by arsenic, but if we cannot recognise such transition forms as are called psoriasis-lupus, we may suspect these cases of being really psoriasis, and not lupus at all, just as serpiginous lupus is often extremely difficult to distinguish from syphilodermia, and owing to this difficulty has sometimes been supposed to be cured by iodide of potassium.

Treatment by inoculation.—Since the present volume of this text-book has been in the press, the preliminary announcement made by Prof. Koch to the International Medical Congress of 1890 (vol. i, p. 1114) has been followed by the publication of his method of treatment of tubercular diseases by injection of a fluid prepared from the products of the specific bacterium. The mode, time, and circumstances of this publication were unfortunate, but the reputation of the discoverer secured respectful consideration for the new plan of treatment. The result of considerable experience appears to be that for cases of phthisis this method of inoculation is sometimes dangerous, often mischievous, rarely beneficial, and, so far as yet known, never curative.

With respect to lupus, Koch's method has stronger evidence in its favour. It has been widely tried in Germany, France, and England, and with the following results. The injection is followed by marked febrile reaction, so far confirming the conclusion above reached, from consideration of its histology and general characters, that lupus is truly tubercular. The bacilli are not killed, but this is of less importance where they are so few. The granulation tissue becomes more vascular, swollen, and painful, as in tuberculous lungs, lymph-glands, and joints. After repeated injections tolerance is established, and the local excitement subsides.

In some few cases the result is decided improvement. In others there is no visible change. What would seem reasonable would be to add scraping to the results of injection, and so get rid of the products of tubercular inflammation in a way that is impossible in the case of the lungs or the joints. Thus inoculation may perhaps be established as a preparatory and auxiliary method to that of destruction by mechanical or chemical means, or as a last resort in obstinate and extensive cases.

But whatever subsidiary place further experience may assign it, the method has scarcely more claim to be regarded as a cure for lupus than for phthisis itself.

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Tuberculous ulceration of the skin.—It may seem contradictory, after the evidence above given, that lupus is itself an ulceration which is accompanied by the characteristic microbe of tubercle, to distinguish another disease which is also ulcerative and also tuberculous. But clinically we are bound to follow the natural history of diseases rather than their mere anatomy, and those which differ in aspect, course, and treatment, demand separate notice.

Lupus, though a deep inflammation, is not exclusively ulcerative; scales, pustules, and other inflammatory products make up much of the diseased structure; and although tuberculous in nature, it is not often associated with other forms of tubercle in the bones, joints, and lungs. But there has long been recognised an ulcer which is distinct in appearance from lupus, which is more like that of syphilis, and which is characteristic of the condition known as tuberculosis of the lymph-glands and joints, the lungs, and the serous membranes.

Tubercular ulceration is usually multiple; the ulcers are rounded, without the thickened border of lupus, often somewhat undermined, not sloughing or phagedænic, but with pale, large, œdematous granulations. They are situated on the face, trunk, or limbs, most often on the latter, and are irregularly placed. They may occasionally be seen on the lips, or about the genital and anal orifices. They are more sensitive than the sores of lupus or of syphilis. They are most often seen in children or very young adults,* and are often accompanied by signs of caries, by caseous glands, or by other tuberculous lesions.

The local appearance, the locality, and concomitants are unlike those of lupus vulgaris or lupus erythematosus. Confusion with acquired or congenital syphilis is easier. But the ulcers are not the result of a sloughing gumma; they have a purplish cyanotic, not a brownish, coppery border; they are more painful, and they are accompanied by the concomitant lesions of tuberculosis, and not of syphilis.

The treatment is chiefly internal, by oleum morrhuæ, steel, good food, and good air, with soothing or gently stimulant local applications.

The only other cutaneous lesions which the writer has seen accompanying the ulceration are—the curious dry condition known as pityriasis tabescentium (p. 846), and pustules, which might be called impetigo or ecthyma, scattered over the limbs. Under the title *Tuberculosis verrucosa cutis*, Riehl and Paltauf described in 1886 an infectious disease derived from handling the skin of tuberculous animals, either dead or alive. It is said to be intermediate between dissection-warts (verruca necrogenica), lupus verrucosus and tuberculous ulceration.

Lupus erythematosus.†—The essential nature of this somewhat rare disease still admits of doubt. There is no question that the sebaceous glands are much affected by it; it is equally certain that a slow chronic dermatitis, accompanied with a violet or rose-tinted erythematous blush, is always present. But it is seldom that one fails to discover evidence of a destruc-

* A remarkable case in an aged woman, reported by Dr van Harlingen, of Philadelphia ('Arch. of Derm.,' April, 1879), may perhaps be regarded as a senile and malignant form of this disease.

† *Synonyms.*—This curious affection was first described by Biett and named *Erythème centrifuge*. It was called by Hebra *Seborrhœa congestiva* (1845), and the same view of its nature has led to the titles *Lupus sebaceus* and *Lupus acnéique* (Hardy). It has also been named *Scrofulide erythémateuse* and *Lupus de Cazenave*. It is, however, more generally recognised by Cazenave's name, Lupus erythematosus (1850).

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tive process of the papillary layer in more or less well-marked cicatrices, and in many instances of undoubted lupus erythematosus the scars are obvious. It is, therefore, still usually associated with the ordinary disease known as lupus, the two forms being distinguished as *lupus vulgaris*, *lupus exedens*, or *lupus exulcerans* on the one hand, and *lupus erythematosus*, *erythematodes*, *sebaceus* or *non-exedens* on the other.* It is, however, very different from lupus vulgaris, in the age of the patients, in its locality and course, and there is no reason to connect this form of disease with tubercle.

The *locality* of this affection is very characteristic. It almost always occupies the face, and usually the bridge of the nose, together with both cheeks; for, in contradistinction to ordinary lupus, it is remarkably symmetrical. The figure produced by this distribution has been compared to a butterfly, a bat, or the sphenoid bone, and when once seen is easily recognised. Lupus erythematosus is also found on the ears, and sometimes on the scalp. The hair is then destroyed, a sufficient proof that lupus erythematosus is not, as it is classed in 'Ziemssen's Cyclopædia,' a superficial dermatitis. It occasionally appears upon the limbs or trunk, sometimes preserving its symmetry, but sometimes being confined to one arm, and most often to the hand. On the trunk and legs it is certainly rare, but in one patient of the writer's it spread over the shoulders and buttocks. Two separate patches are far more often seen than in ordinary lupus, and the disease is much more symmetrical.

It is seldom that we see the first beginning of this disease. It shows itself as an erythematous patch, not unlike that left after impetigo or an early stage of tinea circinata. It spreads at the edge (whence Biett's epithet *centrifuge*), which is marked by injection, swelling, and desquamation, while the centre becomes pale, smooth, and slightly depressed. The sebaceous glands are enlarged, sometimes prominent, resembling acne punctata, sometimes forming black comedones within the affected surface. It thus spreads until it has attained the form and dimensions above described as characteristic. Sometimes, however, fresh spots occur at a distance, and this is decidedly more frequent than with ordinary lupus. The dry, whitish scales, formed chiefly of sebum, suggested the epithet *herpes crétacé* to Devergie, and *seborrhœa congestiva* to Hebra and others.

Its course is extremely slow, and, like ordinary lupus, it is accompanied by neither pain nor itching. It has no claim to be called scrofulous.

On making a microscopic section of the diseased skin, infiltration of the cutis with leucocytes and dilated blood-vessels is obvious; and the congestion and proliferation is most abundant around the sebaceous glands. These cells never become caseous, or soften down so as to form the granulations and pus of an ulcer. They gradually become transformed into connective-tissue corpuscles; and as the fibres thus formed take their place, the papillæ atrophy and the glands shrink and disappear (Neumann, Geddings, Thin). These histological characters appear to show that no sharp line of distinction can be drawn between chronic deep-seated inflammation with hypertrophy and consecutive atrophy on the one hand, and development of such simpler forms of new growth as lupus, tubercle, and syphilis. On the other hand, there is a clearly marked line between deep inflammations with destruction and

* In favour of this view see Mr Hutchinson's 23rd lecture ('On Certain Rare Diseases of the Skin'). For arguments in favour of a more complete severance of lupus erythematosus from true lupus see Kaposi's and Veiel's papers ('Trans. Intern. Med. Congr.,' vol. iii, pp. 162, 167), with comments by Schwimmer and Thin; also Dr Payne's remarks (St Thomas's Hosp. Rep.,' vol. xiii).

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atrophy on the one hand, and superficial inflammations which do not destroy the papillæ and are never followed by ulceration or cicatrices on the other.

Lupus erythematosus occurs chiefly in adults, but sometimes in children. Veiel says that most cases occur between twenty and forty, and the average age of this affection is certainly later than that of ordinary lupus. It is perhaps commoner in women than in men.

Some good observers regard true lupus and this affection as altogether distinct and unrelated both in pathology and in clinical course; but on the whole it appears to the writer that while admitting the difference of type, both are chronic serpiginous inflammations of the deeper layer of the skin, and therefore are related as nearly perhaps as psoriasis and lichen planus.

Lupus erythematosus in some cases simulates nævus. See the account of patients under the care of Mr MacCarthy and Mr Higgins given by Mr Hutchinson in his 'Lectures on Clinical Surgery,' vol. i, p. 284.

The *treatment* of erythematous lupus is that of the milder forms of lupus vulgaris. Alteratives of a stimulant kind take the place of destructive methods. Hebra's diachylon ointment or solution of soft soap (sp. sap. alk.) is sometimes sufficient. Iodide of mercury ointment (one to fifteen) was recommended by Cazenave, and pyrogallic ointment is useful, but the effects of iodoform are often the most satisfactory. In some cases very mild applications can alone be borne, such as unguentum metallorum, yellow oxide of mercury, or unguentum hydrargyri ammoniati. In others, again, the true nature of the disease is shown by the treatment which dermatologists, whatever name they give it, are led to adopt—scarification, and even scraping or the galvanic cautery.

Mr Hutchinson strongly recommends the continued use of an ointment consisting of half a drachm of liquor carbonis detergens to an ounce of petroleum gelatum. The ung. liq. carbonis deterg. of the Guy's Pharmacopœia is stronger, and is also a useful remedy.

Lupus erythematosus disseminatus.—Kaposi has named lupus erythematosus as above described *discoid*, in order to distinguish it from a rare and remarkable form of disease, which he regards, and probably with justice, as a form of lupus. The latter he has named the "disseminated" or "aggregated" variety of lupus erythematosus. Here the patches do not grow by the enlargement of the circumference, but by fresh ones appearing. Moreover the disease is not confined to the face, but is seen upon the trunk, the course is sometimes acute, and the whole character of the disease is far more severe than that of ordinary erythematous lupus, or even of lupus exedens; there is considerable pain, and sometimes synovitis; there is high temperature, nervous symptoms which sometimes end in coma, and in not a few cases the result has been fatal. Cæsar Boeck saw two well-marked cases of this curious disease in Norway.

The acute form is, however, the exception. More often the disease persists with more or less frequent exacerbations, the face appearing as if affected with constant erysipelas. Here also the end is usually death, either from marasmus or from an intercurrent disease.

The writer has only seen one example of this remarkable affection, which occurred in the practice of Dr Cavafy at St George's Hospital. The patient was a woman between thirty and forty; the affection occupied not only the face, head, and neck, but the greater part of the back and trunk. It looked like erythema of a somewhat gyrate form, and there was unquestionable scarring. The patient succumbed to pneumonia.

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Rhinoscleroma.—This uncouth epithet was applied in 1870 by Hebra and Kaposi to a newly recognised form of disease—a hard, smooth infiltration or new growth of the septum of the nose and the adjacent tissues of the alæ nasi and of the upper lip. It has a general resemblance both to lupus and to syphilis, but is said not to be prone to ulceration, a characteristic which would also distinguish it from epithelial cancer. Mr Hutchinson has not seen any case which corresponds with the fourteen or fifteen seen in Vienna, but thinks he has observed cases of lupus which by their unusual hardness and other characters approached rhinoscleroma. He showed a case in an old woman of sixty-eight to the Dermatological Society in April, 1883. A few additional cases have been published in Germany, reference to which will be found at p. 496 of Hans von Hebra's 'Krankhafte Veränd. d. Haut.' The writer has seen very few cases which may have been rhinoscleroma, and only one which was certain. The first occurred before 1870, in a young man at Vienna, which was diagnosed and treated as tertiary syphilis; it consisted in "a thickening and stony-hard induration of the nose," which had lasted for six years when seen. A second case seen a few months later, recorded as one of "syphilitic sclerosis of the nose," not nearly so hard, and accompanied by redness and ulceration, is more doubtful. A third was seen at Guy's Hospital, in November, 1886, in a man of thirty-five; there was dense induration of the upper lip, with surrounding œdema and a little superficial ulceration; it had lasted several years. He also saw the typical and very remarkable case in a patient of Dr Payne's, a young man from South America, which will be found described and figured in the 'Path. Trans.' for 1885. Here the palate and larynx were also affected; and the local ulcerative condition and histological characters were not unlike those of some forms of sarcoma. Dr S. Davies has recorded a well-marked instance from Egypt ('Brit. Med. Journ.,' May 29th, 1886).

The ivory-like induration, the singular locality, and the absence of ulceration separate it from lupus; and Frisch discovered a bacterium not identical with that of lupus ('Ziemssen's Hdbh.,' xiv, 713). Since then Cornil and Alvarez and others have seen it ('Ann. de Derm. et de Syph.,' vi, No. 4, abstracted in the 'Lond. Med. Rec.,' August, 1885, p. 345). It is figured in Dr Payne's 'Manual of General Pathology' (p. 672, and *front.* fig. 1).

The histological characters are not distinctive, for Kaposi found only infiltration of the cutis with very minute leucocytes. Geber recognised giant-cells and spindle-cells ('Arch. f. Derm. u. Syph.,' 1872). Payne found large nidus-cells beside granulation tissue, and "nests" like those of epithelial cancer. In a doubtful case, brought by Mr Morrant Baker before the Pathological Society in 1881, Mr Hutchinson, Dr Cavafy, and the writer were appointed a committee, and drew up a report, which will be found at p. 262 of the 'Transactions' for that year. A figure is given at p. 458 of 'Ziemssen's Handbuch' by Schwimmer and Babes.

Rhinoscleroma has returned after removal in cases reported from Germany and from Italy; but is said to have been favourably influenced by salicylic acid, applied in the belief that it would act as a germicide.

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LEPROSY*

> "Est Elephas morbus qui propter flumina Nili
> Gignitur Egypto in media, neque præterea usquam."
> LUCRETIUS, *De Rerum Nat.*, lib. vi, 1112.

History and terminology—Geographical distribution—Anatomical lesions and course—Histology—The bacillus lepræ—Symptoms and event—Ætiology—Treatment—Other exotic diseases—Frambœsia—Acrodynia, etc.

THIS disease, interesting from an historical point of view, is still of practical importance in many parts of the world; but we have only space here for a very brief account of it, referring the reader for further information to the elaborate article by Kaposi in Hebra's great work, and to Dr Liveing's Gulstonian Lectures for 1873.

Nomenclature.—The names given to the disease by the Greeks were *lepra* and *elephantiasis*; it was divided into *alphos*, *melas*, and *leukos*.

Celsus, however, who describes *alphos*, *melas*, and *leuce* as species of *Vitiligo* (lib. v, cap. xxviii, § 19), portrays leprosy separately and distinctly as a disease affecting the bones and the whole body, almost unknown in Italy, "*quem ἐλεφαντίασιν Græci vocant*" (lib. iii, cap. xxv).

The term applied by Willan and Bateman to leprosy was "elephantiasis Græcorum," while they unfortunately used "lepra" for part of the innocent, white scaly disease which the ancients would possibly have recognised as *alphos*, but which all modern dermatologists call psoriasis.

No doubt many other cutaneous affections, obstinate chromic eczema, syphilis, lupus, and perhaps psoriasis, were confounded with leprosy in ancient times; but there is no question that one and the same destructive form of disease has existed in Palestine under the Mosaic law, in Western Europe during the Middle Ages, and at the present day in many parts of the globe; and this is best named by its historical title, leprosy.

Leprosy appears to have been rare in ancient Greece, and it seems to be not quite certain that the Septuagint translators were correct in rendering *zaraath* of the Hebrew Scriptures by the Greek word λέπρα. The latter term, however, is universally applied to leprosy in the New Testament. It refers to the scaly surface often seen. The Arabic name of true leprosy, according to Dr Greenhill, is *Judzam* (= *lepra Arabum*). *Barat* (= *leuce* = *vitiligo*), or "white leprosy," is nothing but leucodermia. In the Middle

* *Synonyms.*—Lepra vera—Lepra Arabum—Elephantiasis Græcorum—Leontiasis—Satyriasis—Morbus Herculeus.—*Fr.* La lèpre.—*Germ.* Aussatz.

It must be remembered that the terms Elephas and Elephantiasis do not refer to rough skin or huge and shapeless limbs, but to the magnitude of the disease. "Elephantiasis a magnitudine et diuturnitate nomen accepit" (Aëtius).

> "Est lepræ species elephantiasisque vocatur,
> Quæ cunctis morbis major sic esse videtur,
> Ut major cunctis elephas animantibus extat" (Macer Floridus, 1160).

Aretæus says it is called elephas, partly because it is unlike anything else, partly because it is black and terrible, and partly because the skin is rough and cracked ('De Morb. Chron.,' lib. ii, cap. xiii).

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Ages leprosy was known to the school of Salerno as *mal morto* and *mal di San Lazaro.*

Distribution.—Norway is the only European country in which leprosy is still common; there were 900 lepers in the asylums at Bergen and Molde in 1884; it is there known as *Spedalskhed.* It is also found here and there in Sicily and in Malta (28 lepers were reported in the latter island in 1886), in certain parts of Portugal, in the Levant, in the Crimea, and at Astrakan; it is more common in Syria, Arabia (Palgrave), Persia, Bengal, S. India, Burma, and Siam; in Japan and in China, where it is said to have been known for ages; in Egypt, Nubia, the Soudan, the Cape Colony (where it coexists with elephantiasis Arabum), and most parts of the African coast (though apparently it is rare in the interior); in Madagascar* and the Mauritius, St Helena, the Canary Islands, and the Azores; in New Brunswick, Mexico, and the West Indies (especially Trinidad), Central America, Ecuador, British Guiana and Surinam, Bahia, and the coast of Brazil; in New Zealand, the Sandwich Islands, and some other parts of the Pacific.†

Accounts of the disease from many of these places will be found in a report on leprosy by the College of Physicians prepared for the Colonial Office, and issued as a blue-book in 1867. In 1874 Dr Vandyke Carter published an official report upon leprosy in India, and within the last six or eight years Dr Beaven Rake has contributed valuable clinical and pathological papers on the subject from Trinidad.

Unhappily, while leprosy has receded from civilised Europe during the last four hundred years, there is reason to fear that it is reappearing in new countries. Thus it has been introduced within historical times into the Sandwich Islands, and quite recently cases have been reported from various parts of the United States, and from Australia. In Minnesota it appears to have been introduced by Norwegian immigrants, in New South Wales by Chinese labourers; but this explanation of its spread, whether by contact or inheritance, does not apply to certain isolated cases observed. For instance, Dr George Dock sent the writer in 1889 a careful account of two cases, one in a German, the other in an Alsatian, which occurred at Galveston on the coast of Texas, neither of whom had had intercourse with lepers from Mexico, China, or South America. It had not spread to the wives or families of either of these patients.

Varieties.—Leprosy is essentially one and the same disease, but one of two forms is usually predominant—the *nodular* or "tubercular," and the *anæsthetic.* The two, however, are often combined. Either may be preceded or accompanied by pigment spots, which have led to a third species being formed—*lepra maculosa.* All end in an ulcerative stage, and all may lead to loss of members—*lepra mutilans.* "Black leprosy" is the only genuine form; "white leprosy" is not leprosy at all, but leucodermia.

Description.—The disease begins insidiously, usually as an erythematous redness, but in some cases with an outbreak of bullæ resembling those of pemphigus. There follows the appearance of red or violet patches, varying from a finger-nail to the palm of the hand in size, which gradually become darker in colour. At the same places, or independently, appear flat, firm,

* See an interesting account of the disease founded on more than 100 cases seen at Antananarivo, by Dr Andrew Davidson ('Edin. Med. Journ.,' July, 1864).

† See Dr Ransome's maps and statistics, 'Brit. Med. Journ.,' March 1st, 1890; and Dr P. S. Abraham's pamphlet with a map (Epidemiological Soc., 1889).

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raised nodules, consisting of an infiltration of the deeper parts of the skin. The lymph-glands at the same time enlarge. These nodules of tubercular leprosy may shrink and be absorbed, leaving atrophied and sometimes pigmented spots; but more often they soften and ulcerate. The leprous ulcers secrete but little pus, and show few and feeble granulations. They slowly increase both in extent and depth.

Distribution.—The leprous patches usually appear first upon the limbs, and afterwards on the trunk and face. When fully developed in the face the disease produces a singular deformity, which the ancients described as *leontiasis* and *satyriasis*, and which, once seen, is never forgotten. The disease also affects the neck, shoulders, back, chest, and abdomen, but is most frequent in the extremities, especially on the extensor surface. Nodules occasionally occur, even upon the palm and sole. The hands and feet are swollen and distorted, with thickened and rough skin; the ulcers burrow deeply, and affect tendons, bones, and fibrous tissues, until at last toes, fingers, or the entire hand or foot undergo gradual necrosis and fall off.

Some of the mucous membranes are also affected, particularly those of the mouth, nostrils, and larynx, and even the conjunctivæ.

Moreover, the disease involves the great nerve-trunks, where the leprous nodules can often be felt during life.

Histology.—Careful microscopical investigations by Virchow, Thoma, and others showed that the disease consists in infiltration of the deepest layers of the cutis with granulation tissue. Leprosy was therefore classed by Virchow in proximity to lupus, from which, however, it is widely separated by its clinical course, geographical distribution, and natural history.

A bacillus was discovered by Hansen, of Bergen, in 1874, which he described and figured in the 'Quart. Journ. of Micr. Sci.' for 1880 (vol. xx, p. 92).* These microphyta appear constantly in leprous nodules, and in great abundance. The *bacillus lepræ* is 5 μ long, very slender, and immobile. It stains like the bacillus of lupus (see Crookshank's 'Bacteriology,' pl. 23). Attempts at cultivation have hitherto failed.†

Course.—Leprosy is extremely slow in its progress, and it resembles syphilis and lupus in producing but little pain. Patches of anæsthesia are sometimes found, and may be followed by ulceration before tubercles appear. It is said that in rare instances hyperæsthesia precedes or takes the place of loss of sensibility. The anæsthetic spots usually show some amount of atrophy, and the hairs of those parts are small and deficient in colour.

While this terrible disease goes on its course, interrupted from time to time by temporary improvement and healing of the ulcers, but never more than checked, the general condition of the patient is wonderfully little affected. Even perspiration takes place very much as usual. The hair, however, is gradually lost, not only that of the scalp, but also the beard, eyebrows, and eyelashes. There is no fever, the temperature is usually subnormal, and the patient suffers much from cold. The pulse is slow, and the appetite and organic functions, including the quality of the urine, are

* They have since been found by Doutrelepont, Neisser, Cornil and Babes, Köbner, Dr Hillis ('Path. Trans.,' 1883, pl. xxii), Dr Thin ('Med.-Chir. Trans.,' vol. lxvi), Dr L. J. Steven, of Glasgow ('Brit. Med. Journ.,' ii, 1885), Drs Klein and Gibbes, Dr Rake ('Path. Trans.,' 1887), and in fact all modern observers. Köbner tried inoculation unsuccessfully.

† Dr Rake informs the writer by letter from Simla (June, 1891) that he and Dr Buckmaster have succeeded in cultivating the bacillus of leprosy in blister-serum; and are now trying inoculation on animals with the pure culture.

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very little altered. There appears to be no foundation whatever for the assertion of the ancient physicians that the sexual instinct is increased: perhaps the name *satyriasis*, first applied to the distorted and hideous features of the sufferer, was afterwards misinterpreted.

Death seldom occurs directly from leprosy, for there is neither excessive pain nor hæmorrhage nor invasion of vital organs to cause it; but when once fallen into a condition of anæmia and marasmus, the miserable leper is cut off by some intercurrent affection—pleurisy, pneumonia, dysentery, or Bright's disease, all of which have been recorded by the Norwegian pathologists, Boeck and Danielssen, but none with sufficient frequency to show more than an accidental connection with leprosy. The patients become dropsical, in the later stages; and they are often cut off by phthisis, in which the leprous bacilli appear to take the place of those of tubercle.

Ætiology.—The essential cause of leprosy is entirely unknown. It has probably existed from the earliest times, and has only disappeared from civilized Europe within the last 400 years. We may hope that it is in slow but steady process of extinction in other regions.

Notwithstanding the presence of the *bacillus lepræ*, the disease is, under its usual conditions, non-contagious; it is not transmissible by living in the same house, by contact, or even by sexual intercourse. It is, however, possible that contact of actually ulcerating leprous nodules with a fissured skin or mucous membrane might produce the disease, and there is reason to believe that a contagious quality is more marked when the disease is newly introduced. Dr Rake has been unsuccessful in his attempts to propagate it in animals by inoculation. A case of supposed inoculation in a man in 1886, at Honolulu, appears to be inconclusive. Dr Gairdner has lately published a case which seems to show that leprosy may be inoculated by vaccination ('Brit. Med. Journ.,' February 5th and June 11th, 1887).

Whether or not it is under any circumstances contagious, leprosy is undoubtedly though not exclusively *hereditary*, and its occurrence in persons of pure European parentage is excessively rare. Patients in England are usually either half-castes or persons who were born and lived in India, and one of whose parents was perhaps of mixed blood. Moreover the Norwegian emigrants to Wisconsin and Minnesota included 160 lepers, but the disease was found to have died out among them by Dr Hansen, of Bergen ('Arch. f. Derm. u. Syph.,' 1889). In cases of probable infection the incubation period is successively prolonged to nine years or more.

It is doubtful whether leprosy has any predilection for castes or races as such, although at the present day it is, as above stated, almost confined to certain of the dark races of mankind, and where prevalent is rare among the well-fed and well-cared-for classes.

Mr Hutchinson has suggested that leprosy depends upon eating fish, probably fish in a state of decomposition. This view certainly agrees with its presence not only on the sea-coast, but also in the neighbourhood of great rivers and inland lakes; and it also accords with the large consumption of salt fish in the Middle Ages, when it formed the principal animal food throughout the winter. The disease, however, does not appear in many parts where fish, both fresh and putrid, is eaten, and it is prevalent in certain districts where fish do not form an article of diet. See Dr Abraham's paper in the 'Practitioner' (1889).

Leprosy appears to be somewhat more common in men than in women—in Bombay, according to Dr Carter, very much so. It usually begins about

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the time of puberty or in young adults. No congenital case appears to have been recorded.

Treatment.—This is unfortunately almost hopeless, and we must rather look to the gradual rooting out of the disease by improved conditions of life than to therapeutics. Various drugs have been vaunted from time to time as specifics, but have all in turn been discredited. Cod-liver oil is the only internal remedy which can be said to do more than alleviate symptoms. Externally Gurjun and Chaulmoogra oils have been supposed to be valuable. The writer has tried the former in three cases with no benefit. Dr Liveing's much larger experience makes it probable that the latter is sometimes of service. Dr Rake depends more on excision and scraping of the leprous nodules than on any other external treatment.

Injections with Koch's fluid have been tried in cases of leprosy by Babes in France, and by Drs Colcott Fox and Abraham in England. The results do not appear to be so far encouraging.

Mr Hutchinson has recorded a case of gradual spontaneous recovery ('Med.-Chir. Trans.,' lxii, p. 331); and the writer has had a somewhat similar case, which is still under his care, of a man who developed leprosy after living in Brazil, and is now very much better in England. But temporary quiescence of the disease is very common.

Leprosy is the only exotic disease of the skin of practical importance to practitioners in England.

Frambœsia or Yaws, apparently a contagious malady, and by some authors believed to be nothing but Syphilodermia, was known to Bateman, and is described at length by Kaposi in Hebra's 'Handbook.' It is endemic on the West Coast of Africa, and appears to be identical with what is known as *Pian* in Java. Less clear is its relation to *Parangi*, a cutaneous disease, endemic in Ceylon, and to *Verrugas* in Peru.

Radesyge in Norway is, according to Hebra, lupus. *Sibbens*, the old Scottish name of a disease of the skin, was syphilis.

Aleppo evil, the Oriental sore, known also as *bouton d'Alep* or *de Biskra*, and the Penjdeh or *Delhi boil*, has been ascribed to syphilis, but without proof. It is now said to be dependent on a special bacterium and capable of inoculation in animals (Heidenreich, 'Centrbltt. f. Bact. u. Paras. kunde,' January 25th, 1889).

Pellagra, an epidemic erythema, was first observed in Lombardy, and connected with eating diseased maize; it is probably identical with *Acrodynia*, described by Alibert as epidemic in Paris during 1828 and 1829. Winternitz ("Eine klinische Studie ü. das Pellagra," 'Vierteljahresschrift f. Derm. u. Syph.,' 1876) doubts the existence of the former. Acrodynia seems to be endemic in the Levant (Behrend, 'Hautkrankheiten,' pp. 154, 156).

Ringworm appears in peculiar forms in certain foreign countries. Burmese ringworm has been already referred to (p. 866); and Dr Anderson has published an interesting account, with figures, of *Tinea imbricata* from Tokelau, in the South Seas ('Edin. Med. Journ.,' Sept., 1880).

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TUMOURS OF THE SKIN

"Il y a des blessures, dont . . . . la cicatrice reste."
VOLTAIRE, *Tancred.*

Cheloid—Terminology and history—Appearance, course, and symptoms—Histology—Relation to scars—Distribution—Prognosis and treatment.
Multiple fibroma—Distinction from molluscum sebaceum—Anatomy and distribution of the tumours—Their course and treatment—Neuroma—Myoma.
Angioma or vascular nævus—Elephantiasis teleangiectodes—Lymphangioma.
Mycosis fungoides—Kaposi's xerodermia maligna—Carcinoma, sarcoma, and rodent ulcer of the skin.

PASSING from the deep intractable ulcerations, combined with hypertrophic or neoplastic processes, which constitute lupus and leprosy, we now come to the new growths or tumours of the skin in a more restricted sense.

The relation between deep and chronic inflammation of the skin, hypertrophy, and new growth is so close, that lupus, tertiary syphilis, and leprosy might be classed either as deep destructive forms of dermatitisor as cutaneous granulomata; while warts and condylomata, gutta rosea, xanthelasma, and elephantiasis are as much new growths as inflammations. But we have now to treat of neoplasms which are neither hypertrophies nor inflammations.

As in other parts of the body, the tumours of the skin are clinically "innocent," "malignant," or "semi-malignant;" while anatomically they are distinguished as "homologous" or "heterologous" (cf. vol. i, p. 71).

CHELOID.—In the 'Arbre des Dermatoses' of Alibert appears, among many other fantastic names, a new term for what was an undescribed disease —*Keloide.* The etymology of the word was long a puzzle. It was supposed by some to be derived from κηλίς, a mark; by others from κήλη, a tumour. Being taken by Addison in the former sense—*quasi ustione facta macula*—as meaning a scar from a burn, it was transferred to the curious affection still known as "Addison's keloid," but better named morphœa or circumscribed sclerodermia, described above (p. 888). It is now certain, from the researches of Dr Fagge, that Alibert meant by the word "kéloide" to denote the claw-like offshoots which characterise the disease in question, and intended to derive it from χηλή, a crab's claw. The right spelling is now generally used, and Alibert's is recognised as the only "true" cheloid.

Alibert described it as "cancroïde," and Bazin and other French dermatologists have hence called it malignant, and regarded it as closely allied to epithelioma of the skin; but it is not improbable that by "cancroïde" Alibert did not mean "cancer-like," but "crab-like;" at all events, it is not a cancroid tumour in the modern sense of the word.

Biett and Lebert afterwards published cases; Addison gave an account of Alibert's disease in the 'Med.-Chir. Trans.' for 1854 (reprinted in his 'Collected Works'), and a paper by Dieburg appeared in the 'Deutsche

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Klinik' for 1852, No. 33. The 'Clinical Transactions' for 1880 contain a remarkable case of multiple cheloid of the face following smallpox by Dr Goodhart, and a commentary by a committee on the subject.

The affection is a rare one. It begins as a pink, smooth, slightly raised, flat nodule, which increases in extent without becoming relatively more prominent. It is remarkably firm in feel. The centre becomes paler, and is sometimes depressed, and the raised edges are surrounded by a slight erythematous border; the epidermis is completely adherent; it is in and not under the skin. Sometimes, however, especially in the later stages, it spreads to the subcutaneous tissue and forms adhesions to the deeper parts, but it never invades more than the integument.

The most characteristic part of the disease is the presence of radiating bands, which appear after a time, run across the original nodule, and afterwards project from its edge. These undergo contraction in the same way as the cicatrices of a wound, and the whole tumour is sometimes puckered and deformed by this process. In the earlier period the nodule might pass for an hypertrophied scar; in the later stages it still more closely resembles a large indurated and contracted cicatrix, as from a deep burn or a syphilitic ulcer or a carbuncle.

The tumour is usually single, but two or more may exist on the same patient, as in Dr Goodhart's case, and in a young man under the writer's care in 1888. The disease is of very slow growth. It occurs most often in young adults of either sex. From the commencement it is usually attended with pricking and itching, with a sense of constriction, and sometimes there are severe stabbing pains. It is almost always tender to the touch, but sometimes is quite free from pain.

Histology.—Microscopic sections show that the epidermis is thin, but otherwise unaffected; the papillæ are destroyed, and the cutis vera and subcutaneous tissue occupied by bands of dense fibrous tissue, which are quite indistinguishable from those of a true scar. As in all cicatrices, the sweat-glands, hair-sacs, and sebaceous sacs are destroyed in the process. Dr Warren, of Boston, published a valuable histological account of cheloid in the Transactions of the 'k. k. Acad. d. Wissensch.,' Vienna, March, 1868. See also that by Babes in 'Ziemssen's Handbuch,' xiv, p. 434.

Diagnosis.—Neither in the histology nor in the symptoms does there seem to be any obvious distinction between a cheloid tumour and an hypertrophied and painful scar. Hebra, in fact, defines cheloid as an idiopathic or primary cicatrix. Others have maintained that all cheloid tumours are hypertrophied scars, and undoubtedly they often arise from ordinary cicatrices, or from the slight marks left after leech-bites or acne pustules. It commonly occurs upon the shoulders, where acne cicatrices are usually the deepest and most extensive.*

Dr Robert Liveing, while admitting that cheloid growths often begin in scars, finds the distinction between them and hypertrophied cicatrices in two points: first, that the bands of fibrous tissue in cheloid run in definite parallel or radiating bundles, whereas those of cicatrix form an irregular network; secondly, that the cheloid growth invades healthy tissues, which hypertrophied scars never do, and that this is the case even when cheloid appears in a previous scar. The new growth can be distinguished as it invades the old cicatricial tissue.

* Such cheloid growths originating in acne scars must not be confounded with acne-cheloid which is a synonym of Hebra's sycosis frambœsiformis or sycosis capillitii (p. 852).

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Locality.—Cheloid tumours occur most frequently in the skin over the sternum. They have also been observed on the abdomen, neck, shoulders, arms, and face. They are usually single, and very rarely more than two in number, except in the case of cicatricial, so-called false, cheloid, when the new growth may appear in as many scars as were originally present.

We had once in hospital a well-marked case of cheloid affecting the pubes in a patient of Mr Bryant—a man who probably had never had ulceration, syphilitic or other, of this part.

Nature.—Pathologically cheloid is not a mere hypertrophy nor a granuloma, but a fibro-cellular new growth, a true sarcoma, sometimes consisting chiefly of spindle-cells, sometimes more exclusively of fibres. It has two characteristic marks of sarcoma apart from its histology: it is very apt to return again and again after removal, while, on the other hand, it does not reappear in the neighbouring lymph-glands or in the viscera.

A series of excellent models of cheloid, Nos. 454 to 466, were made by Mr Towne for the Guy's Hospital Museum.

Traumatic, or false cheloid, is a hypertrophied scar ("die warzige Narbengeschwulst" of Dieburg). It occurs whenever a burn, ulcer, or other injury produces a scar which hypertrophies and becomes painful.

Prognosis and treatment.—Trustworthy observers have recorded the spontaneous disappearance of cheloid tumours, but this must be extremely rare. They seldom or never ulcerate. They grow slowly, and appear not to menace life, but the pain they occasion is sometimes severe.

Unfortunately, if removed by the knife, by galvano-cautery, or by caustics, the tumours almost always return. Nor have any of the milder applications which have been tried produced absorption. Mr Hutchinson, however, in an interesting paper on the subject ('Medical Times,' May 23rd, 1885), has recorded exceptional cases in which operation proved successful.

FIBROMA.*—This affection, named *molluscum* by Willan, differs altogether from molluscum contagiosum treated of above (p. 848), except in the fact that they both consist of multiple pedunculated tumours. Those of fibroma are not cystic growths, they are not glandular, and they have none of the histological characters of contagious molluscum. They are soft and painless, the skin over them is unaffected, they are more or less pedunculated, they vary in size from a pea to a marble or a fist, and when cut into they show œdematous, inelastic connective tissue. They resemble, both in appearance and structure, the firmer kinds of polypi of the nasal fossæ, the colon, rectum, uterus, and other parts of the mucous membranes. They might, in fact, be well termed "multiple cutaneous fibroma" or "multiple fibrous polypi of the skin."

The number of these tumours is sometimes almost innumerable, as is seen in the well-known case of Virchow which forms the frontispiece to his work on morbid growths ('Kr. Geschw.,' Bd. i, S. 325). Their size varies from a pin's head to a foot or more in diameter.

The celebrated case of Tilesius, of Leipzig, published in 1793, was named molluscum by Willan from the soft fleshy character of the tumours (*corpus tectum est verrucis mollibus sive molluscis*). Bateman recognised that

* *Synonyms.*—Molluscum fibrosum, areolo-fibrosum, non-contagiosum, simplex, pendulum —Fibroma molluscum (Virchow). Esmarch's 'Elephantiasis' (1885) records and figures several remarkable cases. See also Dr Sangster's case with drawings and references in the 'Clin. Trans.' for 1880. The writer recorded seven cases in the 'Guy's Hosp. Rep.' for 1889, p. 388.

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these were not glandular, and were quite distinct from the molluscum contagiosum described by himself. The skin of Rheinhard, the Mühlberg peasant who came under the notice of Tilesius, is still preserved in the museum of Leipzig.

Dr Fagge believed that these tumours begin in the outer sheath of the hair-follicles and sebaceous glands; and in one case he found an enlarged sacculated gland occupying the interior of one of the growths ('Med.-Chir. Trans.,' 1870, vol. liii, pl. vi). This has, however, not been again observed, and the occurrence of similar tumours in the palm and sole seems to prove that the coincidence was accidental.

There appears to be little local predilection for these fibrous polypi. We sometimes see a single one on the face or elsewhere, or they may cover the face, the trunk, and the limbs. They also sometimes appear on the prolabium and the palate, as in Dr Fagge's case, which was also figured in plate xviii of the Sydenham Society's 'Atlas,' and modelled for the Guy's Hospital Museum (No. 497).

Cutaneous fibromata occasion no pain, and single ones may be met with in perfectly healthy persons, to whom they cause no inconvenience. Some of these are congenital, but the typical multiple fibromata are certainly not so. They usually appear in childhood. Multiple fibromata are certainly rare, and probably few cases have failed of being recorded; but one or two polypi are not infrequently seen if looked for.

When they have attained their full growth they undergo no further change, and neither degenerate nor become absorbed. But, as Mr Hutchinson has pointed out, they sometimes lose their firm, fleshy feel, and become flaccid, so as to feel almost like empty cysts. (See the 16th of his Clinical Lectures "On Rare Diseases of the Skin.")

Virchow has recorded a case in which the father, grandfather, and brother of a patient were all affected with multiple fibromata of the skin.

According to Hebra, when they are numerous the patient is usually ill developed in mind and body. But this is certainly not always the case.

The only treatment is removal by scissors or the knife. The polypi show no tendency to return.

Large and numerous fibromata are sometimes associated, in a condition of elephantiasis and œdema, with considerable mobility of the skin (*dermatolysis*: p. 893). Such was the case in Virchow's patient and in the unfortunate person shown to the Pathological Society by Mr Treves (in 1885) as "the elephant man."

Neuromata are multiple, painful fibrous tumours of the nerve-trunks, scattered over both trunk and limbs. They have been long known, and have formed the subject of a monograph by von Recklinghausen ('Ueber die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen,' 1882). Except in the pain which accompanies them, these tumours are indistinguishable from ordinary fibromata. Dr Duhring has described some severe cases marked by paroxysms of neuralgia.

Myoma.—Tumours of unstriped muscular fibre (*liomyomata*) have been described by Virchow ('Archiv,' vols. iii and vi), Klebs, Axel-Key; Rindfleisch, and Besnier ('Annales de Dermatologie,' 1880). They probably take their origin in the muscular bands connected with the hair-sacs. They are of no clinical significance.

Lipomata, or true fatty tumours, never affect the skin itself, but are always subcutaneous.

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GLANDULAR TUMOURS.—*Colloid milium** was the name given by Ernst Wagner in 1866 to an exceedingly rare affection of the skin, which consists in the appearance of a multitude of raised, yellowish, glistening nodules like those of milium (p. 845). They occur in groups, and vary in size from a pin's head to a split pea. They do not contain sweat, but a thick gelatinous secretion.

These nodules are probably new growths, beginning in the sebaceous, not the sudoriparous glands. Their histology is described by Dr Philipson in the 'British Journ. of Derm.' (vol. iii, p. 36), who considers them as being epitheliomata with colloid degeneration. They occur about the eyes, where they look like xanthelasma, but are also seen on other parts of the face. In one case of Dr Liveing's the arm was affected, and in two reported by French writers the whole of the front of the chest was covered. The few patients yet observed have been adults.

The disfigurement has been removed by scraping out the little tumours one by one.

Adenoma sebaceum.—Under this title Dr Balzer published two cases of multiple tumours of the face ('Arch. de Physiologie,' 1885, No. 7, and 1886, No. 5), which were modelled for the museum of St Louis. Dr Pringle has added a third case, which, like the others, occurred on the face of a young woman ('Brit. Journ. of Derm.,' Jan., 1890). The figure he gives has a rough resemblance to acne, but there are no comedones and no suppurating points. The papules or nodules are firm, whitish, and like grains of sago in size and shape. Some look like milium, others more like multiple warts. When pricked inspissated sebum can be squeezed out. Along with these sebaceous tumours are numerous dilated vessels, which, however, disappear under treatment. Microscopical observation shows hypertrophy of the sebaceous glands and of the deep layer of the cutis. The little tumours appear to undergo spontaneous involution.

VASCULAR TUMOUR.†—Excluding moles or pigment-spots, true or vascular nævi have always essentially the same structure. But they vary in appearance, from the smooth, flat, "port-wine stains," as they are called, which sometimes cover the greater part of the face, head, or even trunk, to the circumscribed pulsating tumour-like mass which can be removed by ligature, galvano-cautery, or other mechanical means.

Of similar structure though different pathology are the *stigmata* of gutta rosea and erythematous lupus, and the permanently injected patches which sometimes accompany the cicatrisation of lupus exedens, syphilis, or any other deep form of dermatitis.

There are, however, some rare and remarkable forms of disease of the skin which, though anatomically angioma, differ from true nævi not only in being acquired instead of congenital, but also in their course and event. Sometimes they will, as described by Hebra, while spreading in some directions, return to a normal condition in others; or, again, they may acquire a tumour-like and semi-malignant character, growing rapidly and forming large masses of erectile tissue. They are most often seen upon the extremities, though even here they are happily rare. They are sometimes complicated with fibrous growths, which have not only the pain of neuroma, but also its

* *Synonyms.*—Colloid degeneration of the derma (Besnier)—Hydradenoma (Darier)—Syringo-cystadenoma (Török).

† *Synonyms.*—Nævus flammeus—Nævi vasculares—Angiomata—Mother's marks.

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histological characters. Bruns recorded such cases on the lower extremities as *elephantiasis neuromatosa.*

Virchow and Kaposi described, under the somewhat similar title of *elephantiasis teleangiectodes*, multiple fibro-vascular growths, which begin as separate lobulated tumours, but afterwards form diffuse, vascular thickenings of the skin.

Apparently identical with these is a case in a child under Dr West, which was examined by Dr Liveing and figured by the late Dr Tilbury Fox under the name of *fibroma fungoides* (pp. 352—354 of his work on 'Skin Diseases'). He there described other cases of fibro-vascular ulcerating growths which he considered to be of the same nature. One of those, however, may probably have been syphilitic.

LYMPHANGIOMA.—A curious affection of the skin, which has been described under this name, consists in what looks like a group of vesicles; but, on careful examination, they are found to be more deeply seated than usual, and in the event prove not to be inflammatory at all, but new formations, lasting unchanged for an indefinite period. In one case of the writer's they strikingly resembled the vesicles of zona, appearing in several groups, and arranged in a tolerably regular line. In this instance the affection was complicated by appearing upon a large congenital port-wine stain, and the result was that many of the lymph-cysts became pink by admixture of their contents with blood, and when accidentally ruptured, thick red or black scabs were formed. This coincidence with ordinary vascular nævi (which others also have noticed) as well as histological investigation, seem to prove that the disease is rightly regarded as analogous to acquired vascular nævi. But Mr Hutchinson has described the affection under the unfortunate name of "lupus lymphaticus" ('Path. Trans.,' 1880, with fig.). Several cases have been brought before the Dermatological Society within the last few years. A careful histological description with figures by Mr Stewart will also be found in the 'Path. Trans.' for 1875, in the volume for 1879 (xxx, 474) by Drs T. and T. C. Fox, and in that for the following year (xxxi, p. 346) by Dr Sangster. Kaposi described a remarkable case of lymphangioma in a woman twenty-two years old who had several hundred violet-red pimples, round or oval in shape, some of them as small as a lentil, situated in the cutis and somewhat resembling certain forms of syphilodermia. A minute portion, being excised, showed that the cutis was filled with dilated lymph-spaces lined with endothelium. He named it *lymphangioma tuberosum multiplex.* A useful collection of cases was published by Dr F. A. Noyes, of Melbourne, in the 'Brit. Journ. of Derm.,' Dec., 1890, and Dr Török added a critical and pathological account of the condition (*ibid.*, Jan., 1891).

MYCOSIS FUNGOIDES.*—Under this name Alibert described certain mulberry-masses of ulceration which he supposed to be syphilitic. Bazin in 1851 met with a case which resembled Alibert's account, and invented a *diathèse fongoïde* to explain it. In 1869 Ranvier showed that these curious soft tumours, readily ulcerating into fungous papillary masses, showed histologically a series of lymphatic spaces with cytogenic tissue between (Gilot 'Thèse

* *Synonyms.*—Plan fongoïde (Alibert)—Eczema tuberculatum (E. Wilson)—Lichen hypertrophicus (Hardy); who, however, now recognises its character as a new growth and accepts the title: Lymphadénie cutanée—Papilloma arco-elevatum (Beigel)—Granuloma fungoides—Sarcoma lymphadenoides (Auspitz)—Papillome étalé ou en plaque (Charpy).

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de Paris,' 1862). Auspitz has since discovered a micrococcus in these tumours which he thinks may be characteristic.

Dr Payne ('Path. Trans.,' vol. xxxviii, and 'Rare Diseases of the Skin,' p. 9) has described and figured a remarkable case of granuloma fungoides; and, after discussing the lymphatic and the bacillary theories of the disease, concludes that neither of them is adequately supported by facts. In this, as in the writer's case mentioned below, cultivation failed to demonstrate the presence of a specific microbe.

It is a very rare affection. It has been observed by Landouzy in an infant, but most cases recorded have been in adults, both men and women. The position of the tumours is usually on the trunk, less often on the face or limbs. It begins like patches of chronic eczema, with considerable itching, and after a variable period there follows deep induration and thickening of the cutis, with formation of separate raised swellings. These continue long stationary, sometimes shrivel again, but more often ulcerate and form the fungating tumours which are most characteristic of the disease. They exude a great quantity of clear, watery, colourless serum. The progress is very slow, but with one or two exceptions the result is fatal. No treatment is known to be of service.

The disease resembles both syphilis and leprosy, and, in its early stages, eczema and some forms of lichen. It has a remarkable resemblance, in its developed stage, to the worst form of iodide eruption (p. 835; see Mr Hutchinson's figure in his 'Archives of Surgery,' pls. iii and iv).

We had lately a well-marked example of this disease in Philip ward, which was shown at the Dermatological Society. The patient was a man of sixty-six, who first developed what looked like ordinary dry eczema; and then fungating masses of granulations formed on the various parts of the body, the neck, shoulder, loins, and leg. Some of these sloughed, suppurated, and healed completely, but others formed, and at last one enormous mass on the back. He died from pulmonary gangrene. Sections of the tumour looked like those of a round-celled sarcoma; and there was a similar new growth in one adrenal.

It is doubtful whether the disease should be classed with "granulomata" or with malignant growths. It is clinically and anatomically distinct from the multiple cutaneous sarcomata described in the first volume (p. 83).

XERODERMIA MALIGNA.*—Perhaps the most remarkable of all cutaneous diseases, allied to nævi in its early stages and markedly malignant in its later development, is a rare affection first described by Kaposi in 1870 under the borrowed title of *xeroderma*, a name which had been previously applied to a totally different condition by Wilson (p. 886).

It begins with spots of erythematous appearance not unlike those of measles. Then they fade and form pigment-spots like freckles, with dilated venules. The next stage may be months or years in appearing. When it arrives, the apparent ephelides become atrophic, the skin dry, thin, and wrinkled. Then it gradually contracts, so as to form a smooth, tightly drawn surface, which may evert the eyelids or the lips or contract one of the joints. At the same time fresh brown pigment-spots and stigmata appear

* *Synonyms.*—Kaposi's disease. It has also been called "xeroderma pigmentosum" (Kaposi), "angioma pigmentosum et atrophicum," "liodermia cum melanosi et telangiectasia" (Neisser), "leptodermia maligna" (Cavafy), and "atrophoderma pigmentosum." For bibliography, see the paper by Dr Funk, of Warsaw ('Brit. Journ. of Derm.,' vol. i, p. 182).

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on the affected surface. The former undergo the same atrophic changes, the latter may increase until they resemble congenital vascular nævi.

The disease is not accompanied with itching or pain, yet after continuing in this innocent form for months or sometimes years, the last stage comes on. The vascular spots become warty and ulcerate; fungoid growths of a most malignant character appear, not only in the maculæ, but also in distant places; and death ensues by hæmorrhage or exhaustion.

Hebra and Kaposi together observed only four cases of this remarkable affection. Erasmus Wilson described another under the name "general atrophy of the skin." Kaposi, in 1885 ('Wiener med. Wochenscrift,' No. 44), tabulates only thirty-eight published cases; the youngest patient was five months, the oldest forty; eighteen were males and twenty females. Rüder saw seven brothers affected! A remarkable case was shown at the Clinical Society as one of lupus, and was recognised as identical with Kaposi's disease by Dr T. C. Fox. This same case, with the others in the same family, will be found fully described by Dr Crocker in the 67th volume of the 'Medico-Chirurgical Transactions,' p. 169, with coloured lithographs, and a table of thirty-four cases; most of these were recorded by Rüder in a monograph on the subject, the rest by R. W. Taylor, of New York, by Neisser, and by Vidal.

From this table it appears that the disease has never been yet observed above the age of puberty. One case occurred in an infant four months old, most under two years, one at nine, and one as late as sixteen.

As a rule it occurs in boys and girls indifferently. More than one case is found in a family; twenty-six of the thirty-four cases in Dr Crocker's table belonged to nine families. The disease is never congenital.

The histological characters of the spots are those of vascular dilatation of pigmentation, and of atrophy. The final tumours appear to be always true epithelial carcinoma, not sarcoma.

Treatment has at present been unavailing.

The ordinary malignant growths of the skin are happily infrequent, nor have they many special points of interest; for their pathology is essentially the same as that of the corresponding growths upon mucous membranes; moreover, their recognition is not difficult and their treatment purely surgical, so that but little need be said of them in this place.

Carcinoma fibrosum, or scirrhous cancer, the most typical of all the forms of cancer, rarely affects the skin primarily, though it frequently infiltrates it as the result of primary carcinoma of deeper parts, as, for instance, of the mamma. The writer has seen three examples of the remarkable form of hard, indurating, and widely spread cancer of the skin, described by Velpeau as *squirrhe en cuirasse*. One was a patient under Velpeau himself, in whom the disease had spread from a cancerous breast; another was a patient of Dr Humphry's, of Cambridge. The remarkable and wide-spread induration, before ulceration begins and before implication of deeper organs occurs, renders it peculiar, and causes a superficial resemblance to sclerodermia or to certain forms of lupus. It is usually secondary to mammary cancer.

Epithelioma,* or keratoid cancer (see vol. i, p. 91), is the most common

* The term *epithelioma*, applied by Hannover, of Copenhagen, to this disease, of which he was the first to describe the histology, was discarded by Virchow for "epithelial cancer." Unfortunately "epithelioma" is now used by some German writers to designate molluscum contagiosum.

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form of malignant disease in the skin. Even this is rare, compared with its frequency in the œsophagus and large intestine, and at the labial, anal, and urogenital orifices. Its formerly most frequent seat, the scrotum, is happily no longer so, and "chimney-sweep's cancer" has become a rare curiosity in this country.

Rodent ulcer.—This affection, originally described by Jacob, of Dublin, is now ascertained to be histologically carcinoma. (See Mr Hulke's paper in the 'Path. Trans.,' vol. xxii.) The presence of epithelial cells in the cutis vera, and of the nest-cells characteristic of the horny form of cancer, leaves no doubt of its real pathology. It is, however, the least malignant of cancerous growths, for it spreads slowly, there is little new growth, and it does not affect the neighbouring lymph-glands. It is usually seen near the eye, upon the side of the nose, on the cheek, or the temple. Like other kinds of carcinoma, it is a disease of mature life or old age. Its early stages are those of a small, smooth, pale growth not unlike a wart. If, as is sometimes the case, it has begun in a congenital mole, it retains the pigment of that structure. It often has a pearly aspect, so as to look somewhat like a molluscum tumour, or even like the cysts not unfrequently found about the eyelids. When ulceration begins, it is covered by a rather thin, dark, and adherent crust. It produces little or no pain, and advances so slowly that when it first comes under the surgeon's eye it presents the appearance of a chronic, indolent, indurated ulcer, with sharp, well-defined nodular edges, and no granulations. In its later stages it resembles more nearly its pathological allies, epithelial cancer of the lip, the scrotum, the glans, and the vulva.

Other cases which are clinically rodent ulcer appear to have a different histological structure (Verneuil, 'Arch. gén. de Méd.,' 1854, ii, 458; and Thin, 'Path. Trans.,' 1878, pp. 237, 241). A review of these and other papers from Thiersch downwards, by Dr Hume, of Newcastle, with histological drawings, will be found in the 'Brit. Med. Journ.,' Jan. 5th, 1884.

The diagnosis from tertiary syphilis lies in the ulcer being single, in its not invading the bones or other tissues, and in there being no other sign of syphilitic disease. From lupus it is distinguished by the scab being thin and dark, by its beginning at a much later period of life, and, in the last resort, by microscopical examination of the material obtained by scraping or squeezing the edges of the ulcer.

Under the title of *crateriform ulcer*, Mr Hutchinson has described a sore of the face or head, which is much more malignant than the rodent ulcer just described, since it grows far more rapidly. Ulceration begins at the summit of a rounded nodule, and forms a deep crater-like hollow with indurated walls. It is most common in elderly people ('Path. Trans.,' 1889, and 'Arch. of Surgery,' vol. i, pl. xi).

Both this and the more common 'rodent' ulcer can only be treated as a cancerous growth by excision.

Sarcoma.—Beside carcinoma in the histological sense of the word, the skin is occasionally liable to multiple sarcomata. These are almost always secondary to some internal growth; by their large number, small size, and hæmorrhagic or sometimes melanotic character they may resemble certain forms of purpura or pigmentation. This remarkable affection has been described in the first volume of the present work, p. 82.

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PIGMENTAL, HÆMORRHAGIC, AND NEUROTIC AFFECTIONS OF THE SKIN

"The freckles, blotches, and parched skins,
The worms, that, like black-headed pins,
Peep thro' the damask cheek or rise
On noses bloated out of size."

WHITEHEAD.

Albinism—Leucodermia, congenital and acquired—Its relation to leprosy—Canities—Melanodermia, secondary to dermatitis, syphilis, adrenal disease, malaria, &c.—Ephelis—Lentigo—Chloasma—Relation of melanodermia to leucodermia.
Petechiæ and vibices—Purpura urticans—Peliosis rheumatica.
Trophic neuroses—Zona—Neurotic bullæ—Symmetry in cutaneous diseases.

ANOMALIES OF PIGMENTATION.—We are familiar with degrees of pigmentation of the skin, not only in different races, but also in the wide difference between individuals belonging to the same stock, and even to the same family.

Albinism, or complete absence of pigment, not only from the skin and its appendages, but from the iris and choroid, is always a *congenital* defect in the human race, as in rabbits, mice, horses, and other animals. The so-called "white" elephants are either albinos or piebald.

Albinos occur occasionally among the dark races. The "white" negroes have a dirty, pale skin, colourless hair, and pink irides with dark red pupils.

*Leucodermia.**—This may also be a congenital variety of coloration or "malformation." Piebald horses may be called "abnormal," but we should scarcely say so of cattle, dogs, swine, or guinea-pigs. This condition is, however, far more common in domesticated races than in a state of nature.

A similar congenital "piebald" state of the skin is occasionally seen in human beings. In negroes and in the natives of India it appears to be not uncommon. See a drawing of a remarkable case in a Hindoo given by Mr Hutchinson in his 'Archives of Surgery' (vol. i, pls. i and ii). We sometimes see it in this country as white locks of hair.

When acquired after birth, leucodermia has been, and still is, confounded with leprosy. In fact, "white leprosy," when it does not apply to psoriasis, seems generally to mean leucodermia occurring in patches.

Celsus (lib. iii, c. 25) distinguishes elephantiasis (*i. e.* leprosy) from *vitiligo* (calf's skin, parchment skin), which he divides into three species (lib. v, c. 16):—*V. alphos*, scattered, colourless, slightly rough patches (*psoriasis?*); *V. melas*, pigment-spots, to be presently mentioned under *melanodermia*; and *V. leuce*, still whiter than alphos, with white hairs growing on the

* *Synonyms.*—Λεύκη—Vitiligo—White leprosy—Partial albinismus. The term vitiligo has been also applied to a circumscribed smooth white indurated spot level with or slightly sunk below the surface. This would make it identical with morphæa, *i. e.* with circumscribed sclerodermia (*v. supra*, p. 888). The word may well be abandoned.

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patches (*leucodermia*). But later writers speak of vitiligo, and more particularly of leuce, as varieties of leprosy. The same explanation appears to apply to the Arabic term "Baras," the equivalent of Leuce or Alphos in Greek, and Vitiligo in Latin, which was also intended to denote a white leprosy. The confusion is due to patches of skin occurring in true leprosy, which are either deeper or paler in tint than the surrounding surface. The ambiguity appears still to exist, not only among the natives of Southern India and Ceylon, but among some physicians, judging by their reports in the Blue-book referred to before as published in 1867. Dr Vandyke Carter states expressly that leucodermia is commonly confounded with anæsthetic leprosy.

The skin in leucodermia is perfectly normal except for the loss of pigment. The Malpighian layer and also the hair are affected. There is no anæsthesia. The border is convex, and often a pigmented line separates it from the normal skin around; this was the case in the specimen which Gustav Simon first examined histologically. The patches are usually multiple, sometimes very numerous. They are occasionally symmetrical,* more often irregular, with no predilection for one surface of a limb or the other. They may occur anywhere, but are most frequent on the trunk, especially the abdomen and genitals, where natural pigmentation is deepest.

Leucodermia is more common in hot countries and in the south of Europe than in England, but here cases are readily overlooked, since in most cases they are inconspicuous, and give rise to no discomfort.

Removal of the white patches has been attempted by blisters and other irritants, and also by tattooing. The result is not often satisfactory.

Canities.—General blanching of the hair is a well-known senile change. But, like baldness, it often occurs in early adult life, especially when the hair is very dark. Beside losing its natural pigment, and so acquiring a dull, yellowish, "milk-white" appearance, the hair is apt to become dry and admit air-bubbles, which increase its refractive power, and produce the glistening steel-grey or "silvery" aspect.

Many instances are on record of rapid blanching of the hair of head or face in consequence of mental anxiety or grief. The cases of Sir Thomas More, of Henry the Fourth of France, and of Marie Antoinette have become historical, and it seems impossible to deny the fact that this premature senile change may come on in the course of a few hours. Bichat and Alibert record cases which they actually saw, and Brown-Séquard has seen rapid blanching in his own beard ('Arch. de Phys.,' 1869, p. 442). A grey patch sometimes follows neuralgia (cf. vol. i, p. 387). The late Dr Laycock quoted an instance in which a sepoy was seen to turn grey in half an hour ('Med. Times and Gaz.,' 1862). A young man who once consulted the writer for some slight ailment had perfectly white hair. In answer to inquiry he stated that a few years before he had fallen asleep after a debauch, and on waking in a cold room in the morning found that his hair had turned white. The objection that his beard was brown was answered by the explanation that when the change of colour occurred it had not yet grown.

This sudden change is probably due to development of air-bubbles in the shaft of the hair.

Melanodermia (*melasma cutis, nigrities*).—Increased pigmentation of the skin, like its diminution, may occur either universally or in patches.

A dark skin at birth is always hereditary. In after years it may occur

* See a remarkable case of perfectly symmetrical leuco- and melano-dermia figured by Dr Lesser ('Ziemssen's Handbuch,' Bd. xiv, 2te Hälft., p. 186, fig. 11).

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as the result of exposure to the heat of the sun or to other irritants, or as the result of certain internal diseases.

As the result of hyperæmia or slight superficial inflammation, one sees increased pigmentation produced not only by the sun (eczema solare), but also by the wind in cold weather, or in driving, or by the cold of snow-fields, which, as Alpine climbers know, will scorch the face without sunshine. Among Professor Hebra's patients the writer once saw a youth appear, who had wandered over a great part of Hungary in rags during the depth of winter. The exposed parts of the skin had become almost the colour of a mulatto, yet there had been little or no sunshine. Dr Bowles, of Folkestone, has made some careful experiments on this subject which confirm the result of experience ('Journal of the Alpine Club,' 1888).

Although all hyperæmia produces more or less increased pigment, there is considerable difference in the effect of different inflammatory diseases. The deeper and more chronic forms of dermatitis have very little pigmentary influence, as we should anticipate from their seat lying below the Malpighian layer in the cutis vera. Long-standing eczema and chronic traumatic inflammation produce much pigmentation, as seen in the brown, almost black patches which surround indurated varicose ulcers in old people. Ordinary eczema, however, has little effect, and impetigo and scabies none at all.

Of the superficial inflammations, chronic inveterate prurigo produces the greatest pigmentation, and prurigo pedicularis almost as much, aided probably by the scratching which it occasions and also by the age of the patient; for all pigmentation is slow in childhood and rapid in old age.

Certain forms of erythema are marked by increase of pigment, particularly roseola (pityriasis) maculata, pellagra (acrodynia), and urticaria pigmentosa.

Psoriasis very early and readily causes pigmentation, and the colour is sometimes quite indistinguishable from the coppery hue of a syphilitic eruption. Indeed, we may say that next to syphilis, psoriasis will produce pigmentation in the shortest time. Lichen planus resembles psoriasis in this as in other particulars.

Besides the well-known brownish pigment which gives its characteristic colour to even early forms of specific eruption, a somewhat rare form of syphilide has been described by French authors as the "café au lait" form (*syphilide pigmentaire*). The writer has seen cases, both in Paris and in London, of ill-defined brownish maculæ occurring on the neck of women who were the subjects of secondary syphilis (p. 899); they are sometimes associated with patches of leucodermia.

Pigment has already been mentioned as occurring in some cases of sclerodermia and in the malignant kind of atrophic nævi of the skin called "xeroderma" by Kaposi (p. 929). The remarkable increase of pigment in the course of Addison's disease has been fully described in the chapter on that subject. Similar pigmentation, though far less intense, is observed as the result of malaria (vol. i, p. 330), and occasionally in the cachexia of cancer.

Maculæ—Ephelides—Lentigo.—It remains to mention circumscribed pigment-patches, which occur without inflammation and independently of any other morbid sign. The most familiar are the small, dark brown or yellowish spots which, when they occur on the face, are named freckles (*ephelides*). They are no doubt, as their name implies, the result of exposure to the sun. They occur most frequently on the face, but also upon the hands and arms when these are bare. They are almost confined to xanthochroic complexions,

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and are particularly common in persons with red hair, blue eyes, and the delicate pink and white skin which so often goes with them. These freckles, like the diffuse pigmentation of sunburn, disappear in time, though much more slowly.

Precisely similar minute dark spots appear in covered parts of the skin, and in mucous membranes, sometimes along with the melasma of Addison's disease or with pigmentation from malaria, and sometimes in conditions of health. Others are congenital, and may then be described as pigmentary nævi or "mothers' marks." When combined with a congenital papillary growth, often covered with a strong growth of hair, they are called "moles."

Chloasma.—More diffused and less intense patches of pigment occur upon the forehead of pregnant women, and have long been known under the name *chloasma uterinum.* In some cases they appear during each pregnancy and disappear after delivery. The word chloasma was at one time extended to the pigmented patches on the trunk which we now know to be due to a fungus, and call tinea (or pityriasis) versicolor. But there seems no reason why at present the term should not be reapplied in its original signification.

Similar pigment-spots on the forehead and about the eyes are symptomatic of ovarian irritation, and appear in some cases of dysmenorrhœa with each menstrual period. (See eight cases reported by Dr Champneys with valuable comments in the 'St Barth. Hosp. Rep.,' vol. xv.)

Such pigmentation may also be the result of sexual excesses in male subjects, but this cannot be distinguished from the dark circles round the eyes which often accompany severe attacks of headache, especially megrim. All these cases may be grouped together by their clearly neurotic origin. They must be carefully diagnosed from not unfrequent instances in which lamp-black or other pigment has been designedly applied to the face, forehead, and eyes by hysterical or otherwise deceitful women.

Lastly, there are certain cases in which patches of pigmentation occur in various parts of the body, unconnected with local irritation and without any internal disease.

These cases of idiopathic circumscribed melanodermia are decidedly rare, and are generally associated with leucodermia; that is to say, white patches occur in the pigmented surface. The former are sharply defined and have convex borders, the dark surface is most marked close to the white (beyond the effect of contrast) and gradually shades away into the normal skin. Most cases may be called either melanodermia or leucodermia, or both at once; and apparently cousist in an irregular distribution of pigment. The white patches come usually first.

Treatment.—Solutions of corrosive sublimate, such as "virgins' milk" and "Gowland's cosmetic" (p. 842), are believed to have the power of removing freckles. The mingled patches of white and dark skin just described are best left alone, but circumscribed pigmentary nævi which cause disfigurement on the face may, if small, be removed by excision or galvano-causis.

Cutaneous hæmorrhage.—The most important conditions in which ecchymoses, whether the small ones like fleabites (hence called *petechiæ*) or the larger ones named *vibices*, are seen upon the skin are those of scurvy and of purpura. In both cases hæmorrhage occurs in other parts as well as the cutaneous surface, and in scurvy the ætiology of the disease is characteristic.

In certain other cases—besides ecchymosis from mechanical injury—hæmorrhage accompanies cutaneous diseases.

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The peculiarities of hæmorrhagic smallpox, measles, and scarlatina have been already described (vol. i, p. 202, and also pp. 181, 190).

In eczema, scabies, psoriasis, lichen, and prurigo, ecchymoses never occur except as the result of scratching. Slight hæmorrhage often tinges the contents of the bullæ of pemphigus, especially in the gangrenous and cachectic forms of the disease; but it seems not to occur either in Herpes gestationis or in Pemphigus foliaceus.

All forms of erythema are liable to be complicated with hæmorrhage. It is a rare complication in urticaria (when it constitutes the *Purpura urticans* of Willan), very common in erythema nodosum, when it produces the subsequent bruise-like pigmentation, and most frequent in the forms of erythema which occur in the course of rheumatic fever (p. 690). This last condition seems to have been first observed by Schönlein, who named it *Peliosis rheumatica*. The erythematous patches appear acutely, with fever and synovitis. They are most often seen on the back of the hands and feet, the forearms and shins, but may also affect the thighs, hips, and trunk; they are not often symmetrical. Either from the beginning, or soon after their appearance, the redness is found no longer to fade on pressure; hæmorrhage has taken place. Successive crops of these papules or large patches may occur, each lasting about a week, and disappearing with only a slight macule to mark its place.

There is no need of a special name for this disorder. It is a true erythema, whether occurring in the course of rheumatic fever or in persons who have already suffered from that disease, and we have seen that both this connection with rheumatism and the liability to hæmorrhage are characteristic of the whole group of erythemata (p. 824). On this subject see "A Case of Rheumatic Purpura," with notes, by Dr Wickham Legg ('St Barth. Hosp. Rep.,' vol. xix).

Neurotic affections of the skin.—The only cutaneous disease which is certainly related to nervous disturbance is the following.

ZONA.*—The names for this disease owe their origin to the fact (with which the elder Pliny was acquainted) that it passes round the trunk of the body like a girdle. The brief notice of it given by Pliny also implies the knowledge of another striking feature, namely, that it is limited to one lateral half of the cutaneous surface. He says, "*enecat, si cinxerit,*"—"it kills, if it encircles;" and a popular tradition to the same effect still exists in England. Nevertheless zoster is never fatal, and does not pass over to the other side of the body from that first affected.

The most elementary acquaintance with anatomy could not fail to suggest, to anyone who had observed the distribution of the eruption in a case of shingles, that it corresponds exactly with that of the peripheral distribution of one or more of the dorsal nerves. Accordingly this has been recognised for many years; and, with it, the necessary consequence, that certain eruptions on the face and limbs, which follow the course of the nerves supplied to those parts, are identical with it. To these also the name of herpes zoster is now given, although when so applied it loses its meaning, since the affected area no longer has the form of a belt.

Anatomy.—The *eruption* of shingles consists of vesicles. These are of

* *Synonyms.*—Herpes zoster—Zona ignea—Ignis sacer (Celsus)—Exedens præcordiorum herpes (Tulpius)—Erysipelas phlyctænodes (Cullen)—Shingles; a corruption of Cingulum = zona and zoster, a girdle.

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flattened form, and larger than those of eczema, being often as big as split peas; they are arranged in clusters of perhaps twenty or thirty, each cluster lying on a reddened and slightly swollen patch of skin; when the vesicles are thickly set, they often run together, and form flat bullæ of irregular shapes.

Some years ago Dr Haight, of New York, found an opportunity in Vienna of investigating their structure with the microscope. His observations showed that their roofs consist of the horny layer of the cuticle, with some of the superficial elements of the rete mucosum adherent to the under surface; their floors are formed by the bare summits of the papillæ, with the deepest elements of the rete occupying the depressions between them; their cavities are traversed by numerous bands, consisting of masses of the intermediate elements of the rete, drawn out into long spindle-cells and cells with several tapering processes. The fluid which the vesicles contain is at first transparent, but after a time the presence of floating leucocytes renders it opalescent, and ultimately it may become purulent, or acquire a purple colour from the escape of blood through the softened tissues beneath. The cutis itself seems always to take some share in the inflammation, leucocytes being scattered in the spaces between its fibrous bundles, and along the vessels and nerves. When pus is formed, if the roots of the vesicles have been removed by the friction of the clothes, ash-coloured surfaces are exposed, looking like layers of false membrane. In other words, the histological changes in zoster are essentially identical with those which have been described in variola (vol. i, p. 204).

The number of clusters is very variable, from a single one to ten or even more. They are generally developed, not all at the same time, but in quick succession; those come out first which lie nearest the roots of the nerve whose branches they follow. After a few days fresh ones cease to make their appearance. There is a short papular stage; and some of the latest clusters not infrequently abort, without going beyond it. In certain very mild cases, when only one or two clusters are formed, none of them pass into a vesicular condition. Even if the disease should be of considerable severity, the eruption begins to dry up from the fifth to the eighth day; the centres of the vesicles become depressed, yellowish or brownish crusts form, and in the course of the third week these fall off, leaving reddish or purple stains. But when the cutis is thickly infiltrated with pus-cells, its superficial layer undergoes destruction, and an eschar has then to be thrown off: thus the process of healing is retarded, and an indelible cicatrix results. The distribution of such cicatrices in the course of a particular nerve shows at once the nature of the disease from which they arose. When zona attacks the forehead, it is particularly likely to leave permanent scars.

Bärensprung and other writers have given names to numerous varieties of zoster, according to the nerves affected; but the refinement is needless. When the disease attacks the face, the nerve which it follows is the fifth, the greater part of which answers to the sensory portion of an ordinary spinal nerve. Indeed, it is remarkable how exactly the clusters of vesicles sometimes map out the points of emergence of the several twigs of the trifacial nerve from their bony canals. When the first division of the nerve is affected, the loose tissue of the upper eyelid becomes extremely œdematous and swollen, so that the affection may be mistaken for erysipelas by a careless observer. Another peculiarity of this form (supra-orbital zona) is that it is often attended with ulceration of the cornea and iritis, by which the

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sight may be seriously damaged. Mr Hutchinson has remarked that the ocular affection never arises unless the eruption occupies the distribution of the nasal twig. When the two lower divisions of the trifacial nerve are involved, a few vesicles often appear on the mucous membrane of the mouth and palate. Paget has recorded an instance in which necrosis of the alveoli followed infra-maxillary zona, so that some of the teeth fell out.

Cervical zoster, and that which affects the upper limb, follow exactly the distribution of the several nerves. In some instances of brachial zoster the vesicles reach down to the fingers, but this is very exceptional; in the great majority of cases they do not extend below the elbow.

When the second and third intercostal nerves are affected, the intercosto-humeral branch produces a very characteristic eruption down the inner side of the arm as far as the elbow or rather lower.

On the trunk, which is by far the most frequent seat of the disease, the area occupied by the eruption of course slants more and more downwards as it approaches the pubes. It often happens that one or two vesicles lie slightly beyond the meridian plane, both at the linea alba and at the spine; this probably depends upon the fact that the nerves of the opposite sides overlap in their distribution, just as in the Siamese twins there was a part of the connecting band which received nervous filaments from each of them.

In the lower limb the distribution of zoster presents this peculiarity, that it is almost invariably confined to the buttock and thigh. Mr Hutchinson says that it never extends below the knee, and the only instance to the contrary is one figured by von Bärensprung in which there were a few small papules as low as the middle of the calf. Often the eruption closely follows the distribution of the external cutaneous or anterior crural nerves or that of the small sciatic; but frequently one of these nerves is affected along with the ilio-inguinal branch, or the posterior divisions of the lumbar or the lateral branch of the last dorsal or some of the sacral nerves, and thus somewhat confused groups of vesicles are seen covering the gluteal, inguinal, or upper femoral regions.

Side affected.—True zona is always strictly unilateral; and there is probably no difference in the liability to it of the right or left side. In ninety-four consecutive cases the writer found that forty-one were on the right and fifty-three on the left side.

Mr Hutchinson once saw a zoster in the course of the fourth dorsal nerve on the *right* side associated with a frontal zoster on the *left* side. In one of von Bärensprung's cases an ordinary zoster, limited to the *right* half of the thorax, was accompanied by a single vesicle in the *left* axilla, the patient having been suffering from severe burning pains on both sides.

Symptoms.—Zona runs an acute course of a week or two, and is seldom attended with fever or disorder of the general health. In children, who are liable to it at all periods after the first year, it commonly runs its course without any unpleasant sensation, or is merely accompanied by a little numbness and tingling. Von Bärensprung tested the cutaneous sensibility with a pair of compasses, and found that in two cases it was considerably increased, while in a third it was diminished. Sir Thomas Watson relates a curious case in which zona affected the scalp, and in which the patient, who had for seven years been plagued with continual noises in the head, became free from this symptom, and remained so for eighteen months afterwards. He also mentions another case, of a man in whom the eruption came out in February, and who suddenly lost a cough which had teased him

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all the winter. On the other hand, von Bärensprung met with two cases of zoster affecting the distribution of the fourth cervical nerve, in each of which vomiting occurred at the commencement,—a consequence, he supposes, of "sympathetic irritation" of the vagus and phrenic nerves.

But by far the most important subjective symptom of herpes zoster is pain of a neuralgic character, and referred to the same nerves, the distribution of which is followed by the eruption. This is entirely absent in young patients, but in adults it is generally present, and in old people it is apt to be exceedingly severe. Von Bärensprung cites a case in which there was only a single patch, of the breadth of two or three fingers, but in which the tenderness was such that the patient kept the part covered with the palm of his hand all night and all day, lest his linen should come into contact with the vesicles. When the eruption gets well the pain commonly subsides; but in some instances it continues long afterwards, for months, or even for years, with scarcely any abatement of its intensity. This suffering may last ten years; and Trousseau mentions an instance in which the pain lasted for fourteen years. Sir Thomas Watson alludes to a case of this kind in which the patient, a lady, could at all times bring on the pain by drinking some cold liquid. The persons in whom shingles leaves behind it this terrible neuralgia are always advanced in age.

Sometimes the pain precedes the development of the eruption by several days. This has led certain writers, among whom was Anstie, to regard herpes zoster as a mere complication of neuralgia, analogous to several other curious "trophic" changes which are met with in that disease. But in opposition to such a view it must be urged that in the majority of cases of shingles (in two out of three, according to von Bärensprung) pain is altogether absent, and that when it is the earliest symptom the cutaneous affection always appears within a definite period, a fortnight at latest. Some instances have, indeed, been recorded of an eruption, regarded as zoster, which seemed to be caused by pressure upon the corresponding nerve-trunks, or which occurred in the course of ordinary neuralgia. Thus Charcot and Cotard have published a case in which one half of the neck and one shoulder were covered with the vesicles, the cervical nerves of that side being compressed by cancerous disease of the vertebræ. Charcot is said to have met with another case in which a patient, during a second attack of sciatica, presented herpetic vesicles on the lower part of the thigh. In these instances it seems not improbable that the eruption really was shingles, and that it was caused by an extension of the morbid process from the trunks of the nerves to the ganglia on the posterior nerve-roots. But there are other cases of which such an explanation is less easy. Thus von Bärensprung reports an observation by Esmarch of abscesses about the pelvis and under the gluteus maximus, where *post mortem* the great sciatic nerve was found to be swollen and reddened. About five weeks before the patient's death an eruption of groups of vesicles appeared on the back of his leg, and on the sole of the foot as far as its middle. In this instance the seat of the cutaneous affection was unlike that of zoster, for that scarcely ever goes below the knee. The same objection applies to Charcot's oft-quoted case of a man who suffered from pain in the leg and back of the foot after a gunshot wound of the thigh, and in whom an herpetic affection repeatedly developed itself upon those parts. Moreover, the fact that the eruption recurred is of itself sufficient to show that it was not really zoster, for shingles never relapses.

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We have had more than one case of recurrent bullous or vesicular eruptions following injuries to nerves, and one of typical zona in a patient suffering from cervical pachymeningitis, but it is very rare for any such organic disease of the cord or nerves to be present when zona appears.

Pathology.—There can, however, be no doubt that zona is a true trophoneurosis. This seems proved by its exact nervous distribution; and if further arguments are needed, one may be found in the fact that herpes zoster is associated with anatomical changes in the ganglia of the posterior nerve-roots. That these ganglia are the starting-points of the disease was suggested by von Bärensprung in 1861. His opinion was verified by Charcot and Cotard, who found (in a case already referred to) that whereas the nerve-roots were healthy, the ganglia and the nerve-trunks to a little distance outside the intervertebral notches were much reddened and slightly swollen, their stroma being also unduly rich in nuclei. Precisely similar appearances were afterwards discovered by von Bärensprung himself in a child which died soon after an attack of shingles; and additional evidence to the same effect has since been obtained.

Mr Hutchinson regards zona as resembling an exanthem, particularly in its power of protecting against itself; in a series of a hundred cases collected by him there was only one in which there was any history of a previous attack. But this may depend on the fact that shingles is of infrequent occurrence; and in 100 cases under the writer's care no fewer than four of the patients gave a history of a previous attack of the same disorder, and two of them showed scars which confirmed the statement.

With regard to the *causes* of herpes zoster almost nothing is known. It occurs equally in both sexes, and at almost every age. It has repeatedly been observed in persons who were taking arsenic. In one instance, a patient of the writer's assured him that his father and a younger brother had suffered from a similar unilateral and painful acute eruption before.

Cases.—Among 100 consecutive cases of zona which came under the writer's care in private or hospital practice, there were 61 in male and 39 in female patients. The time of life varied from infancy to old age; 15 patients were between one and nine years old, 31 between ten and twenty, 10 between twenty and thirty, 11 between thirty and forty, 8 between forty and fifty, 6 between fifty and sixty, 9 between sixty and sixty-five, one was seventy-seven, and one eighty-five years old.

The distribution was as follows. There were nine cases of supra-orbital and one of infra-orbital zona. In sixty-six cases the intercostal nerves were affected, in eight the great auricular, lesser occipital, or descending branches of the cervical plexus, in two the external cutaneous branch of the brachial plexus, in eight the lumbar or sacral nerves, in nine the small sciatic, the external cutaneus of the thigh, or branches of the anterior crural nerve.

Treatment.—There is but little to be done. The vesicles must be protected by a soft linen rag, with a pad of cotton wool or a flannel bandage. Some writers recommend that flexible collodion should be painted over them to facilitate their drying up; or a little starch powder may be dusted over the affected part as soon as any discharge appears. Children require no medicine whatever.

The treatment of the neuralgia which sometimes follows shingles is unsatisfactory. Bazin is said to have used arsenic with success; but as a rule it utterly fails. Dr Fagge used to prescribe vinum colchici, and in

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several instances the pain has quickly subsided while the patient has been taking this medicine. Subcutaneous injection of morphia gives at least temporary relief, and in old patients some form of opiate is almost always needful. Locally, anodynes are generally applied, but without much benefit. Cucain is perhaps the most efficient. Von Bärensprung found blisters useful, and they have been applied with good success over the roots of the affected nerves.

Pruritus leads to scratching, and how important an agent this is has been shown in our accounts of eczema, prurigo, scabies, and urticaria. These pruriginous diseases are in striking contrast to syphilodermia and lupus.

But it has been supposed that disorders, not of sensory but of trophic nerves, produce cutaneous diseases other than zona. The existence of trophic nerves is a physiological fact, but their presence gives so easy an explanation that we must beware of admitting it without adequate proof.

Area has been referred to a neurotic cause, but there is no proof of it. Little if any anæsthesia is to be detected, and the patches do not follow the course of cutaneous nerves.

Leucodermia has been referred to the same cause. But here again there does not seem to be any reason for ascribing the disease to nerves, but the difficulty of finding a better explanation. All that can be said in favour of the neurotic origin of *morphœa* will be found in Mr Hutchinson's 'Clinical Lectures,' vol. i, p. 313.

The formation of the wheals of *urticaria* may be ascribed to vaso-motor nerves, and it sometimes follows mental emotion with great rapidity.

Certain *bullous* eruptions also appear to be connected with neuroses. There are many instances of painful, usually vesicular or bullous, forms of dermatitis following injury to a nerve. Some remarkable cases have been recorded by Dr Schwimmer, of Buda-Pesth, Dr Leloir, in France, and Dr Weir Mitchell in his admirable monograph. On the injuries of nerves see also Mr Bowlby's more recent work on the same subject (cf. vol. i, pp. 404, 530).

It has been held that symmetry points to a nervous origin of a cutaneous disease; but some authors hold symmetry to be the mark of "blood diseases," and asymmetry that of neuroses. Neither belief seems to be well supported. Symmetry points neither to a constitutional nor to a blood disease. All general diseases are symmetrical because the human body is so; a one-armed man would be unsymmetrically affected by scarlatina or psoriasis. Again, psoriasis and eczema are symmetrical because they affect the skin of a certain structure and surroundings which is found on the corresponding parts of the limbs, ears, and other parts. The only disease of the skin which we know to be of nervous origin—zona—is, like neuralgia, markedly unsymmetrical.

See, however, on this subject the monograph by Leloir, 'Recherches sur les Affections cutanées d'origine nerveuse,' 1882, and a valuable paper by Dr Crocker, with numerous references ('Brain,' October, 1884, p. 343).

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REMARKS ON THE PRACTICAL CLASSIFICATION AND DIAGNOSIS OF CUTANEOUS DISEASES

"Mais ce qu'il y a de fâcheux auprès des grands, c'est que, quand ils viennent à être malades, ils veulent absolument que leurs médecins les guérissent."—MOLIÈRE.

IN the preceding chapters the diagnosis between two diseases which may be mistaken one for the other has only occasionally been stated in a formal manner. If the characteristic symptoms and circumstances of a malady are ascertained, they form the only and sufficient bases for its diagnosis; and although it is a useful exercise for a student to make lists of the distinctive characters of two or more diseases, the attempt to fix them in a tabular form is of little service to others, and perhaps tends to artificial memory of words rather than to familiarity with things. Symptoms differ endlessly at the bedside, and none of them are really what is called "pathognomonic." Moreover, the diagnosis of cutaneous diseases in particular often turns upon very slight differences in the form of lesion or in the distribution, which it is impossible to put into words; while much of what is called diagnosis is not distinction between one pathological condition and another, but only between certain more or less arbitrary forms which have been fitted with still more arbitrary names.

In the present chapter it is proposed to treat briefly of this question of diagnosis, on which, in its true meaning, all successful treatment must rest; and after all, our patients, great or small, desire to be cured.

There are not a few diseases which are so rare that they rank as little more than as curiosities. Such, for instance, are Pemphigus foliaceus, Urticaria pigmentosa, Favus, and Xerodermia maligna.

Many important diseases of the skin again are exotic, as Leprosy and Lichen agrius, and only of practical importance for English readers who may practise their profession in India or the colonies.

Neglecting these, there are certain common affections which, differing by more or less important characters in appearance and in histology, nevertheless agree very closely in their general pathology, in their causes so far as they are known, and, what is most important, in the kind of treatment which is generally suitable.

From a practical point of view, then, looking chiefly to questions of prognosis and treatment, we may arrange diseases of the skin as follows:

I. *Factitious eruptions.*—We must never forget the possibility of the affection before us being artificial. All kinds of dermatitis, eczema, erysipelas, pemphigus, impetigo, may be simulated by the application of various irritants. Pigmentation also has been often imitated with success. Such artificial diseases will generally be found upon the arms, rarely on the face, and scarcely ever beyond reach of the patient's hands. The persons who are guilty of such attempts at imposition are usually either deliberate

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malingerers, like prisoners in gaol, or else they are hysterical young women and neurotic girls or boys. When one's suspicions are once awakened, it is seldom difficult to detect the imposture. Mustard, cantharides, and some other irritants can be distinguished by help of the microscope.

II. *Traumatic eruptions.*—In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the disease that the eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis: *sublata causa tollitur effectus.*

Pediculi in the hair should be carefully looked for in all cases of impetigo in children, pediculi vestimentorum in prurigo of old people. Scabies itself is but an extremely definite and well-characterised dermatitis resulting from the presence of a living source of irritation. But beside these well-known cases of parasitic dermatitis it will be found that some supposed cases of purpura in children are nothing but fleabites, resemblance to which originated the name petechia. Moreover, many cases of infantile prurigo, urticaria, and ecthyma are due to the presence of bugs or gnats. In adults pediculi pubis may sometimes be found in the axillæ as well as in their proper region, and when they have been destroyed by mercurial ointment the patient is at once relieved from pruritus.

In many trades an irritant must be sought in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocers' itch). So with many of the "chemicals" used in a variety of modern handicrafts. Constant wetness of the hands in washerwomen, in scrubbers, in potmen, and many others, produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides, the heat of the fire of the pigment spots on the shins of elderly people. Sweat, again, is a very common irritant, producing the erythema which usually accompanies sudamina and also intertrigo of opposed surfaces. Scratching as a cause of traumatic dermatitis has been repeatedly referred to.

III. *Febrile rashes.*—We must take care never to forget the possibility of a cutaneous eruption being part of an acute exanthem. The use of a clinical thermometer is a great help in this respect, but the writer has seen a man with typhus (and the rash fully out) appear as an out-patient for a skin disease, and modified variola and varicella are not unfrequently mistaken for acne or impetigo.

IV. *Syphilodermia.*—When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor a symptomatic eruption, we may next consider whether or not it is due to syphilis. In this inquiry it is undesirable to ask questions, the answers to which are as apt to mislead as to guide aright.

(1) We should first consider the *colour* of the affected skin, remembering, however, that the pigmentation which gives the so-called coppery or raw ham tint to a syphilitic eruption is the same which is sooner or later produced by all forms of dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo may all produce shades which bear the closest resemblance to syphilodermia.

(2) The lesions of syphilis are *multiform* or polymorphic. It is rare in

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any but syphilitic affections to find mere hyperæmia in one part and associated pustules, papules, scales, or ulcers in others; and it is not often that a syphilitic eruption exhibits only a single elementary lesion.

A pustular eruption in an adult should always suggest the question of syphilis when that of scabies has been answered in the negative.

(3) Syphilitic eruptions for some unknown reason *do not itch*, and the exceptions to this rule are remarkably few; they usually occur during the stage of scabbing of pustular rashes or during the healing of tertiary ulcers. An ordinary secondary syphilide may, however, as a rare exception, be so irritable that wheals and scratch-marks are produced. On the other hand, psoriasis is often free from irritation, while the degree of itching of eczema, and even of scabies and prurigo, varies greatly.

(4) The local *distribution* of syphilitic diseases is a great aid in diagnosis. Specific eruptions are certainly not, as is often stated, symmetrical; the early roseolous rash is only so because it is general, and therefore, upon a symmetrical surface like the human body, more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line. But we do not see symmetrical patches of syphilide in corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. In all but the earliest syphilides the affected patches are very decidedly and constantly *unsymmetrical*, irregularly scattered over head, trunk, and limbs, and chiefly remarkable for having no well-marked seats of predilection.

The forehead, especially about the roots of the hair, is, however, very frequently the seat both of the early and middle erythematous, scaly, and pustular syphilides, and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position we should at least consider the question of syphilitic origin.

(5) These signs alone or in combination serve to distinguish early specific roseola from erythema, eczema, scarlatina, and measles, and the later eruptions from eczema, lichen, scabies, impetigo, and psoriasis.

The eruptions of *congenital syphilis* which are most liable to be mistaken are—the so-called pemphigus of infants, which is known by its affecting the palms and soles; rupia, which, by the form of the crusts and the ulcerated surface beneath, may always be distinguished from impetigo; an erythematous rash of the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery colour, its blotchy distribution, and more defined margin.

The *tertiary ulcers* of syphilis are distinguished by their appearing on unusual places, by their punched-out edges, circular or so-called horseshoe shape, and by their usually producing little pain or discomfort. Tertiary ulcers have no predilection for the outer side of the leg, but inasmuch as the part above the inner ankle is for anatomical causes the chosen seat of varicose ulcers, most ulcers in the first position will be syphilitic, and in the latter not. For the same reason most ulcers on the arms are found to be tertiary.

V. *Tineæ.*—The next great group of skin diseases includes those which are due to vegetable parasites—tinea versicolor of the trunk, eczema marginatum of the perinæum and thighs, tinea circinata of the neck and other parts, tinea sycosis of the chin, and tinea tonsurans of the scalp. Here the general characters detailed in the chapter on the tineæ are generally

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sufficient to show the nature of the affection to a practised eye, but in all doubtful cases the microscope should be employed.

Tinea of the scalp is rare in adults, and tinea circinata still more so; tinea marginata occurs only in adult males.

VI. *Primary superficial inflammations.*—To distinguish the superficial from the deeper kinds of dermatitis we should notice whether the cutis alone is infiltrated and thickened, or whether it is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to the acarus, to pediculi, and to other direct irritants, and excluding those which are the result of vegetable parasites and of syphilis, fall with respect to their treatment into three large groups:

(1) The first, represented by impetigo and most forms of eczema, are subacute, and accompanied with burning itching and pain, sometimes with a slight degree of fever. They are to be treated by local remedies designed to reduce the hyperæmia, diminish the exudation, and calm the irritation, aided by light diet, free diluents, laxatives, and diuretics. In short, they are to be treated according to the modern antiphlogistic method.

(2) The second group of superficial inflammations of the skin is typically represented by psoriasis, but includes lichen planus, the more chronic, dry, and obstinate forms of eczema, and true prurigo. They are chronic, with little irritation, exudation, pain, or active signs. They are best treated locally by tar or allied preparations, internally by arsenic.

(3) The third group is that of Erythemata. Here the indication is to correct some internal disorder of which the eruption is the symptom.

VII. *The acne group.*—Acne, both in its pathology and ætiology, differs from other forms of dermatitis. The age of the patient and its distribution are sufficient for diagnosis. It is at once a superficial and a deep dermatitis, and is often followed by scars. Its treatment consists entirely, or almost entirely, in local applications directed to the correction of the sebaceous affection. With acne may be classed Sycosis and Furunculus.

VIII. *Deep affections.*—When we have ascertained that the affection of the skin is deep, that is to say, that it goes below the papillary layer, the field for diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms, excluding the pustular affections which affect the skin deeply and produce scars only at isolated points, such as acne, variola, and zona; and excluding, thirdly, leprosy and other exotic diseases, we have to distinguish in the great majority of cases which come before us in this country—first, traumatic and varicose ulcers; secondly, gummata and syphilitic ulcers; thirdly, lupus; fourthly, rodent ulcer and carcinoma of the skin.

With regard to the first of these, we must not assume, because a sore upon the skin is said to be the result of a blow or a kick, that it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare, and usually obvious from the age of the patient, the pain they occasion, their tumid margins, and their blood-stained secretions. Moreover, they are, with few exceptions, confined to the neighbourhood of the orifices of the

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body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to be sure of. Its locality, its slow and painless progress, and its belonging to the latter half of life, usually serve to distinguish it from lupus, and its being single, excessively chronic, and unaccompanied by nodes or other syphilitic lesions, are the best characters for diagnosis from a tertiary ulcer.

Between lupus and syphilis the difficulty of diagnosis is occasionally extreme. Lupus, however, is rarely more than single, syphilis is usually multiple; both are commonly free from pain and itching, but in syphilis the colour tends from red to rusty brown, in lupus from red to violet blue; the scars of syphilis are depressed and pigmented, those of lupus hypertrophic and white; the edges of a lupous ulcer are beset with nodules, those of syphilis are either thin and smooth or indurated by chronic inflammation; lupus is in the majority of cases a disease of the face, syphilitic ulcers are quite as frequently on the limbs or trunk; lupus is a disease of the skin alone, syphilis affects the subjacent tissues also.

THE END.

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